Objective measurement of arthritis by thermography

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Summary. Heat is one of the classical sympotoms of inflammation. Quantitative thermography (T) using a numerical index is a valid means of assessing arthritis in peripheral joints, Details of a standardised technique are given, and examples of the application to clinical drug trials.

Key words: thermography, quantitation, arthritis, anti-inflammatory.

A) INTRODUCTION

The cardinal symptoms of inflammation are traceable to the earliest records of clinical observation. They are ((dolor, calor, rubor, tumor et functio laesa>>. Thermography (T.), a rapid and efficient mean of recording heat, is an ideal technique for demonstrating localised inflammation[8] Arthritis, frequently a chronic inflammatory lesion, is very difficult to monitor accurately. Gross clinical changes occur and many systems for scoring the inflammatory symptoms are used.[16] Tab. I shows that many clinial tests are based on combinations of dolor, tumor and functio laesa.

Since pain is usually the dominant factor some measurement should reflect the subjective state of the patient. with the development of good analgesic drugs, improvements in function or state may be shown by subjective tests. These tests do not necessarily show any change in inflammatory state, and relief may be transient.

Rheumatologists and Orthopaedists have made very limited use of T. since it became available to medicine. The location of arthritic joints is not usually the key clinical problem in diagnosis. The high cost of equipment is therefore unjustified for many. However, the problem of assessment, particularly in the selection and evaluation of treatment, is important.[3'6, 17]

B) QUANTITATIVE THERMOGRAPHY

Quantitation of T. provides a means of following minor changes in joint temperature and hence the effects of drug and physical therapy. An ideal system will have similar normal values from differing parts of the locomotor system. It should also have an adequate scoring range for the abnormally high temperatures found in the various arthritides.

COLLINS et al[4] published a system for quantitative T. using a Thermographic Index (T.I.). This was based on the concept that under given conditions normal peripheral joints could be cooled to a predictable temperature range. By measuring isotherms from a standard window of the T., a single figure value was obtained. Two temperature ranges were chosen, 26-32°C for lower limbs and 28-34°C for the joints of the upper limbs. By the formula:

$$\text{T.I.} = \frac{(\Delta t \cdot a)}{A}$$

t = 26 or-28" C
a = area of each isotherm at O.5°C
A = total area

a range of values was obtained for ankles, elbows, hands and knee joints.

On this system, values of less than 2.5 were obtained from the joints of most normal subjects.[12]

A number of experiments have been carried out to ascertain the reliability of this system. On a daily basis, individual control subjects show some fluctuation, but usually within this range of normality. However, abnormal exercise or exposure to extremes of temperature, and smoking, can afect the readings even after 15min acclimatisation at 20°C. Providing such variables can be controlled, the T.I. is found to be most reproduceable during the morning

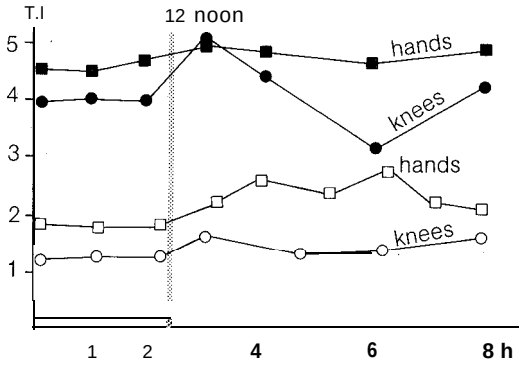


Fig. 1. The change in T.I., throughout an 8 h day time period in a normal (□○) and a rheumatoid arthritic (■●) subject. Mean values for both hands and knees are plotted.

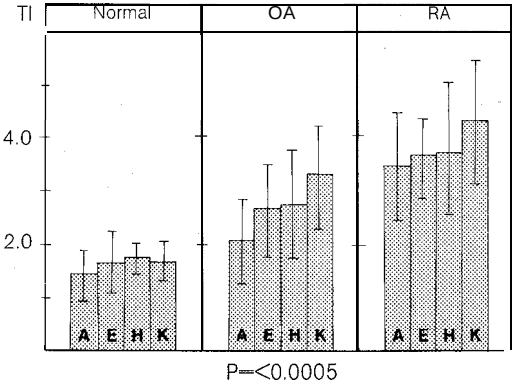


Fig. 2. Mean T.I. (and SEM) from the peripheral joints of a group of control, untreated osteo-arthritic (O.A.) and rheumatoid arthritic (R.A.) subjects. Each meanjointvalue was significantly raised ($p < 0.0005$) above its respective control. A = ankles, E = elbows, H = hands, K = knees.

hours before midday. The T.I. has been used to measure the circadian rhythm of joint temperature in both normal and arthritic subjects. While the results may vary considerably between subjects, the T.I. has consistently shown a rise at midday, defined by the chronobiologist as «the acrophase».[10] This phenomenon has been confirmed by studies over 3 months in the individual subject.[12] Afternoon temperatures have not, in general, proved as stable as the forenoon readings (Fig. 1). The Author has therefore adopted a standard procedure, where serial measurements for drug evaluation in arthritis are only made in the mornig period. However, it is probable that if the response to a dynamic test is employed rather than the basal value, the effect of time will be less marked.

It has been demonstrated that the infra-red emission from a knee relates to the intra-articular temperature measured with thermocouples. The temperature is related to the vascularity of the synovial membrane. Changes in vascularity have been shown, after treatment with steroids, by plethysmography and by isotopes, and correlate with temperature." In other studies the localised pathology of the joint, the enzyme and protein content and leucocyte migration index of the exudate, and the patients' plasma viscosity, have all been correlated with temperature.[9], [13] Absolute values of T.I. for diagnostic purposes have not been useful. However, when an unselected group of patients with osteoarthritis were examined, the mean joint indices (n = 200) were significantly warmer than the controls. Similarly, a group of patients with rheumatoid arthritis were significantly hotter than the osteoarthritic group. Both groups of patients were examined before treatment with anti-inflammatory drugs (Fig. 2).

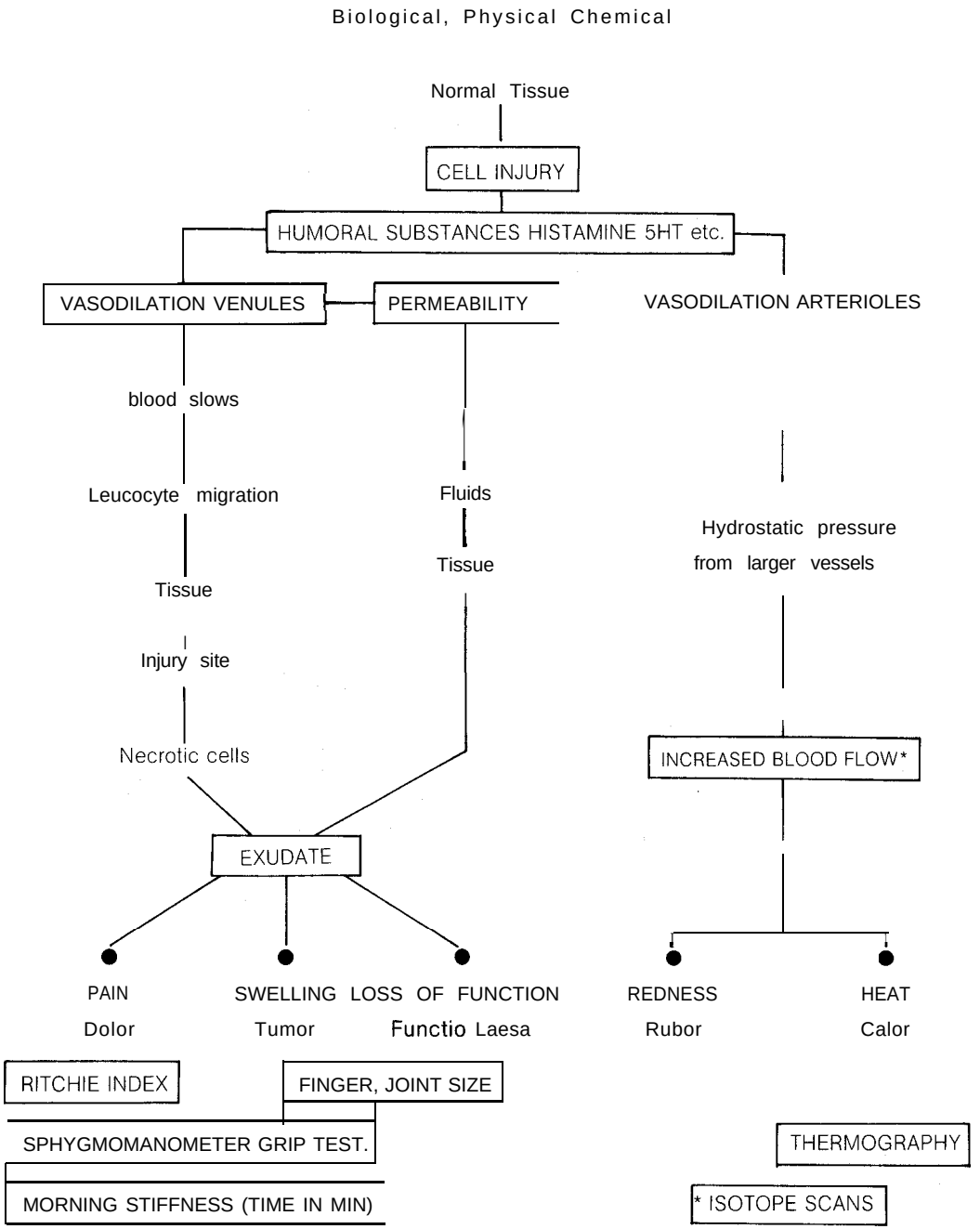
C) METHOD

The requirements for quantitative T. are: a) *controlled ambientemperature*; β) *thermal reference source* for calibration; *y) stable* T. with parallel stand; *8) image processing system,* either: i) small computer with image analysis; ii) integrator or planimeter for T.I. measurement; iii) data/image storage and remote processing facilities.[7]

The i) is ideal, allowing immediate processing, with data storage and retrieval. The ii) is the least expensive but calls for careful use especially when window selection is made on a small video screen. All 3 of these systems are now available, and more use of quantitative T. is being made in Rheumatology.

Once the position for the patient is standardised and quantitation achieved, a succession of scans from differing joints of the body may be made quickly and simply. Furthermore, the reporting of a numerical T.I. for each joint scanned, wiht stated ranges of normality, makes the interpretation of results more simple. Values may be reported and serialisedin the case notes, giving instant indication of the regional progress of treatment in that patient.

Tab. I. **Theorical scheme for the origin of the** Symptoms **of inflammation.**



D) CLINICAL DRUG TRIALS

1. In **arthritis,** the T.1 provides useful objective data in the evaluation of new drugs or design of drug regimes. These techniques have proved to be a sensitive indicator of anti-inflammatorv action (Fig. 3). An earlv observation that analgesic drugs do not suppress the T.I. as much as oral anti-inflammatory drugs, has proved very useful. <<Wash-out)>

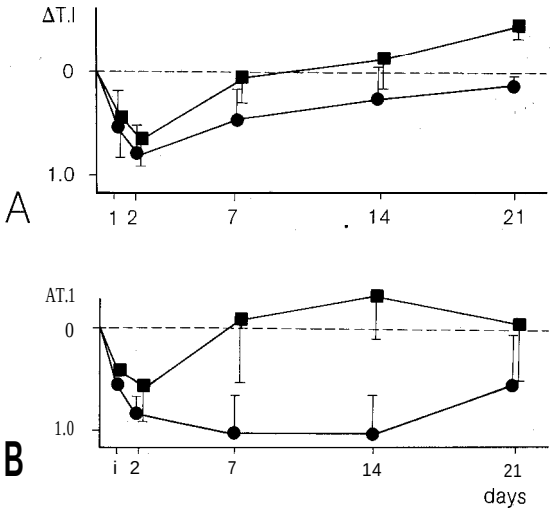


Fig. 3. Comparison of a new large molecule prednisolone, tributyl acetate (cedelcortone TBA) given as a single knee injection in 10 patients (B), against a small prednisolone acetate in a similar group (A). In both groups the injected knee (■) fell more than the contralateral joint (●). A more persistent localised effect occurred in B, in which the index was significantly reduced in the injected knees.

therapy with analgesics can therefore be employed prior to the anti-inflammatory drug, to provide base line data.' A number of trials have been monitored by T., including double blind crossover studies.' The technique has been applied to both experimental animals and man, and provided valuable data in dose response studies of new compounds.[2], [12] Further data has shown that there is a different response time for different joints to oral anti-inflammatory drugs. On the commencement of treatment, a significant reduction in T.I. from joints of the hand will be achieved by most effective drugs in 2 wks. Less significant changes occur in the ankles. At least 3 wks may be needed to achieve similar results in the larger joints (e.g. the knee).

This data is useful for the meaningful evaluation of clinical drug studies. The distribution of arthritis in individual patients will therefore bias the time to show response to treatment. The improvement at 2 wks, to a number of oral anti-inflammatory drugs (aspirin derivatives), has proved to be independent from the severity of arthritis measured by the T.I. Although greater improvement may be anticipated in patients with mildly active disease (and slightly raised T.I.), in this study T. improvement was not related to the degree of inflammation or to the type of oral non-steroid used.[13]

2. Beyond the practicalities of anti-inflammatory treatment, the same T.I. has proved useful in evaluating **Paget's disease** of bone. Providing the site is close to the surface, the T.I. reflects bone vascularity, and its level has correlated with bone pain. Therapy by injection of Salmon Calcitonin reduces the T.I. It has thus provided a useful medium for the dynamic recording of drug response and may precede clinical changes by several weeks. Alkaline phosphatase and T.I. have been shown to correlate.[14],[15] Further studies on the design of treatment regime for PAGET'S disease are in progress.

E) CONCLUSION

The true potential of T. in pharmacology research has yet to be developed. The stability of equipment and reproduceability of the method must be estabilised before a reliable quantitation can be used to show parenteral or oral drugs effects. The effects of anti-inflammatory drugs on inflamed peripheral joints are well demonstrated by these techniques.

REFERENCES

1. BACON P.A.: The recognition of anti-rheumatic drugs. *MTP Press Pbl., 19, 275-284, 1978.*
2. BACON P.A.,DAvIEs J.,RINGE.F.J.: The use of quantitative thermography to assess the anti-inflammatory dose range for fenclofenac. *Proc. Roy. Sot. Med., 70,* 18-19, 1977.
3. CHUDACEK *Z.: Medical Thermography*. Acta Universitatis Carolinae: STEINHART L. Ed. Prague, Mon. LXXVI, 6, 81-84, 1977.
4. COLLINS A. J., RING E. F. J., COSH J. A., BACON P. A.: Quantitation of termography in arthritis using multi-isothermal analysis. Ann. *rheum.* Dis., 33,113-115,1974.
5. DIXON A.S.J., DAVIES J.D.: A double-blind, crossover comparison of piroxicam and indomethacin in the treatment of rheumatoid arthritis. *Piroxicam Symposium Proceedings,* Wiesbaden. Academy Prof. Information Services Inc. Pbl. New York, 1980.
6. ENGEL J.M.: Thermographische Diagnostick in der Rheumatologie. *Akt rheumatol., 4, 25-37,* 1979.
7. ENGEL J.M., COSH J. A., RING E.F. J.: PAGE-THOMAS D.P., VAN WAES P., SCHOENFELD D.: Thermography in locomotor diseases- Recommended procedure. *Eur. J. Rheum. Inflamm., 2, 299-306,* 1919.
8. GOLDIE I.: Thermographic evaluation of results of sy-

novectomy in rheumatoid knee joints. *Acta Orthop.* Stand., 40, X2-391, 1969.
9. HALL N. D., BIRD H. A., RING E. F. J., BACON P. A.: A combined clinical and immunological assessment of four cyclophosphamide regimes in rheumatoid arthritis. *Agents and Actions. 9.* 97-102. 1979.
10. REINBERG A.: Circadian changes in the temperature of human beings. In *Medical Thermography,* N.J.M. AARTS, M. GAUTHERIE, E.F.J. RING Eds., Karger Publ. Base1 *Bibl. Radial., 6,* 128-139, 1975.
11. RING E.F. J.: Thermography and rheumatic disease. In *Medical Thermography,* N.J.M. AARTS, M. GAUTHERIE. E.F.J. RING Eds., Karrer Pbl.. Basel. *Bibl. Radioi, 6,* 91-106, 1975.
12. RING E.F. J.: Quantitative thermography and thermographic index. *Verh. Dtsch. Ges. Rheumatol., 6, 287-*288, 1980.
13. RING E.F. J.. COLLINS A. J.: Quantitative thermography. *RheuM Phys. Med.,* 10,337-341, 1970.
14. RING E. F. J., DAVIES J., BACON P. A., GRABER J., COSH J.A.:Quantitative evaluation of calcitonin therapy in Paget's disease. *Ann. rheum. Dis., 38, 494,* 1979.
15. RING E.F. J., DAVIES J., BARKER J.R.: Thermographic assessment of calcitonin therapy in Paget's diseases. In *Bone Disease & Calcitonin.* J. KANIS Ed.: Armour Pharmaceutical Co. Ltd. Pbl., Eastbourne, U.K., 1976.
16. TISELIUS P.: Studies on joint temperature, joint stiffness and muscle weakness in RA. *Acta Rheum. SCand., Suppl., 14,* 12-97, 1969.
17. ZYSNO E.A., RUSCH D.: Thermographische Methoden in der Rheumatologie. *Z. Rheumat.,* 31,231-235,1972.

Thermography in the study of the sacro-iliac joints

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Summary. Thermography (T.) is a useful complement in clinical and instrumental differential diagnosis of sacro-iliac diseases. The Authors emphasize the opportunity of further considering the T. features of each of the sacro-iliac diseases either for diagnostic purposes, or for the study of their natural course, and of their envolvement during treatment.

Key words: sacro-iliac joints, ankylosing spondylitis, thermographic examination.

A) INTRODUCTION

The sacro-iliac joints, like other joints [4,6,8], can be affected by inflammatory or osteodegenerative diseases, but also by autochtonous processes such as the so called ((osteitis condensans ilii >>, or the early stage of ankylosing spondylitis.

B) METHODS

The use of instrumental techniques, like radiographic and scintigraphic investigations,"" [11] is therefore particularly useful as a means of supporting the traditional clinical diagnosis.

Thermography (T.) has recently been added to the above techniques, serving the purpose of singling out those processes producing an increase of the tissue metabolic activity, with heat production and skin thermic changes.

With the use of T. it is indeed possible to point out the presence of hyperthermic areas in the inflammatory diseases like ankylosing spondylitis [3,9,10] (Fig. l), chronic rheumatoid sacro-ileitis, infectious sacro-iliac arthritis, psoriatic sacro-ileitis, or neoplastic diseases;[7] normal T. features in osteoarthritis, or hypothermic areas in osteitis condensans ilii.

C) RESULTS

According to Authors' experience, which includes the study of 30 cases of rheumatic diseases with sacro-iliac involvement shown clinically and radiographically, one concludes that it is possible to delineate constant T. features in some rheumatic diseases.

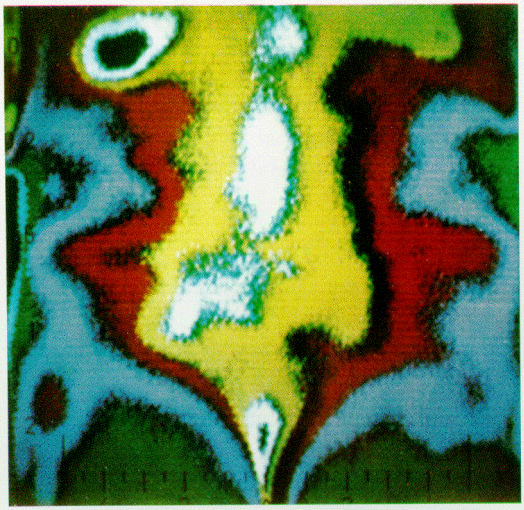

Fig. 1. Advanced ankylosing spondylitis. Hyperthermia of spine and sacro-iliac joints (<< losange >> pattern).

T., besides having an iconographic meaning and permitting the differential diagnosis of the above mentioned diseases, also consents the evaluation of inflammations either in their natural course or during pharmacological treatment[2]

T. is an extremely sensitive method that is able to point out the beginning of the inflammatory process even before its clinical manifestation. T. is certainly particularly useful in those cases of pelvic spondylitis in which clear radiologic features did not follow a dubious clinical picture.

It is superfluous to add that T. is of easy realisation and that it is free from those risks that result from repeated radiological examinations.

REFERENCES

1. BACARINI V., Maini C., MEROLA G., Pigorini F.: La scintigrafia ossea nella spondilite anchilosante. Atti Conv. su <<La ricerca reumatologica in Italia >>, 15,1978.
2. BACON P.A., COLLINS A.P., RING E.F. J., Cosh J. A.: Thermography in the assessment of inflammatory arthritis *Clin. rheum Dis., 2, 63, 1976.*
3. COSH J.A., RING E. F.J.: Tecniques of heat detection used in the assessment of rheumatic diseases. *J. Ra*diOL. *Elect.,* 48, 84-89, 1967.
4 DE SEZE S., RYCKEWAEKT A.: *Maladies des os et des articulations.* Flammarion Ed., Paris, pg. 980, 1954.
5 FoRESTIEK J., ROTES-QUEROL J., JACQUELIN F.: Les articulations sacro-iliaques dans la spondylarthrite ankylosantc. Rev. *Rhum., 17, 407-448,* 1950.
6 GILLESPIE H.W., Lloyd-Roberts G.: Osteitis Condensans. Brit. *J. Radiol., 301: 18,* 1953.
7 Lovisatti L., DAlL PALMA F., Gortenuti G, MAKCHI F.: L'indagine termografica nella patologia ossea. *Termografia* Medica. Atti I Simposio Nazionale. Cesena, l-2 sett. 1973, 297, Gaggi, Bologna.
8 LUCHEIUNI T'.: *Reurnutologia pratica* SEU Ed. (Torino) (pg. l95), 1956.
9 ROMIeu! C.L., LAURENT J.C., SIMON F., BLOTT.MAN J.L.: La telethermovision dynamique en patologie osteoarticulaire. Termografia *Medica.* Atti I Simposio Nazionale. Cesena, 1-2 sett. 1974, 275, Gaggi, Bologna.
10 SODOWSKA-WROBLEWSKA M., KRUSZEWSKI S., GAKWOLINSKA H., FILIPOWICZ-SOSSNOWSKA A.: The thermographic examination of sacroiliac joints. *Acta Thermogruphica, 1,* 54-61, 1976.
11 VAN LAere M., VEYS E.M., MIELANTS H.: Strontium 87m scanning of the sacro-iliac joints in ankylosing spondylitis. *Ann. rhrum. Dib., 31,* 201-206, 1972.

Hand thermography in normal subjects and in scleroderma

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Summary. Thermographic (T.) hand images in scleroderma have a characteristic appearance when compared to normals, This difference is most distinctive on preliminary films but differences in rewarming can also be detected. Hand T. may be a useful method for demonstrating Raynaud's phenomenon.

Key words: hand thermography, scleroderma, rewarming in thermography, Raynaud's disease.

A) INTRODUCTION

Patients with scleroderma frequently complain of cold intolerance and have manifestations of RAYNAUD'S phenomenon. The heat patterns in the hands of sixty-six patients with scleroderma from the UCLA Clinical Research Center were evaluated before and after a cold stimulus. The same procedure was also performed on 20 volunteers in order to establish the normal thermographic (T.) appearance of the hands.

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B) PROCEDURE

A preliminary image of the hands, placed palms down on a table top, was recorded with T. The hands were then immersed in ethyl alcohol (cold stimulus). The hands were then

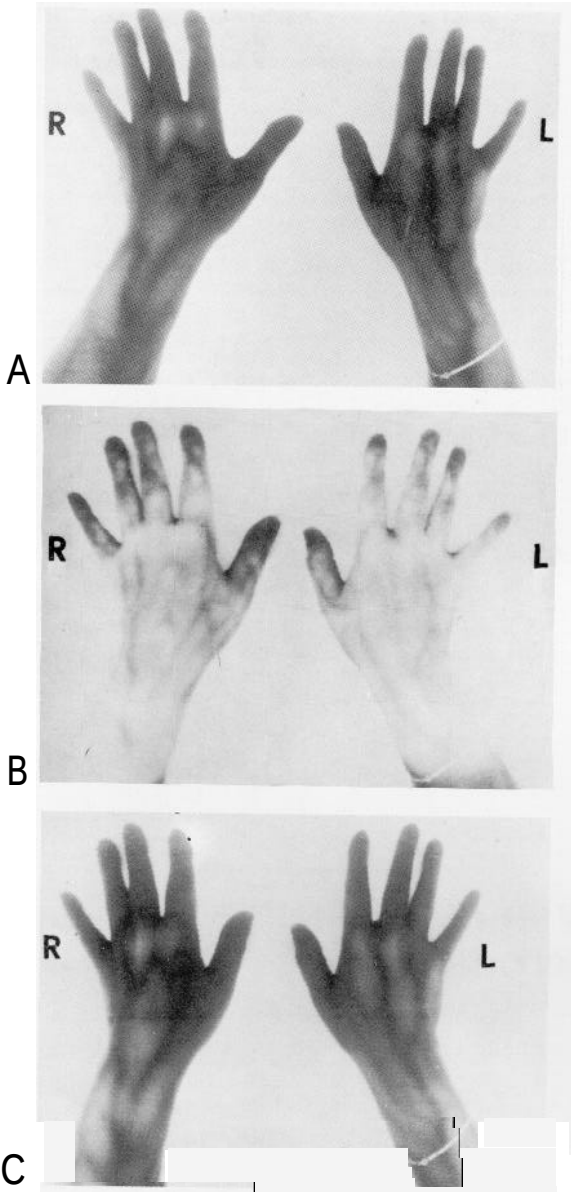


Fig. 1 A-B-C. A) Preliminary images of a normal subject's hands (black is relatively warm, white is cool). B) T. repeated directly after immersion in alcohol. C) T. repeated after 15 mins of rewarming.

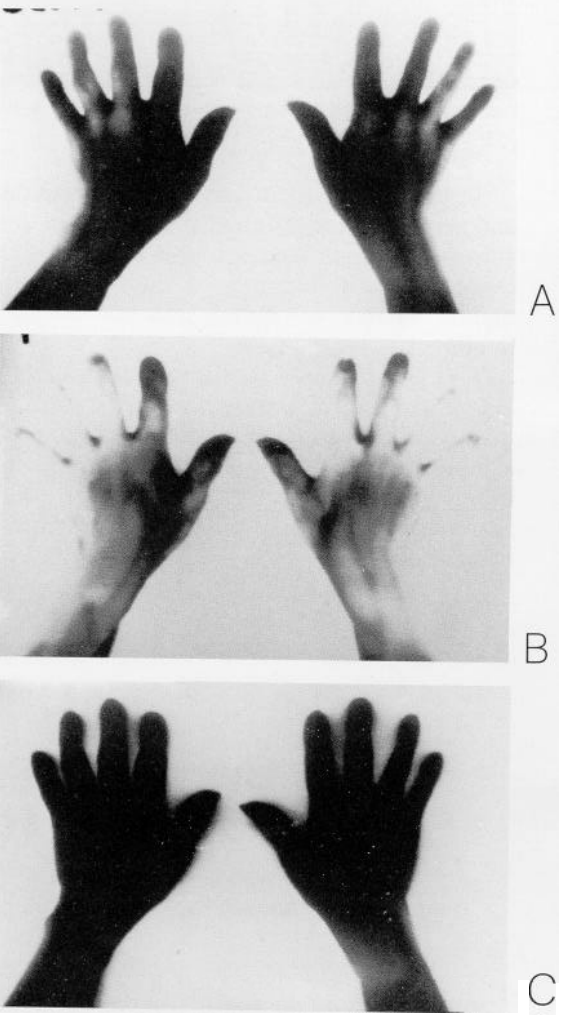


Fig. 2 A-B-C. A) Control, B) Cold stimulus, C) 45 mins after. Some subjects (from both normal and scleroderma groups) demonstrated a rebound phenomenon after cold stimulus (B) so that after rewarming the hands demonstrated markedly increased heat (C) compared to preliminary image (A).

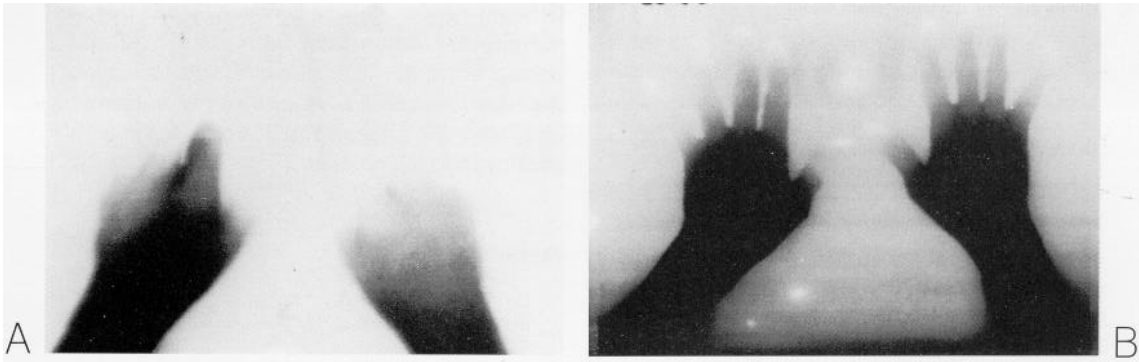


Fig. 3 A-B. A) The typical T. in apatient with scleroderma reveals decreased peripheral heat patterns on a preliminary stll-dy. B) Delayed rewarming even 45 mins after immersion in alcohol.

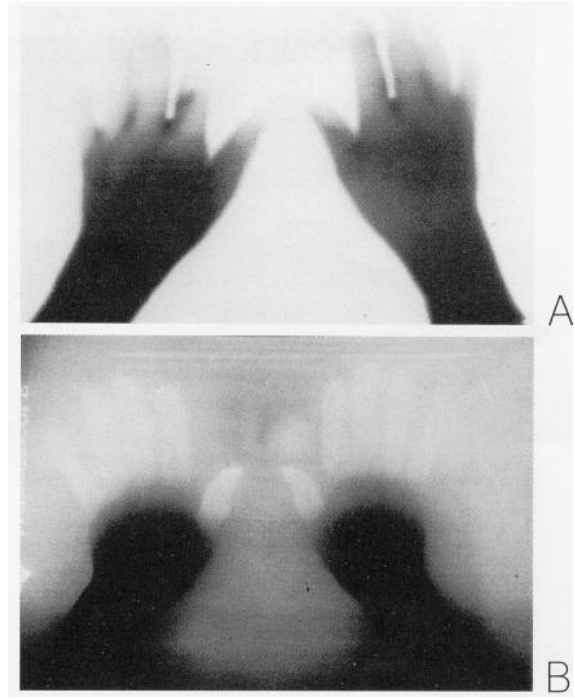
returned to the table top, and images recorded at 5 mins intervals as the hands rewarmed.

C) RESULTS

The distribution of heat in each of the normal hands corresponded in most cases (19/20) with previously reported findings.' For the purposes of this paper the symmetry and relatively equally intense heat of wrists and fingers was the most relevant characteristic of the normal T. (Fig. 1A). Immediately following alcohol immersion, the superficial heat was markedly decreased (Fig. 1B). In 50% of normals (10/20), the heat distribution returned to the base-line or preliminary image after 15 to 30 mins (Fig. 1C). In others, rewarming was delayed by variable intervals, so that the rewarming pattern was not as reliable in determining a normal hand T. as the preliminary image. Occasionally, the rewarming pattern was more intense than on the base-line. This «rebound» response was noted in 3/20 normals and also in 2/66 scleroderma patients and is of unknown significance (Fig. 2).

The typical preliminary pattern in the scleroderma patients (64/66) was asymmetry in heat distribution in the hands and decreased superficial heat in the fingers when compared to the wrist (Fig. 3A). Rewarming after alcohol immersion is almost always delayed beyond 30 mins and may not occur even up to 45 mins **(63/66)** (Fig. 3B).

As noted above, one <<normal>> volunteer had deficient superficial venous heat on the T. (Fig. 4). She was found on subsequent clinical evaluation to have RAYNAUD'S phenomenon



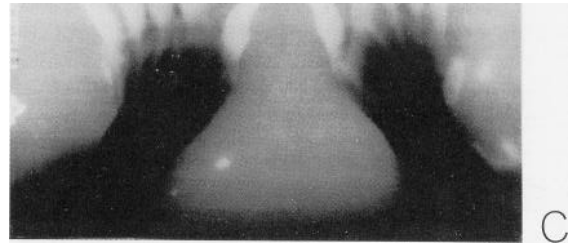


Fig 4 A-B-C A) Control. B) Cold stimulus C) 45 mins after. T. on one <<normal>> subject showed deficient heat distally. She was later shown to have RAINAUD'S disease.

(blanching followed by cyanosis of the fingers upon cold exposure). Thus in this case the T. revealed an unsuspected abnormality.

D) DISCUSSION

Patients with scleroderma have a hand infra-red emission image that is distinct from the image of normal patients. Patients with scleroderma also have a characteristic rewarming pattern on T. performed following a cold challenge. The response of normal subjects to the same cold challenge is variable. Thus the preliminary film is most distinctive in differentiating normals from patients with scleroderma. An abnormal T. in one of the volunteers disclosed an unsuspected early RAYNAUD'S phenomenon.

REFERENCES

1. ACCIARRI L., CUGOLA L., MASO R., NOCARIN L.: The thermographic hand. *Acta Thermographica, 3, 65-15,* 1978.

Thermo-regulatory function in skin: an aspect of psoriasis

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Summary. Thermography (T.) and thermistor thermometry have documented a difference between response of normal subjects and subjects with active psoriasis of the hands to a 20°C ambience, and to thermal challenge by immersion in 0°C water. The normal response to cool and to cold is an immediate drop in temperature of the extremities. The majority of psoriatic subjects (7/10) did not react to cold challenge. After thermal challenge with heat (immersion in 44% water) psoriatic subjects and normal subjects respond in the same way.

Key words: skin, thermo-regulation, psoriasis, thermography.

A) INTRODUCTION

Observations stemming from thermographit (T.) and thermistor thermometric (T.T.) studies of psoriatic and normal skin are the subject for the current report.

B) THERMO-REGULATION IN PSORIASIS

Some facts about psoriasis will show why thermal measurements were of interest. Psoriasis is a heritable skin disorder, a prevalent genodermatosis affecting 2% of the U.S. population. It is Characterized by erythematous lesions - from guttatae to plaque to annular to gyratae - with typical silver scales The recognized pathologic process in psoriasis is an accelerated epithelization of less than 28 days. Anatomically, the epidermis is bloodless. Thinned suprapillary epidermis overlying tortuous dermal capillaries characterizes psoriasis and accounts for the ready bleeding seen in scratched psoriasis lesions. Ultra-violet radiation in conjunction with topical tars that sensitize the skin to ultra-violet is one of the well established therapies.

The remission in part or in toto of psoriasis in summer-times is well known, often even without exposure to sun. This is significant because it suggests that psoriatics respond to altered ambient temperature.

WARSHAW, in previous work,' reported an increased thermal gradient in psoriasis plaques. While T. showed elevated temperature in psoriasis lesions, T.T. showed colder temperature in the same lesions than in the surrounding skin.

Clues from the clinical course of psoriasis, the burgeoning literature on thermo-regu-

latory physiology[1, 2, 3, 5, 8, 9] and temperature measurement of psoriasis lesions have all led to the present study.

C) METHOD

For the current study, 15 individuals with hand psoriasis were examined in a room with a constant temperature of 20°C (68°F) after 10 mins accomodation. The same number of normal individuals were thermographed.

For the second portion of this study, 7 individuals with active psoriasis of the hands were thermally challenged by hand immersion in ice water (0°C) for 60 s. A normal subject was studied with each psoriatic. T. were taken initially and right after the cold and heat challenges, and at 2 mins intervals for 3 more sets of T. after each challenge.

D) RESULTS

In 14/15 subjects with active psoriasis, the distal phalanges were at temperatures ranging from 25°C to 35°C. On the hands of the normal controls, distal fingertip phalanx temperature was consistently low; all were below 25°C and 8 were below 18°C.

Only l/15 psoriatic patients adapted to the 20°C ambient like the normal subjects. While the normal adaptation to such a cool environment was a drop in distal phalanx temperature, and a drop in temperature of the superficial veins on the dorsum of the hand, the psoriatics showed adaptive response to cold.[7] In that group, the superficial venous circulation remained warm-unchanged after 10 mins accomodation to the cool 20°C ambience.

In the second portion of this study, 6/7 psoriatics showed an even more pronunced difference in T. pattern right after a challenge by hand immersion in ice water. The persistence of high distal phalanx temperature is clear, and must be due to the failure of peripheral vein constriction.

E) DISCUSSION

The psoriasis T. indicates a defective vasomotor response or a fault in thermal sensing. In the hand there are both a deep artery vein circulation and a more superficial vein supply on the dorsum. After cold challenge, the high temperature of that superficial circulation (on the dorsum) reveals a malfunctioning hemodynamics in psoriasis. The normal responce to cold should be immediate vasoconstriction and a drop in superficial vein temperature.

The anatomic site of the psoriasis process has been in dispute. With abnormal response to cold in the superficial veins, psoriasis is plainly not wholly an epidermal process. Thermo-regulatory physiology should provide a provocative field for study in regard to psoriasis.

The rapid rate of epithelization associated with psoriasis can be explained by T. findings. An increased thermal gradient with elevated temperature below surface in the psoriasis plaque could account for the known accelerated epithelization. In chemical reaction, a rise in temperature of 1°C will significantly raise the rate of reaction. In similar manner, it is plausible that psoriasis plaques can complete epidermal turnover in 4 to 7 days instead of the normal 28 day transit.[4], 6, [10]

A severe heat loss occurs at the psoriasis plaque. A single lesion, or possibly 5 plaques involving perhaps 1% of skin surface may do little to change thermal balance, but extensive psoriasis covering over l/4 or more of body surface means that the body is no longer functioning well as a homeotherm. Fortunately, if there is a breakdown of automatic thermoregulation, it is offset by behavioral regulation, so that thermal comfort can be maintained. Behavioral response minimized the consequences of cutaneous heat loss in psoriasis.

F) CONCLUSION

To recapitulate, the technologies of T. and T.T. have been utilized to show a difference between psoriatic and normal response to cool ambient temperature of 20°C and also to cold challenge by immersion in ice water (0°C). The normal response to low ambient temperature is a drop in temperature of the extremities.

The majority ofpsoriatics did not react at all to a cold challenge. After heat challenge (immersion in 44°C water), there is a lesser difference between psoriatic and normal acco-

modation. Psoriatic skin functions more normally on exposure to high temperature. The thermal measurement technologies open an entirely new perspective on previously a dermatosis of unknown etiology. Psoriasis is characterized by heat loss at the site of the skin lesion, and by incorrect thermal adaptation to changes in ambient temperature, particularly to cold. Psoriasis can be classified as thermoregulatory disorder.

Finally, such a conclusion should open inquiries into the cutaneous role in thermal homeostasis in many skin disorders.

REFERENCES

1. FRENS J.: The influence of skin temperature on thermoregulation in *Medical Thermography. N.* J.M. AARTS, M. GAUTHERIE, E.F. J. RING Eds. Karger Pbl., Basel, *Bibl. Radiol., 6, 218-223, 1975.*
2. GELFANT S.: The cell cycle in psoriasis in reappraisal. *Brit. J. Derm., 95, 577-590, 1976.*
3. GOODWIN P., HAMILTON S., FRY L.: Cell cycle in psoriasis. *Brit. J. Derm., 90, 517-524,* 1974.
4. HARDY J.D.: Models of temuerature reeulation In *Essays on temperature regulations* BLIGH-J., MOORE R.E. Eds. North Holland-American Elsevier, New York, 163-185, 1972.
5. IVANOV D., KONSTANTINOV V., MALOVICHKO W., DANILOVA N., TRUSOVA V.: Physiological mechanisms of skin thermosensitivity. *Progr. Brain Res., 43,* 143-149, 1976.
6. MITCHELL D.: Physical basis of thermoregulation. *M. T.P. international review of science - physiology series* one vol. 7. Environmental physiology, ROBERTSHAW D. Ed., Butterworths, London, University Park Press, Baltimore, Pbls., l-32, 1974.
7. STEINER G.: Biochemical basis and regulation of thermogenesis. In: *Pharmacology of Thermoregulation,* SCHONBAUM E., LOMAX P. Eds., Karger Pbl. Basel, *42-56, 1973.*
8. STOLWIJK J.A., HARDY J.D.: Partitional calorimetric studies of responses of man to thermal transients. *J. Appl. Physiol., 21, 967-977, 1966.*
9. WARSHAW T.G.: Thermal studies in psoriasis. J. *Invest. Derm., 60,* 91-93, 1973.
10. WEINSTEIN G.D., FROST P.: Abnormal cell proliferation in psoriasis. *J. Invest. Derm., 50,254-259,* 1968.

Use of thermography for evaluation and treatment of chronic pain (mainly recurrent headaches)

by P. RUEGSEGGER

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Summary. Since 1961 more than 1000 patients have been treated for various pain patterns, mostly recurrent headaches, resulting in elimination or regression in the majority.[8, 9, 10] Thermography (T.) has been added since 1969 for quantified diagnostic and therapeutic evaluation in parallel with clinical examination in the Author's internistic-cardiological practice. T., after yrs of daily use, is perceived as a remote sensing extension of the clinician's hand, its future seems to lie in the evaluation of chronic pain.

Key words: thermography, evaluation, treatment, recurrent headaches, chronic pain,

A) INTRODUCTION

Thermography (T.) of heat emission patterns of the various clinical disorders encountered in the Author's internistic practice over the past 10 yrs" was found to be most informative in unexplained pain. Since T. is a rapid imaging device of heat emission patterns, it can be used to quantify, to measure objectively

the extent and intensity of local inflammation, heat (calor) being a cardinal sign of it. By the same reasoning, the clinical validity for diagnosis demands logically rigorous interpretation of T. in the context of the remaining cardinal signs in form of swelling (tumor), pain (dolor), redness (rubor) and disordered function (functio laesa) as established from clinical examination and history. And, since the interpretation of T. depends on the presence, or absence of any combination of these cardinal signs, by monitoring the thermal emission of Local Tissue Reaction (L.T.R.) the Author is able to make a distinction between the palpable pain of an inflammatory L.T.R. with higher and the subjective soreness without palpable pain of an ischemic L.T.R. with lower than normal heat emission; and he is able to monitor the healing phase of regenerative L.T.R. after disappearance of swelling and from the heat emission pattern of reactive hyperemia.

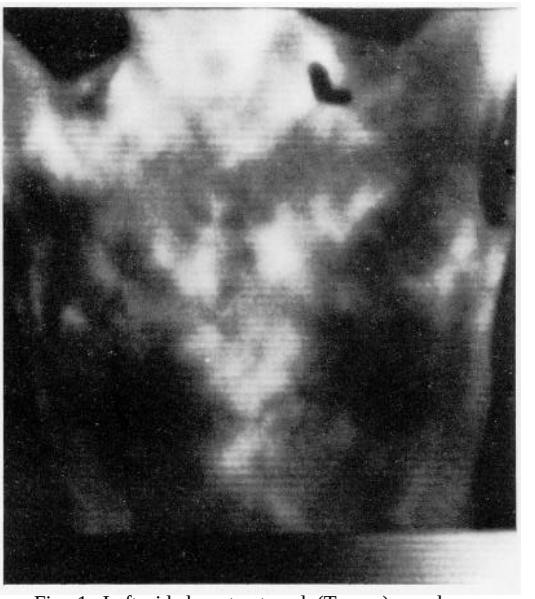

Fig. 1. Left sided costo-sternal (TIETZE) syndrome.

B) THERMOGRAPHY OF PERIPHERAL PAIN PATTERNS

Differential diagnosis of peripheral pain patterns is swift by the unique capbility of visually locating the site and displaying the topography of single and interconnected injury patterns. The pathophysiology of pain patterns of higher complexity will be discussed in the section of headaches. The T. shown were either recorded in the analog mode, ranging from black to white (hot), or simultaneously with isothermic color thermograms (white-hot) for comparison. In the differential diagnosis of pre-cordial pain, Fig. 1 shows a costosternal (TIETZE) syndrome with a focus of increased heat emission at the site of precordial pain in the left third intercostal space. Fig. 2 shows T. of a young car dealer with crushing chest pain one day after he lifted the rear of a car all by himself. There is increased heat emission resembling a flowering tree blossoming up along the sternal border bilaterally into the pectoralis muscle areas, (bilateral rupture of pectoralis muscle fibers: Fig. 2A). Fig. 2B shows regression of healing 14 days later. Fig. 3 shows a case of bursitis of the left shoulder. The clover leaf shaped emission

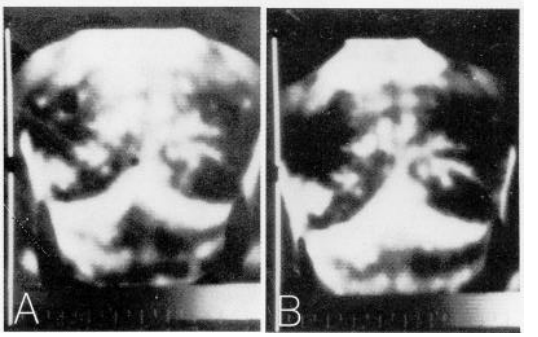

Fig. 2 A-B. Multiple chest wall injuries, in acute stage (A) and during regression (B).

pattern follows the underlying shape of the subacromial bursa distended by inflammation. (Fig. 3A). Clinically, pain extended over entire shoulder with numbness down the arm. Fig. 3B relates the improvement with regression of hyperthermia. A potentially important use of diagnostic T. is demontrated by the serial technique. Fig. 4 shows the evolution of serious back and neck injuries in a steel worker within a 3 month period. Six months prior to the first T. while climbing a ladder, he had rammed his head into a concrete bin with full force so as not to fall to his death. This case is

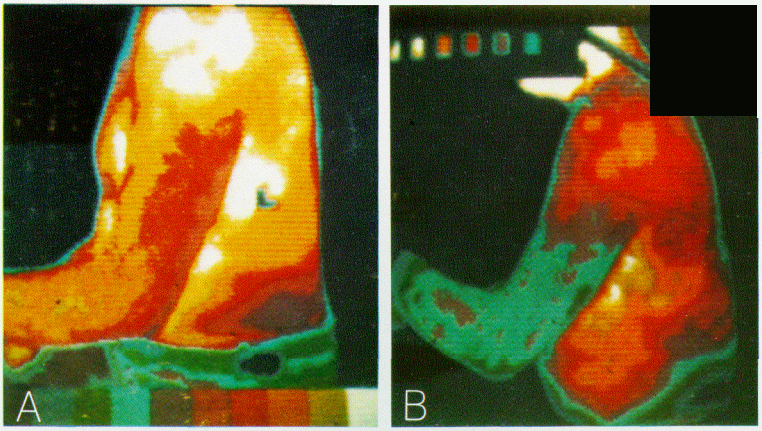

Fig. 3 A-B. A) Acute bursitis of the left subacromial bursa. B) Follow-up after improvement.

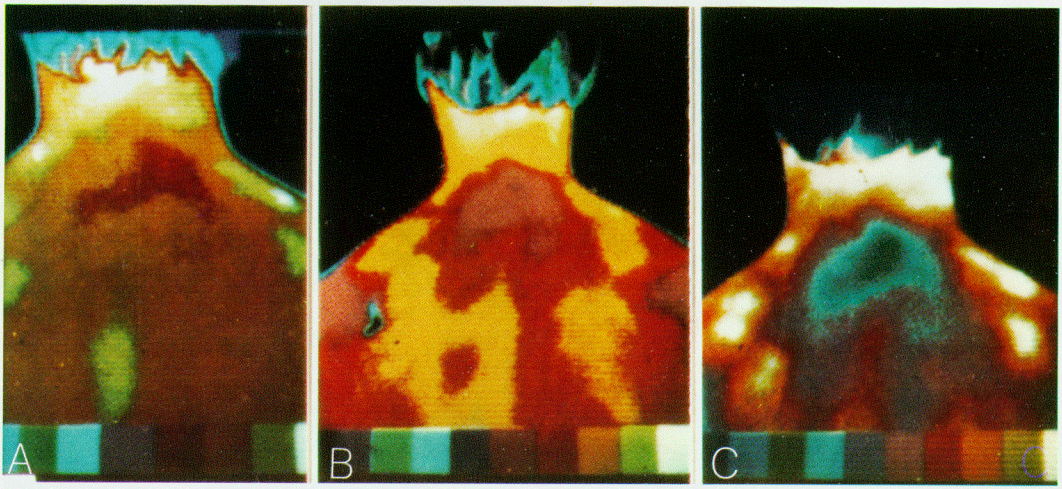

Fig. 4 A-B-C. Multiple head, neck and back injuries. Evolution of abnormal heat emission patterns in serial exams. A) T. on 10/31/1978. B) T. on 11/20/1978. C) T. on 12/11/1978.

remarkable from the legal standpoint **because** this man had lost his disability benefits in the face of negative x-ray evidence prior to T., but regained full compensation after the judge in court saw the T. evidence 3 months later. Reflecting on the problems of clinically accurate interpretation of T. in view of the existing controversary about the validity of T. for mass screening purposes, please note the obvious tact that the T. shown so far were recorded at the time and at the site of clinical examination: The clinical diagnosis of existence of an inflammatory L.T.R. as common denominator to all appears secure because of the presence of 3 cardinal signs (calor, tumor, dolor). Conversely, interpretation without correlation is not secure.

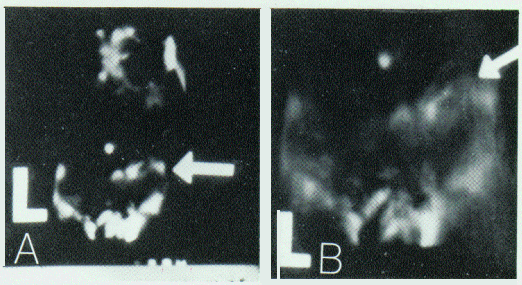

Fig. 5 A-B. Acute appendicitis. T. recorded 2 h prior to operation. Left to right reversal because of mirror recording in the supine position.

Fig. 5 shows heat welling up from the right lower quadrant of a high sitting acute appendicitis, the localization of which was confirmed by operation within hours. Interpretation of

the increased heat emission in these T. is less secure than in the previous pictures. Artifact appears excluded, because deep seated tenderness with overlying surface heat increase has been observed in other cases of appendicitis. This leaves open two possibilities: unless we want to assume that a fountain of increased heat has reached the surface via a vascular shunt which appears topographically unlikely, the locally increased heat emission must be attributed to the vascular response of a viscerocutaneous reflex known to exist.

C) CHRONIC RECURRENT HEADACHES VERSUS OTHERS

1. Observations on chronicity and pattern stability.

In comparison to the peripheral pain patterns just discussed, headache pains are more complex because they are in a different class of perception. Headache pains, by eating into our minds are changing the perception of the world around us. Chronic headache, unlike any other pain, has a conditioning effect on the patient's mind and thus on his behavior by leading to a life of pain-avoidance in a world perceived as essentially hostile.* The triad of headache portraits composed 9 yrs ago[10] charactherized patients with chronic recurrent headaches of 20-58 yrs duration: vascular migraine, muscular tension headaches and neuralgic headaches. The T. patterns of chronic recurrent headaches are similar in all types: marked asymmetry of heat emission, marked hot, marked cold in all (Fig. 6A). Regression and lessening of abnormalities coincided with clinical improvement and gradual disappearance of pain within 4 wks.[10] (Fig. 6B).

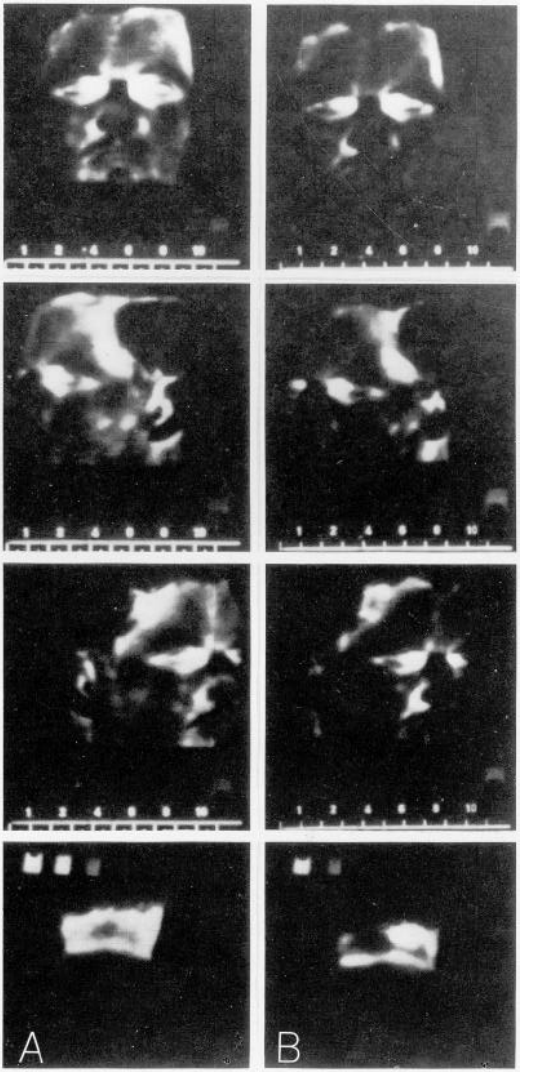

Fig. 6 A-B Chronic recurrent headaches Muscolar tenslon headaches, dally for 30 yrs. A) T pattern before treatment. B) T. pattern after 49 days of local-systemic treatment.

Fig. 7 relate to a patient who complained of headaches day and night with pain all over. These pictures represent a group of patients with agitated depression. Their T. patterns are characterized by a general increase of diffuse heat emission with hyperthermic orbital regions, unlike the asymmetry of patterns observed in chronic recurrent headaches. Our group of patients with such patterns is not large enough to make this distinction statistically valid. Clinically none of these patients had inflammatory tissue reactions and none responded to elimination treatment effective in many other forms.

The advent of T. for mapping of headache patterns emphasizes the importance of minute clinical examination of headache patients even more. Such examination most often, even during the interval between attacks, shows the presence of painful nodularities at stress points around head and neck. Consequently it appeared quite logical to expect T. to have an illuminating effect upon the headache problem.

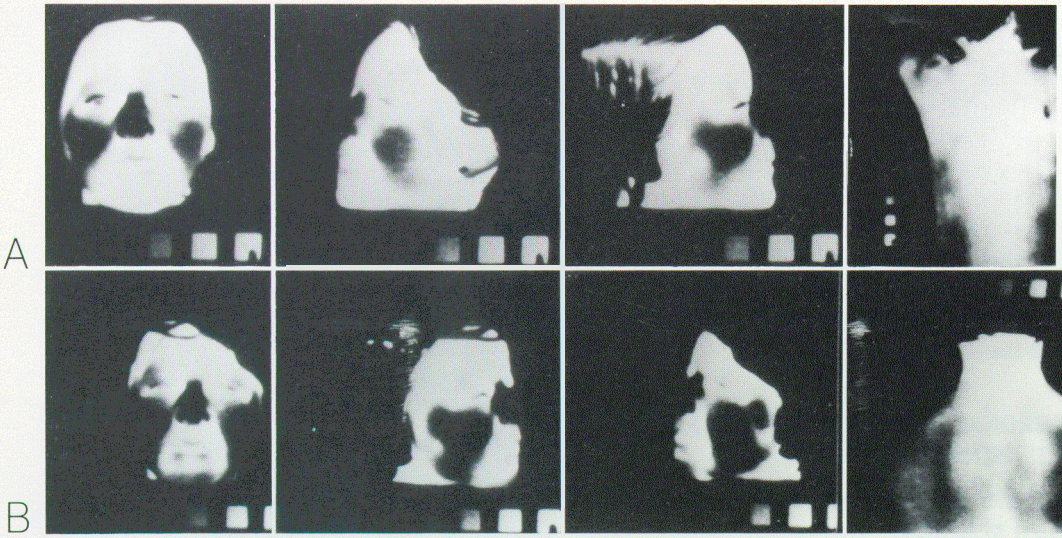

Fig. 7 A-B. Headache of chronic depression. A) Diffuse symmetrical hyperthermia of face with orbital regions and neck on day 1. 1Β) T. pattern essentially unchanged after systemic anti-depressant treatment.

Fig. 8 shows a patient with heat emission oscillating with attacks of cluster headaches every 10 mins. In another case of intractable cluster headaches (Fig. 9) kept awake all night for 6 yrs by crushing pain over the right temple, the T. show ischemic coldness over the right forehead and temple pervaded by linear heat emission probably from branches of the right auriculo-temporal artery. A group of cool round spots is seen over the right temple and forehead consistent with similar signs described in the 1iterature.[3] The colour T. (Fig. 9B) of the same man over a 4 month span from left to right and top to bottom show gradual increase of heat emission over the entire head after daily treatment with nicotinic acid and isoxsuprine. The headaches disappeared completely after the first week of treatment. The patient claims that his previous depression has disappeared and that his memory has improved. After symptom free remission of 6 months patient developed a cerebrovascular accident with transient left-sided hemiparesis, from which he has since recovered. In retrospect, it appears that the hypothermic area over the right forehead was suggestive of an imminent stroke, despite the preceeding clinical improvement. Fig. 1OA Shows the T. of a fanatical tennis player with neuralgic headaches on the left and a cervical syndrome

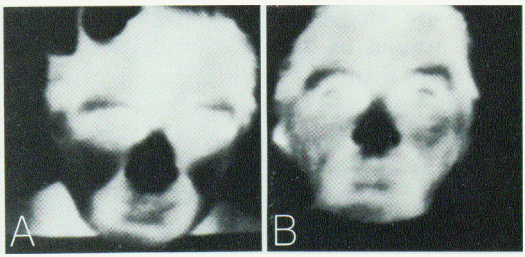

Fig 8 A-B CluSter headaches with mIgraIne type attacks of pain every 10 min from left templw to entire forehead. A) During the attack. hyperthermia of the forehead and coldness of the face in contrast B) Normal heat emission after treatment and cessation of attack.

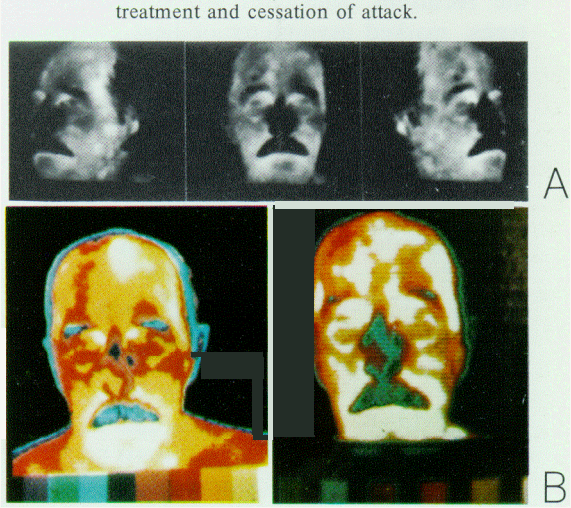

Fig. 9 A-B. B) Cluster headaches right temple, of 6 yrs duration. A) Prior to treatment. B) After treatment.

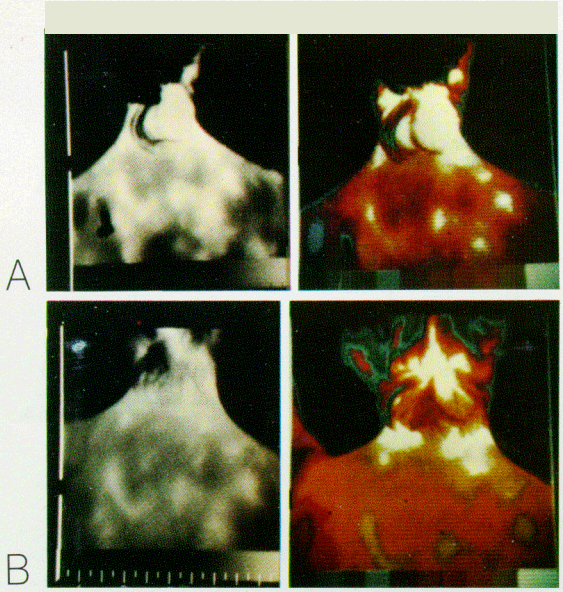

Fig. 10 A-B. Inter-connected pain patterns.

on the right with epicondylitis of the right elbow (not shown). These interconnected pain patterns responded to combined local and systemic treatment within weeks (Fig. 10B). Clinical relapses observed later on as an effect of overexertion and mental strain were promptly reflected by a flare-up in heat emission in the symptomatic regions demonstrating again the close correlation between the T. asymmetry and the clinical picture.

2. The development of headache theories and treatment.

By coincidence 25 yrs ago, while doing basic research on the mechanism of the heart attacks [4,6] the survival of heart muscle after ischemic injury[4,7] and the mechanism of local tissue reactions in general, the Author was confronted with the same problem at the cellular level, which he had met in his headache patients at the clinical level. For reasons of analogy the Author started to search for similar phenomena in headache patients to explain the mechanism of chronic recurrence and the pattern stability of pain in time.[8] Based on past experience with the mechanism of regulation of tissue repair, fibrinolysis and clotting systems it was postulated that the remarkable pattern stability is related to a deranged repair mechanism of tissue injury residing within the patient, a malfunction self-perpetuated by homeostasis.[7,8,9] To arrive at an understanding of the nature of the derangement responsible for chronic recurrence and to find a rational for elimination treatment of this vicious cycle the case of a young woman, age 16, with a history of daily intractable muscular tension headaches since age of 10, is analysed.

On clinical examination her face was pale from general vasoconstriction, the facial features were rigid due to spastic contraction of the entire scalp muscle ring extending to neck and shoulder roofs. And within these swollen areas near the origin of the major occipital nerve on the right and groups of tender nodularities in the lateral trapezius regions bilaterally.

The upper-most T. of Fig. 11A shows the increased heat emission of multifocal inflammation in the right and in the left trapezius region at the site of palpable tender nodularities described before. The neck shows regional hyperthermia with maximal heat emission from the right suboccipital. Oral systemic treatment with combinations of Valium and tanderil for anti-spastic and anti-inflammatory effect resulted in diffuse cooling of the

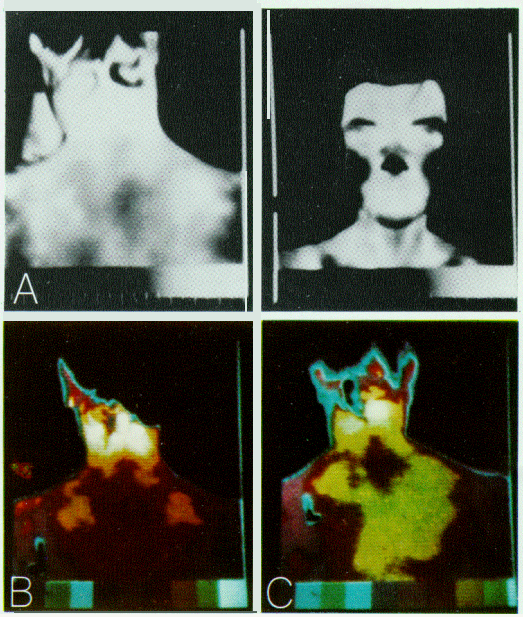

Fig. 11 .A-B-C. Chronic recurrent muscular headaches. A) Day 1. B) Day 14. C) Day 17.

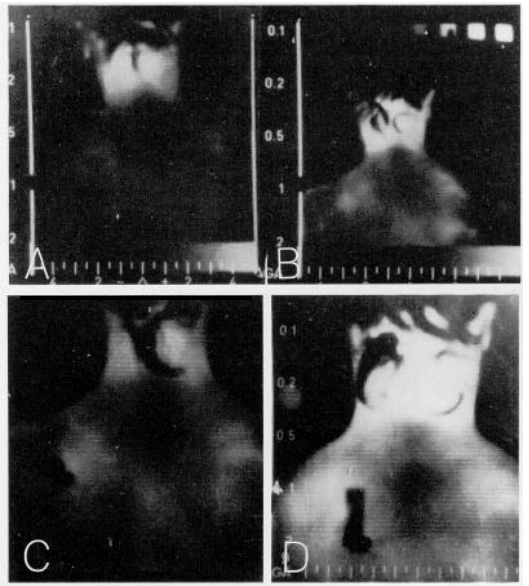

Fig 12 A-B-C-D Same patient as in Fig 11 A) Day 77 B) Day 100. C) Day 152. D) Day 204.

body surface after 14 days (Fig. 11B). Nevertheless, and this is crucial, pain persisted unabated and focal inflammation persisted until local treatment was added on the 14th day. After multiple injections of steroid-procaine mixtures into these focal lesions of inflammation, delineated exactly by T., the daily headaches ceased within 3 wks. And there is a rapid resolution of focal inflammation just 3 days later associated with reactive hyperemia and warming of the entire neck area in the T. of Fig. 11C. Fig. 12 illustrates the fluctuating change of heat emission patterns during clinical resolution of inflammation within 100 days. The increased residual heat emission after 200 days reflects regenerative hyperemia.

D) BASIC RESEARCH CONCEPTS OF TREATMENT RATIONALE FOR HEADACHES

1. Treatment resistant ((penetration blocks»: the observation of cessation of chronic recurrent headaches after direct local treatment of these inflammatory lesions as demonstrated by serial T., suggests the existence of penetration blocks rendering oral therapy ineffective.[8, 10]

2. «Refective headaches»: cybernetic term for self-sustaining headaches due to feedback derangement in muscle system of head and neck. From the cybernetic standpoint the capability for periodic attacks is maintained by triggerspots or regions of persistent inflammation with penetration blocks, which account for the stability of pain patterns, which can be mapped by infra-red heat pictures.[8]

3. Headache classification: a) *simple, self-limiting;* b) *acute or chronic headaches secondaty to known causes* (head trauma, brain tumor, hemorrhage, *metastases,* mal-occlusion etc.); *c) reflective headaches:* all self-sustaining pain patterns of vascular, neuralgic, muscular or mixed types.[8]

4. Present therapy and future prospects: therapy directed at interrupting the feedback loops of headache consists of steroid-procaine injections into regional penetration blocks and/or palpable trigger spots under T. guidance, combined with systemic antispasmodic and anti-inflammatory medication to minimize flare-ups. Experimental evidence suggests future possibile forms of therapy: enzymatic dissolution of penetration blocks by induction of highly fibrinolytic state at the site of excessive microblockade with fibrin deposition. A mathematical model of the kinetics of fibrinolysis has been developed for computer simulation and clinical investigation.[2, 3, 7]

E) DISCUSSION

T. as a heat-sensing extension of the clinician's hand helps to make a distinction between patterns of pain, their site, origin and their possible etiology. Pain, whether inflammatory, ischemic in type or referred from distant disorders along the sensory pathways leading to the brain, may surface in our consciousness and be perceived as a subjective sensation not clearly related to events in the periphery. The palpable pain of local inflammation may hardly register centrally, whereas the soreness of regional ischemia may surface as unbearable subjective pain.

T., by quantifying heat emission of various tissue reactions with clinical correlation, increases the certainty of diagnosis, helps to

unravel complex pain patterns and at last to confirm or disprove the physical nature of subjective pain.[1,5,8] T. is demanding: by measuring components of clinical disorder in parallel with our own physical examination it measures the accuracy of our very own examination. The tendency of this heat-sensing machine of interacting with the examining physician is attested to by the observation of closer agreement between T. measurements and clinical findings in time. Beyond these capabilities as an interacting imaging tool, serial T. tends to measure treatment effectiveness, be it welcomed or not. These effects are admittedly subtle and subject to misinterpretation, but sooner or later the therapeutic choices will be influenced by the cognitive dissonance created. And, by reducing the amount of uncertainty in diagnosis and therapy, the apparent intractability of many forms of chronic pain may yield. The ease of quantifying local tissue reactions and other exothermic processes is expected to induce the development of new forms of treatment, more local, directional and measurable,[1,5] the study of feedback loops and their derangement in the light of local and systematic regulation of heat emission itself.",'*

Author's studies on pain mechanisms suggest the existence of a hierarchy of feedback-regulated mechanism at the molecular level of fibrinolysis [3,7] the cellular level of microblockade[8] the muscle-system level with gamma reflex contractions' and the cerebral level of feedback induced pain-spasm cycle of various forms of headaches. The introduction of cybernetic terminology[11,12] permits linkage of basic feedback studies with clinical pain patterns for which one previously had no vocabulary, such as the periodic recurrence, the relaxation-oscillation of cluster migraine, the run-a-way reflex headaches, or the chronicity of muscle inflammation stabilized by deranged feedback.

REFERENCES

1. CHINC C., WEXLER C.: Peripheral thermographic manifestations of lumbar disc disease. *Appl. Radiol.*, 7, 53-110, 1978.
2. LINIGER W., RUEGSEGGER P.: Fibrinolysis: mathematical and experimental studies. *Thromb. Diath. Haem.*, 17, 412-417, 1967.
3. LINIGER W., RUEGSEGGER P.: A mathematical model of fibrinolysis. *Math. Bioscienc.*, ,1, *263-285*, ,*1967*.
4. NYDICK I., RUEGSEGGER P., BOUVIER C., HUTTER R. V., ABARQUEZ R. E., CLIFFTON E. E., LAUDE J. S.: Salvage of heart muscle by fibrinolytic theraphy after experimental coronary occlusion. Am. *Heart J',*, 61, 83-100, 1961.
5. RING E.F.J., COLLINS A.J.,BAcoN P.A., COSH J.A.: Quantitation of thermography in arthritis using multi-iso-thermal analysis. *Ann. Rheum. Dis., 33, 353-356*, 1974.
6. RUEGSEGGER P., NYDICK I., ABARQUEZ R.E., REICHEL F., CLIFFTON E.E., LAUDE J.S.: Effect of fibrinolytic (plasmin) therapy upon the pathophysiology of myocardial infarction. *Am. J. Cardiol., 6*, 519-524, 1960.
7. RUEGSEGGER P.: Fibrinolytic therapy of myocardial infarction: theory and clinical results. *Angioloqica,* 16, l-8, 1968.
8. RUEGSEGGER P.: Refective headaches. *Headache, 9*, 201-206. 1970.
9. RUEGSEGGER P.: Man and his headaches. *Headache*, 10, 126-130, 1970.
10. RUEGSEGGER P.: Thermographic configuration and distinction of clinical headache varietes. *Proc. internat. headache symoosium Elsinore.* Denmark, Mav 1971. Sandoz Ltd. Pbl. Basle, Switzerland, 1971.
11. WIENER *N.: Cybernetics.* MIT Press, Pbl., Cambridge, Mass (U.S. A.), 1965.
12. YAMAMOTO S.W., BROBECK J.R.: *Physiological controls and regulations.* Saunders Co. Pbl., Philadelphia, (U. S. A.), 1965.

Orbito-frontal thermography: a diagnostic tool in occlusive disease of the carotid artery

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Summary. Thermography (T.) has been recommended as a simple, non-invasive alternative to angiography (A.) in the diagnosis of occlusive disease of the extra-cranial carotid system. The colour T. patterns of the orbito-frontal area with the A. findings have been correlated in 68 patients under investigation for this disease. Internal carotid arterial stenosis of more than 80% was

selected as the level of significant radiological abnormality. Medial orbital T asymmetry of 1°C more was Interpreted as abnormal. T. had a diagnostic sensitivity of 82%, a specificity of 93% and an overall accuracy of 88%. Bilateral significant stenosis usually cannot be Identified by this method. With pre-operative A. and T. baseline studies, T. seems to be a delicately precise tool for post-operative monitoring of patients treated by carotid endarterectomy.

Key words: thermography, carotid artery occlusion, stenosis

A) INTRODUCTION

Angiography (A.) is the definitive diagnostic study for stenotic and occlusive lesions of the extra-cranial carotid arterial system. Thermography (T.) has been recommended as a simple, rapid, non-invasive alternative. The T. patterns of the orbito-frontal area in 68 patients under investigation for carotid disease have been correlated with the A. findings and the accuracy of T. determined.

B) METHODS

All T. were performed with an AGA Thermovision 680 system in a draft free room with constant temperature at 20°C (± 0.5°C). Patients seated before the camera were allowed to cool for 5 mins before T. After satisfactory grey scale and colour T. had been taken with Polaroid film on the 5 and 10 sensitivity scale, digital compression of the superficial temporal a. for 2 mins was used as a provocative test and the T. repeated.

Carotid A. were obtained in all patients (36 bilateral, 32 unilateral). When unilateral examinations only were performed, the contralateral carotid arterial system was assumed to be normal. In 21 patients who underwent carotid endarterectomies, post-operative T. were obtained for comparison. Both the A. and T. were interpreted independently.

1. Grey scale T. were interpreted as abnorma1 if the typical pattern of supra-orbital cooling was apparent on visual mspection. Colour T. were interpreted as normal if there was therma1 symmetry or an asymmetry of not more than 0.5°C in the medial orbital areas (Figs. 1 and 2). Colour T. were interpreted as abnormal if the medial orbital asymmetry of collateral branches from the ipsilateral external carotid system were recorded (Fig. 3).

2. Carotid a. were defined as significantly abnormal if complete occlusion or more than

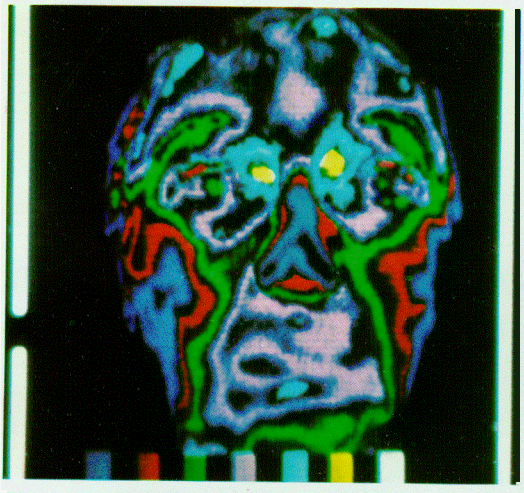


Fig. 1. Normal T. The right and left medial orbital areas (yellow) are thermally symmetrical.

Fig. 2. Normal T. The left medial orbital area (black) is 0.5 0.5°C. cooler than the right (yellow). This thermal asymmetry of 0.5°C has been selected as the upper limit of normal.

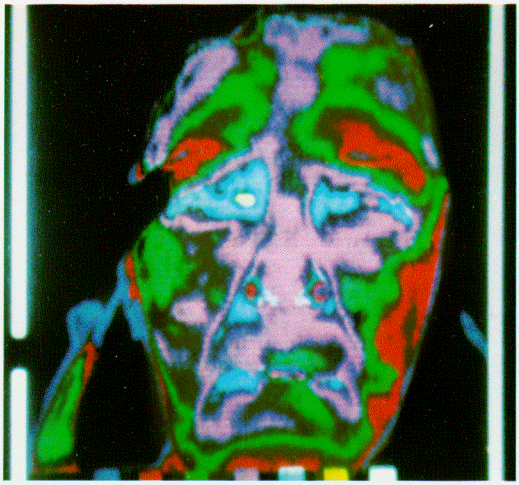


Fig. 3. Abnormal T. The left medial orbital area (blue) is 1°C cooler than the right (yellow). A. demonstrated occlusion of the left internal carotid a.

Tab. I. **Accuracy of thermography in occlusive disease of external carotid system** (>80%): 68 patients with studies angiography and thermography

A \ T	Positive	Negative	Total
Positive	23	**5** (T. false neg.)	**28**
Negative	3 (T. false pos.)	37	40
Total	26	42	68

T. sensitivity: 82% (23/28)
T. specificity: 93% (37/40)
T. accuracy: 88% (60/68)

80% stenosis of the internal carotid a. was demonstrated.

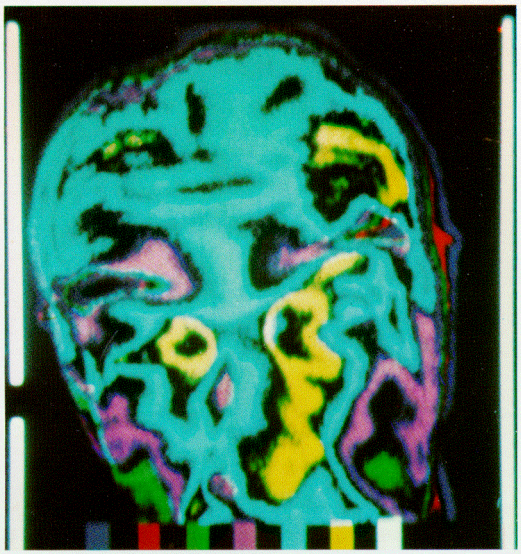
C) RESULTS

Visual interpretation of grey scale T. was found to be so unsatisfactory that they were used mainly as anatomical references for interpretation of the colour T. On the 5 sensitivity scale were more difficult to assess than those on the 10. Based on the thermal asymmetry demonstrated in the medial orbital area, an overall accuracy of 88% was obtained (Tab. I).

D) DISCUSSION

On the recommendation of N euro-surgical Department, greater efforts were concentrated on determing T. changes in patients with A. evidence of complete occlusion or of more than 80% stenosis of the internal carotid a. The one provocative test used (digital compression of the superficial temporal aa.) magnified the thermal asymmetry in some cases but added nothing to the diagnostic accuracy. In contrast to the grey scale T. which were found to be extremely difficult,[1, 3] visual interpretation of the colour Polaroid T. especially at a sensitivity of 10, is simple and rapid.

The thermal pattern of the medial orbital areas usually will be symmetrical and indistinguishable from normal (false negative) in patients with bilateral significant (more than 80%) stenosis of the internal carotid a. They will also be false negative in patients with unilateral significant stenosis who have developed an intracranial collateral blood supply from the contralateral carotid system or from the vertebral basilar system. They will also be normal (false negative) in those patients with unilateral significant stenosis in whom the collateral circulation to the ophthalmic a. has been derived from the ipsilateral external carotid system. However, the increased blood flow through the frontal branch of the superficial temporal a. and/or the angular branch of the internal maxillary a. often produces thermal and anatomical asymmetry of these vessels which enables the examiner to suspect the presence of significant stenosis even though the medial orbital thermal patterns are symmetrical (Fig. 4, A-B). In patients with such false negative initial T., the information available from pre-operative A. studies and thermal patterns make it possible to monitor their post-operative course with quite delicate precision. The T. disappearance of evidence of collateral supply from the ipsilateral frontal branch of the superficial temporal a. or the ipsilateral angular branch of the internal maxillary a. is evidence that the collateral is no longer required to supply the ophthalmic a. after the lumen of the diseased internal carotid a. has been restored. In such patients the symmetrical medial orbital thermal pattern may not change whatsoever.



A

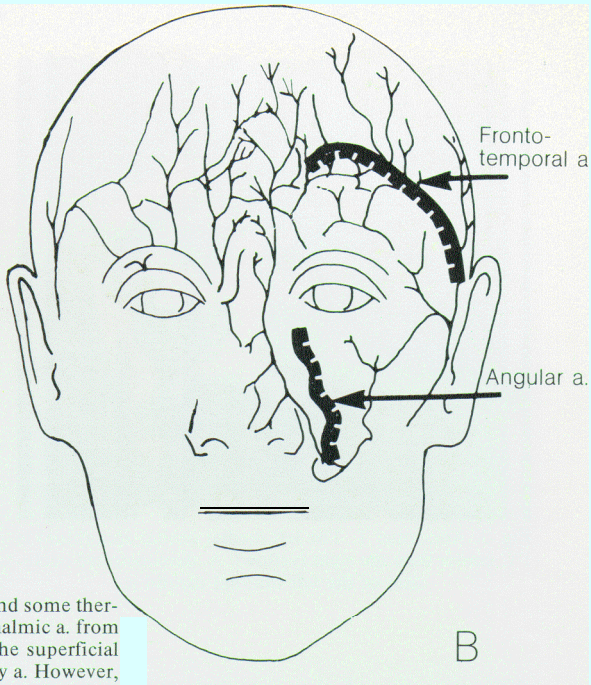


Fig. 4 A-B. A) Abnormal T. There is a marked anatomical and some thermal asymmetry of the collateral circulation to the left ophthalmic a. from the left external carotid a. through the frontal branch of the superficial temporal a. and the angular branch of the internal maxillary a. However, the medial orbital areas are thermally symmetrical (Black). A. demonstrated complete occlusion of the left internal carotid a. B) Diagram of the branches of the left external carotid system identified with T

Twenty-one patients with significant A. evidence of stenosis through their pre-and post-operative course were studied. Twelve demonstrated T. improvement either by a decrease or disappearance of medial orbital asymmetry or by decrease or disapearance of the thermal and anatomical evidence of collateral circulation derived from the ipsilateral external carotid system. Eight demonstrated no change. In 1 patient, a dramatic cooling of the involved medial orbital area with marked increase in thermal asymmetry identified post-operative thrombosis of the internal carotid a.

The wisdom of using T. as a screening test for identification of occlusive disease of the extra-cranial carotid system in an asymptomatic aging population is questionable because many patients with such disease never become symptomatic. To burden them with unnecessary anxiety over a possible future stroke can do more harm than good. At present the most valuable use of T. is in monitoring patients throughout their post-operative course. With the combined information from a T. and a carotid A. as a baseline, long term accurate follow-up of all patients with occlusive disease of the extra-cranial carotid system should be possible with T. alone.*

REFERENCES

1. Capistrant T.D., Gumnit R.J.: Detecting carotid occlusive disease by thermography. *Stroke*, **4**, 57-64, 1973.
2. Correll J.W., Sane P., Quest D.O.: Post-operative thermography in carotid occlusive disease: in *Medical Thermography*, Uematsu S. Ed. Brentwood Publishing Corp., Los Angeles, 99-106, 1976.
3. Wood E.H.: Thermography in the diagnosis of cerebrovascular disease, *Radiology*, **85**, 270-283, 1965.