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Inhaltsverzeichnis (CONTENTS)

Originalien (ORIGINAL ARTICLES)

I.Lamprecht, E.Schmolz, L.Blanco, C.M. Romero
Infrared thermography of a tropical water plant.....91
(Infrarot-Thermographie an einer tropischen Wasserpflanze)

L. Chaerle, F.De Boever, D.Van Der Straeten
Infrared detection of early biotic stress in plants.....100
(Frühe Entdeckung von vitalem Stress bei Pflanzen mit Infrarotthermographie)

P. Melnizky, K.Ammer, O.Rathkolb
Thermographic findings of the lower extremity in Patients with Type II diabetes.....107
(Thermographische Befunde an der unteren Extremität bei Patienten mit Typ II Diabetes)

D.D.Pascoe, B.L. Slaten, R.C.Purohit
Thermal Responses of the Chest Region to Wearing Protective Barrier Suit
During Recovery in a Warm and Neutral Environment.....115
(Temperaturverhalten der Brustregion in der Erholungsphase in einem warmen bzw neutralen Umfeld nach dem Tragen eines Schutzanzugs)

R.C.Purohit, D.D. Pascoe, A. Heath, D.G. Pugh, R.L. Carson,M.G. Riddell, D.F. Wolfe
Thermography: Its role in functional evaluation of mammalian testes and scrotum.....121
(Die Bedeutung der Thermographie zur Beurteilung der Funktion des Hodens und Skrotums von Säugetieren)

Berichte (REPORTS)

Instruktionen für Autoren.....88

Instructions for authors.....90

Graham Machin:
Bryan Chu.....127

Registration form: 9th European Congress of Medical Thermology..... 133

News in Thermology

5th International Congress of Thermology.....128

3rd Asian Pacific Thermology Congress..... 128

QIRT Meeting in Dubrovnik..... 129

AAT Meeting in Orlando.....129

9th European Congress of Medical Thermology.....129

13th THERMO-Conference in Budapest 2003.....130

Veranstaltungen (MEETINGS)

Veranstaltungskalender.....131

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Infrared thermography of a tropical water plant

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Summary

Background: Thermogenic plants (e.g. Araceae, Nymphaeaceae) show increased flower temperatures during their inflorescence to disseminate odours and to attract pollinators. These high temperatures are results of a change to an alternative metabolic pathway and a burst in respiration and heat production. The giant tropical water lily Victoria cruziana belongs to such thermogenic plants and elevates the temperature in the floral chamber to + 10 K against ambient.

Aim of the study: The main body of experiments concerned indirect calorimetric determinations of the metabolic burst during flowering, accompanied by some contact thermometric evaluations. Infrared thermograph was used to receive contact-free temperatures and pictures of temperature distribution in the blossom and on the giant leaves.

Methods: More than 60 Victoria blossoms and an equal number of leaves were investigated in a greenhouse pond of a Botanical Garden. Some of them were photographed by a hand-held, un-cooled micro bolometric infrared (IR) camera (AGEMA 570 PRO) and the pictures processed with the software Irwin 5.0.

Results: IR pictures show maximum temperature increases of about +9 K in the centre of the blossom around the opening to the floral chamber. Other central organs follow with less significant values while the outer parts of the blossom are near to the air temperature. Interesting thermal surface structures were observed in the huge leaves.

Conclusion: IR thermography is an excellent tool in investigations when temperature distributions are essential and not only point values. Thermogenic plants with their strongly increased metabolism are promising objects of research in this field.

Keywords: IR thermography, metabolic bursts, thermogenic plants, thermometry

Thermology international 2002; 12 (3): 91-99

Infrarot-Thermographie an einer tropischen Wasserpflanze

Hintergrund: Thermogene Pflanzen (z.B. Araceen, Nymphaeaceen) steigern ihre Blütentemperaturen während des Aufblühens, um Duftstoffe zu verbreiten und Bestäuber anzulocken. Sie erreichen dieses Ziel durch die Aktivierung eines alternativen Stoffwechselweges, verstärkte Atmung und deutlich erhöhte Wärmeproduktion. Die tropische Wasserlilie Victoria cruziana gehört zu der Gruppe der thermogenen Pflanzen. In der Blütenkammer findet man Temperaturerhöhungen von bis zu +10 K gegenüber der umgebenden Luft.

Ziel der Studie: Die meisten Experimente befassten sich mit der indirekt-kalorimetrischen Bestimmung des Stoffwechsels während des Blühens. Parallel dazu wurden Temperaturmessungen mit Widerstandsthermometern durchgeführt. Die Infrarot-Thermographie sollte dazu dienen, Temperaturen kontaktfrei zu bestimmen und ein Bild der Temperaturverteilung in der Blüte und über die Blätter zu erhalten.

Methodik: Mehr als 60 Victoria-Blüten und ebenso viele Blätter wurden im Gewächshaus-Wasserbecken eines botanischen Gartens untersucht. Einige von ihnen wurden mit einer ungekühlten, mikrobolometrischen Infrarot(IR)-Handkamera (AGEMA 570 PRO) photographiert. Die Aufnahmen wurden mit dem Programm Irwin 5.0 weiterbearbeitet.

Ergebnisse: IR-Bilder zeigen maximale Temperaturerhöhungen von etwa +9 K im Zentrum der Blüte rund um den Eingang zur Kammer. Andere innere Strukturen sind nicht ganz so warm, während die (äußeren) Kelchblätter etwa die Lufttemperaturen aufweisen. Interessante thermische Oberflächenstrukturen sind in den riesigen Blättern zu erkennen.

Schlussfolgerung: IR-Thermographie erweist sich als eine ausgezeichnete Messmethode, wenn nicht nur Punkttemperaturen, sondern Temperaturverteilungen in Pflanzen von Bedeutung sind. Thermogene Pflanzen mit ihrem während der Blüte extrem gesteigerten Stoffwechsel sind vielversprechende Untersuchungsgegenstände.

Schlüsselwörter: IR-Thermographie, Stoffwechsel, Thermogene Pflanzen, Thermometrie

Thermology international 2002; 12(3):91-99

Introduction

Plants are rather unusual objects for thermographic investigations, at least compared with technical, medical or zoological applications. This is due to the large and unfavourable surface to volume ratio of most plant parts, to the strong evaporation necessary for the transport of water within them and to the easy heat dissipation from the plant tissues to the air. Moreover, plant metabolism is in most cases lower in terms of energy turnover as compared to that in animals with their elaborated facilities for locomotor activities. Thus, only few papers on plant thermography are found in the literature: for instance on ice nucleation and propagation in plants (1-3), on plant pathology (4-7), the performance of seedlings (8) or on a new calibration approach for leaf energy balances and stomatal behaviour (9).

But there is a group of plants that is predestined for thermographic investigations: the thermogenic plants. They show a short time dramatic increase of their metabolism during the inflorescence period that was called a "metabolic burst" or "explosion" and a "respiratory flare-up". This burst is accompanied by growth of the floral temperatures with a few degrees, more than 10 K or in some cases even more than 30 K. The blooming period may last only some hours, one or two days, or even several weeks. Some elegant survey papers deal with this interesting plant group (e.g. 10-14). The best-known members in this family are some aroids like "lords-and-ladies" ("Aronstab") *Arum maculatum* and *A. italicum* (15-17), the American skunk cabbage *Symplocarpus foetidus* (18,19) and the frequently investigated voodoo lily *Sauromatum guttatum* (20-23). Besides them the sacred lotus *Nelumbo nucifera* was intensively examined including thermometric and calorimetric methods (12,13,24,25). And of course the present object, the giant tropical water lilies *Victoria amazonica* (in former times called *regia*) and *cruziana* (26-29).

Several reasons are discussed in the literature for the biological sense of the metabolic burst and the strong temperature increase. The first one is the protection of sensitive floral parts during cool periods and the promotion of a quick and successful development. But as many thermogenic plants grow in the tropics where dangerous cold nights are rare, other reasons, connected with an effective pollination, might be more important. The high floral temperatures facilitate the dispersion of different odours that attract pollinators over far distances when optical clues are no longer effective. Moreover, many thermogenic flowers posses floral chambers, which offer an abundance of food and a warm shelter for pollinators during cold nights, serve as attractive mating places and as an ambush against predators (11,13, 19, 30-33).

Victoria cruziana, the smaller one of the two thermogenic tropical water lilies, occupies a mean position in the scale of all known thermogenic plants (34). The metabolic burst endures less than two days and the temperature does not increase more than about 10 K. But its floral chamber is large and a good example for the advantages offered by such plant structures to pollinators. Moreover, *V. cruziana*, the local "Yrupe", flowers near to the water surface with its temperature stabilizing effect so that it differs considerably from all the other thermogenic plants and their local environment. Further incentives were to study plant metabolism near the water surface under even more difficult experimental conditions than for the sacred lotus (24) and perhaps to extend the research later *in situ* at the Amazon.

Material and Methods

Location and plants

All investigations were performed in a greenhouse of the Botanical Garden of the Free University of Berlin. *Victoria cruziana* grew in a 4 x 16 m² pond with a depth of 0.40 m. The water

temperature remained constant at 30 °C throughout the whole season while the air temperature with a nominal value of 24 °C fluctuated considerably during the day because of direct sun irradiation. Therefore, the air temperature was continuously registered in a shaded place and moreover directly at the site of the blossom to determine its microclimate. The relative humidity in the greenhouse was always near to 100 %.

Victoria cruziana, investigated by us, is the slightly smaller sister of the famous *Victoria amazonica* (in former times: *regia*) with its gargantuan floating leaves. But also the leaves of *V. cruzian*a are likewise impressive with their upright rims of 7 cm, a final diameter of 1.50 m and a surface of 1.8 m². Figure 1 shows these huge leaves that resemble a pie case and that are also called "floating tea trays" in their home country. Their thin upper tissue is supported by a radially symmetric, spin-web like system of vertical ribs forming air pockets between them. In an out-grown leaf the air pockets sum up to more than 50 litres and render the astonishing buoyancy that may carry a person of corresponding weight. Nevertheless, leaves show notches in the rim, which allow the rainwater to flow off and not to press the leaf under water (Fig. 1).

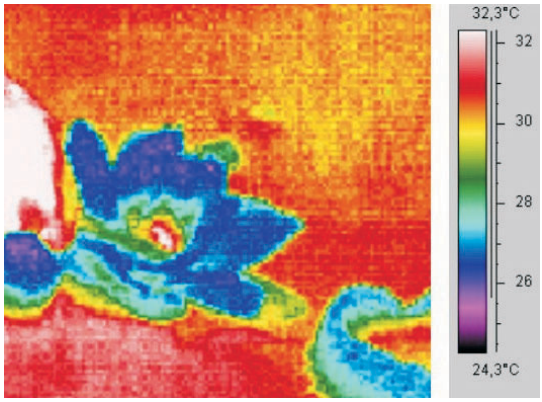
The blossom of *V. cruzian*a develops as a bud under water and later on half emerged on the water surface (Fig. 1). The flowering proper proceeds a few centimetres above the water level within two days. In the first morning the bud slowly opens and shows the white petals between the green sepals, spreads the flower leaves more and more till it reaches the afternoon-state shown in Fig. 1 with the main part of the petals bent down to the water. At this time the colour changes to a pretty pink and the first traces of a sweat attractive odour become detectable. This is the thermogenic period of the blossom when it increases its metabolism drastically and when its floral chamber temperature climbs up for the first time. Due to the show character of the greenhouse, *Victoria* blossoms could not be cut for laboratory experiments but had to be investigated *in situ*, i.e. with thermometers and data loggers floating aside them and performing IR thermography standing in the pond. During summer 2000 about 60 blossoms and a corresponding number of leaves could be analysed in this way.

Thermography

The infrared false-colour thermographic pictures (Figs. 2, 3 and 4) were taken with an un-cooled infrared camera (AGEMA 570 PRO, Darmstadt/Germany). The hand-held camera was so light that photos could be taken in the pond near the flowers and leaves. Its thermal sensitivity amounted to 0.1 K at 30 °C, the spectral range to 7.5 to 13 µm and the size of the focal

Figure. 1
Experimental conditions for the investigation of *Victoria cruziana* blossoms in a greenhouse pond. The blossom is about 10 cm above the water surface. Its diameter measures 25 cm, that of the leaves in the background around 100 cm. In the lower left corner two flower buds are emerging from the water, directly behind the blossom a few days old leaf grows. The high rims of the leaves with the notches for rainwater drainage are clearly indicated.

Figure. 2
False-colour IR photography of a late-afternoon *Victoria* blossom slightly above the water surface. Water and air temperature are 30 and 24 °C, respectively. The maximum temperature in the centre of the blossom is 32.7 °C, the minimum one of the upright petals 25.7 °C. The white area at the left side represents the experimenter's hand with a maximum temperature of 35.3 °C, outside of the scale at the right. For more details see text.



plane array to 320 x 240 pixels. Pictures were further processed by the software Irwin 5.0 that works under Microsoft Windows 95 with different analysis functions like maximum and point temperatures, profiles and histograms. Sometimes it is advantageous to indicate special temperatures in the photography by overlaid values. They are incorporated by software

Figure 3
Out-grown *Victoria* leaf exhibiting the supporting structures discussed in the text. The elevations in the surface have a lowest temperature of 27.0 °C, 3.0 °C above air temperature. The troughs between them are with 29.9 °C near to the water temperature (30.0 °C). For more details see text. Please also notice the thermal reflection of the leaf rim in the background.

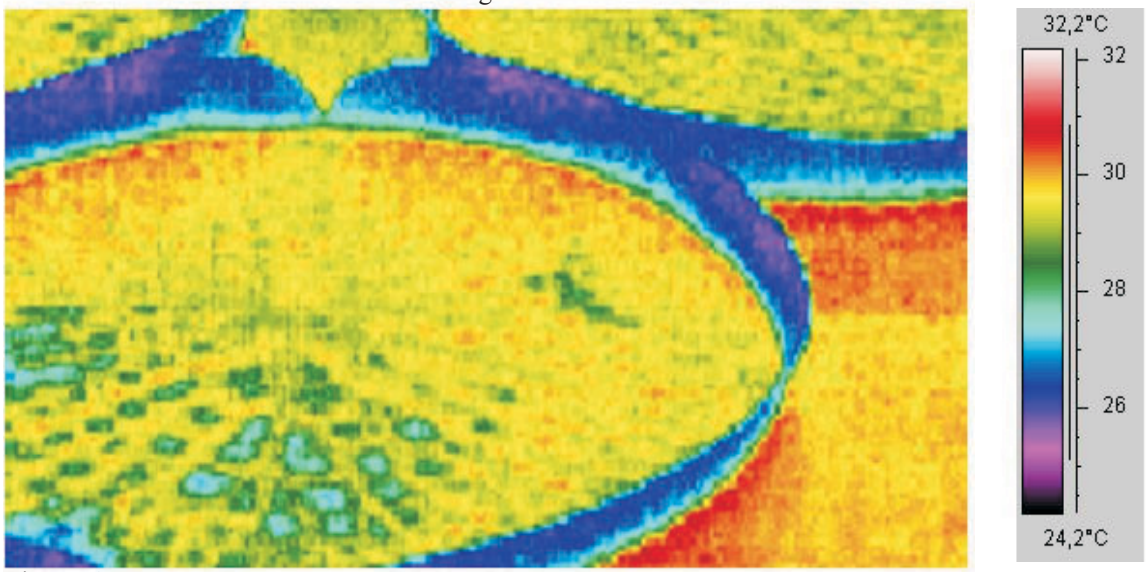
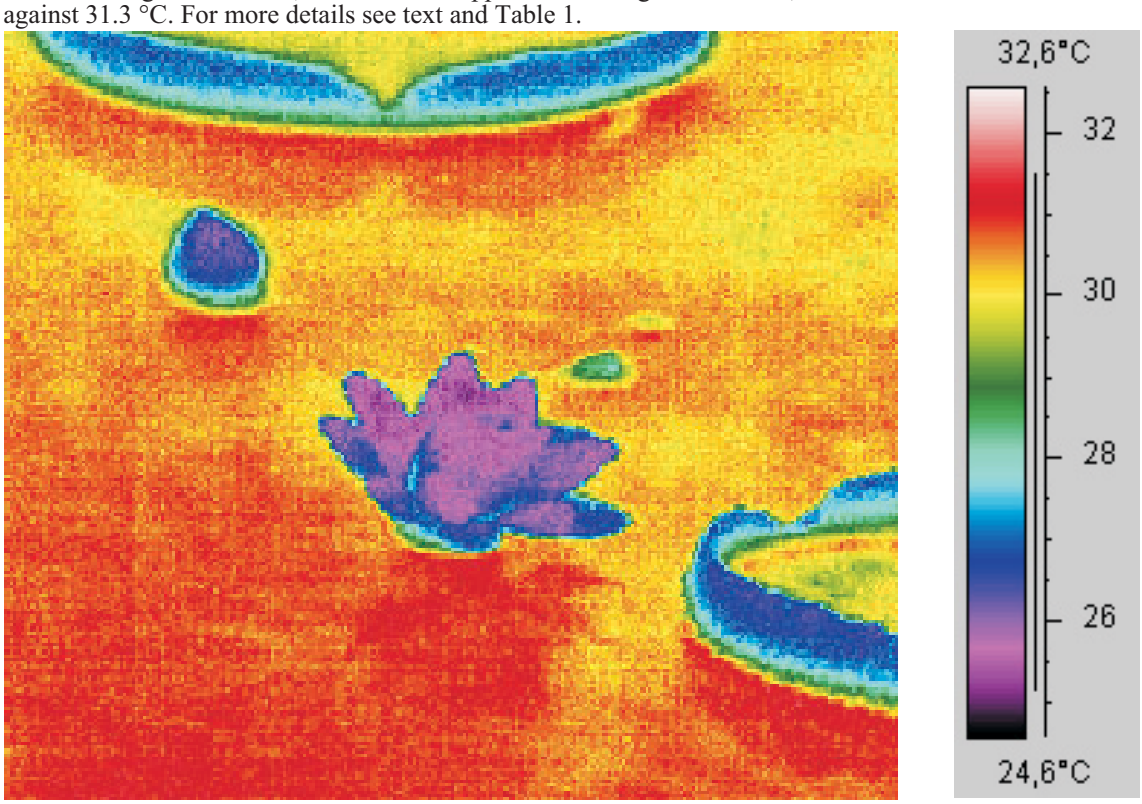


Figure. 4
Thermal reflection of two Victoria leaves (back- and foreground), a bud and a blossom. The coldest parts in the picture (25.0 °C) are the vertical petals of the blossom, their reflection amounts to 30.9 °C, the bud shows 26.9 against 31.1 °C, the rim of the upper leaf 28.0 against 31.0 °C, that of the lower one 27.5 against 31.3 °C. For more details see text and Table 1.



programmes like Adobe PhotoShop 5.5 or MGI Photo Suite 8.06.

A small hand-held IR thermometer (THI-300, Tasco, Japan) with a temperature range from 0 to 300 °C was used in addition to determine point temperatures in a contact-free manner and to look into the floral chamber during the thermogenic phase. A further application concerned the determination of surface temperatures of the *Victoria* leaves, which are less suited for a usual contact thermometry.

Thermometry

As the manual thermographic and IR thermometric measurements were only performed one or two times per day, automatic continuous temperature monitoring accompanied them during the whole period of the metabolic burst. Two contact thermometers (10 kΩ NTC resistors) were inserted into the floral chamber from the top and the side. They were connected to matchbox sized data loggers (HOBO Temp, Series 01 or four-channel HOBO RH, Temp, Light, External, Series 08; software BoxCar 2.06 and BoxCar® Pro 4.0; Onset Computer Corporation, Pocasset, MA/USA) that floated aside the flower on a Styrofoam plate. They stored up to 1800 temperature points between –20 and +70 °C with a resolution of 0.1 K.

Results

Victoria blossoms

The inflorescence programme begins already half a day before the opening of the blossom (35) when the central temperature in the blossom rises one or two degrees above the air temperature. This value is kept constant till to the late afternoon when the plant slowly starts to heat up, usually between 5 and 6 pm. This is the moment depicted in Figure 1 for an overview of the experimental situation and in Figure 2 as a thermographic determination. The blossom is about 10 cm above the water; the green sepals and most of the light pink petals are bent down so that the central stamen and paracarpels are visible in their beautiful deep rose. Touching them one can feel the warmth, even more when one shifts a finger through the paracarpel "tunnel" into the floral chamber. Calorimetric experiments in parallel indicate that at 7:30 pm (± 30 min; mean European Summer Time) the respiration switches over to a significantly increased rate and the "metabolic explosion" starts (36).

Figure 2 illustrates the thermal situation in the late afternoon. The open *Victoria* blossom is bent by the experimenter's hand (white area at the left with about 35 °C) towards the observer to facilitate the view into the floral ground. Stamen and paracarpels, as upper cover of the floral chamber, present the maximum temperature of 32.7 °C, i.e. 8.7 K above the air temperature of 24 °C. Two concentric, slightly cooler rings with 30.5 and 28.3 °C surround the central tunnel. The upright standing petals, more exposed to the air than the central parts of the flower, show a mean temperature of 26.3 and a minimum of 25.7 °C. This is at the same time the absolute minimum in the picture. The parts at the periphery of the blossom and thus nearer to the water or in direct contact with it are at the water temperature, not due to their own metab-

Table 1

Thermal reflection of *Victoria cruziana* at the water surface. Temperature distribution (in °C) in the plant objects and their mirror-images and differences between the means of the two distributions (in K).
(b.g. = background; f.g. = foreground)

Plant Part	Object	Reflection	Mean Difference	Figure
Blossom	26.7 - 27.4	31.4 - 32.3	4.8	2
(Hand)	32.4 - 34.9	32.2 - 32.7	-1.2	2
Leaf rim b.g.	25.8 - 27.6	29.5 - 30.6	3.4	3
Leaf rim f.g.	25.9 - 27.1	30.4 - 30.9	4.2	3
Leaf rim b.g.	27.3 - 28.0	30.5 - 31.0	3.1	4
Leaf rim f.g.	26.5 - 27.5	31.0 - 31.3	4.2	4
Blossom	25.6 - 26.1	30.8 - 31.1	4.1	4
Bud	26.2 - 26.9	30.6 - 31.1	4.3	4
Mean ± s.d.			4.01 ± 0.56	

olism, but due to conductivity. The observed temperature differences are the maximum possible ones by the leaf metabolism. Under the experimental conditions in the greenhouse with about 100 % relative humidity no evaporation and thus no cooling effect occurs that would lower the indicated temperatures.

Victoria leaves

The most impressive parts of this water lily are its huge leaves, especially as they are always visible, unlike the only few hours fully opened beauty of the blossoms (during the night!). Figure 3 shows the false-colour IR picture of a fully-grown leaf of about 1.50 m diameter and an upright rim of 7 cm. It clearly indicates the radial underneath spider-web like supporting construction of ribs and air pockets with their different temperatures. The leaf surface is not completely flat but exhibits small knolls above the pockets and separating troughs between them. The air in the pockets isolates the tissue from the water so that the coolest knolls exist at 27.0 °C while the neighbouring troughs are at 29.9 °C due to the direct contact with the water. The rim is at temperatures between 25.8 and 27.6 °C, the lowest values found in the picture. The notch in the rim for the drainage of rainwater is nicely seen in the false-colours.

Thermal reflection

A specific feature of the IR photography bothered the authors for a while: the thermal reflections of the blossoms, buds and leaf rims (Fig. 3 and 4). One is used to see mirror-images in the visual range in the same colours as the object itself, might-be a bit more intensive or more to grey. But no shift in wavelength and thus in energy occurs (see Fig. 1). That is not, however, the case with false-colour figures. The reflections are shifted to higher temperatures, on average by 3 to 5 K.

The rim temperature of the leaf in the background of Figure 3 varies from 25.8 to 27.6 °C, but its reflection in the water surface between 29.5 and 30.6 °C. The leaf rim in the foreground exhibits corresponding mean values of 26.5 and 30.7 °C, respectively. The mirror-images in Figure 4 are even more impressive. The obtained reflection values are compiled in Table 1 with a mean difference for all of 4.0 ± 0.6 K. The only exception from the upward shift is the reflection of the hand: as its temperature (about 34.5 °C) is above that of the reflecting medium (water, 30 °C), its mirror temperature is moved downward (see Discussion).

Discussion

The data presented in this paper are just "snapshots" of a special moment in the flowering episode of the tropical water lily *Victoria cruziana*. But of a very special one, when the usual plant metabolism switches over to its "energetic burst" and the strong increase in floral temperature. Some tissues of the blossom exhibit values about 9 K above the air (Fig. 2) before the metabolism slows down in the following morning and the differences diminish. During this period pollinators are entrapped in the floral chamber around the fertile female parts until a second, smaller metabolic burst and temperature increase takes place in the afternoon. Now the male parts of the flower become fertile and the tunnel opens again to release the visitors covered with pollen. It is transported to the next fragrant blossom in the female state of development. In the following hours the glamour of the flower disappears, the petals close partly and the blossom is slowly drawn under water (36,37). By the succession of a first female and afterwards male state of the blossom self-fertilization is impeded and cross-pollination supported.

Under usual conditions, 1 mol of NADH that enters the respiratory chain of animal or plant metabolism renders 3 mol of ATP, which serve as energy source for a manifold of physiological processes. When an increase in heat production and thus in body temperature is more important for the organism than chemical energy, respiration and oxidative phosphorylation are uncoupled and only heat is produced, e.g. during arousal from hibernation or in newborn mammals. Thermogenic plants use a similar trick during inflorescence with slightly different means: they change from their usually active cyanide sensitive cytochrome pathway to a less active "alternative, cyanide-resistant" electron transport chain. There they get only 1 mol ATP instead of the normal 3 mol and employ the majority of energy in form of heat to warm up (38,39). In some of the thermogenic plants this shift is triggered by a compound that was called "calorigen" in earlier investigations (40,41) and that turned out to be salicylic acid (42).

It was pointed out in the literature that the metabolic turnover rates of some thermogenic plants compare fairly well with those of mammals or birds of equal size (10,11). Max Kleiber presented his famous allometric "mouse-ele-

phant curve" for the basal metabolism of mammals (between 0.02 and 600 kg) as a logarithmic equation

$$\log M = 1.83 + 0.756 \log W \pm 0.05$$

with M the metabolic rate in "kcal/day" and W the body mass in "kg" (43). Inserting a mean mass of 0.28 kg for a *Victoria* blossom (36), one arrives at 25.8 kcal/day or 1.25 W for the blossom and at a mass specific turnover rate of 4.47 mW g^{-1}. This is significantly higher than the calorimetrically determined rate of 0.61 mW g^{-1} for the entire flower (36). But as the thermogenic active tissue constitutes only a small part of the mass, the calculated value is in the correct order. By chance, Kleiber (43) cites adult female rats of 0.282 kg and a 28.1 kcal/day (4.83 mW g^{-1}) basal metabolism. The stigmatic cup of the *Victoria* blossom – the part below the hottest section of 32.7 °C in Figure 2 – produces 4.79 mW g^{-1} (36), an accidental, but astonishingly nice confirmation of the above statement.

Figures 2, 3 and 4 show the bothering phenomenon of thermal reflection with temperature shifts in the mirror-image. In the daily life we are adapted to the visible range of the electromagnetic spectrum where an object and its reflection are analysed by the colour-sensitive cones in the retina of the human eye or by different film layers in a camera. In contrast to this situation, thermographic false-colour images exhibit the temperature distribution in the field of observation. Planck's law for blackbody radiation leads - by frequency integration over the chosen wavelength range - to the statement that the total energy emission of a body is proportional to the fourth power of its temperature. The total emissivity ε and the Stefan-Boltzmann constant σ appear in this relation, but no longer a special wavelength. Because of energetic reasons, emissivity ε and reflectivity ρ add up to 1 (or 100 %) so that objects with high emissivities have only poor reflecting qualities and *vice versa*.

A thermographic camera receives radiation from three different sources: the main object (in this case: the water of the pond), a second object reflected by the first one (the *Victoria* blossom, bud or leaf rim) and a contribution from the atmosphere (which can be neglected under the experimental conditions). If the emissivity ε_1 of the first object is less than 1 (in the present investigation around 0.95) the second object adds with its own radiance times the reflectivity ρ_1 of the first object (around 0.05). Thus two components instead of usually one are received so that the camera gets the impression of a changed temperature. Whether the mirror-image temperature is shifted up- or down- ward depends on the situation (Table 1): if the reflected object 2 is cooler (the *Victoria* blossom and leaf) or warmer (the hand) than the reflecting object 1 (the water) (29,44,45).

Conclusion

On a first glance thermogenic plants seem just to be one further curiosity of nature, perhaps of some academic interest but without any real utility. Nevertheless, this botanic family shows the ingenious fantasy of nature: the development of sophisticated metabolic pathways, even means of an effective thermoregulation in some of its members (13), heat production rates that equal those of mammals of the same size, and elaborated timing relation with the pollinators and their special demands, and a large variety of optical, olfactory and mechanical tricks to lure, deceive and captivate their pollinators (11).

Victoria cruziana is a positive exception in this family, convincing with a beautiful outlook and a sweet, appealing fragrance and the leaves offering a further attraction. IR thermography is extremely helpful in this case to show the temperature distribution along the flower and the leaves, which is difficult to obtain with usual contact thermometers. As many insects are sensitive also outside the visual range (in the ultraviolet and/or the infrared) it might be that IR pictures like Figure 2 are more similar to the pollinator impression than usual photographs (Figure 1) and thus more conclusive for pollinating strategy investigations.

Acknowledgment

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Infrared detection of early biotic and wound stress in plants

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Summary

When plants are able to recognise an attacking pathogen, they mount a hypersensitive response, characterised by cell death at the site of infection. In certain mutant and transgenic plants, such a process occurs spontaneously. In the model system of resistant tobacco challenged with tobacco mosaic virus, an increase in surface temperature was thermographically observed, localised within the infection sites, before any visual cell death symptoms became apparent. In addition, an increase of leaf surface temperature was visualised immediately after infection. This response was independent of the presence of tobacco mosaic virus, and is caused by abrasion damage during the infection procedure. In conclusion, thermography has a clear potential to visualise early plant leaf responses to wounding and to infections resulting in cell death.

Key words: thermography, robotisation, plant transpiration, pathogen infection, wounding, cell death

Die Entdeckung von frühzeitigen infektiösen und Wund-Stress bei Pflanzen durch Infrarot

Wenn Pflanzen einen pathogenen Angriff entdecken könne, reagieren sie darauf mit einer überschießenden Antwort, die durch den Zelltod im Bereich der Infektion gekennzeichnet ist. Bei bestimmten muntanten und transgenen Pflanzen, kann so ein Prozess spontan auftreten. In einem Modell, bei dem resistente Tabakpflanzen mit Tabak-Mosaik-Virus kontaminiert wurden, konnte an den Kontaminationsstellen ein Anstieg der Oberflächentemperatur thermographisch erfasst werden, ohne dass ein Zelltod beobachtet wurde.Der Temperaturanstieg wart unmittelbar nach der Infektion nachweisbar.Diese Antwort war an die Gegenwart von Tabak-Mosaik-Virus gebunden, sondern ist der Ausdruck der oberflachlichen Schürfverletzung während der Infektion. Zusammenfassend hat die Thermographie ein großes Potential frühzeitige Reaktionen von Pflanzen auf Verletzung und Infektion mit der Konsequenz des Zelltodes sichtbar zu machen.

Schlüsselwörter: Thermographie, Robotisation, Pflanzentranspiration, pathogene Infektion, Verletzung, Zelltod

Thermology international 2002, 12(3): 100-106

Introduction

Based on previous results, demonstrating that spraying of tobacco leaves with salicylic acid (SA) induced an increase in leaf surface temperature (1), we hypothesised that the endogenous production of SA during the hypersensitive response of resistant tobacco to tobacco mosaic virus (TMV) (2) would also lead to an increase in leaf temperature, associated with the infection sites. Moreover, since mutant and transgenic plants in which cell death spontaneously occurs were known to accumulate high levels of SA, we expected to visualise a comparable phenomenon in these plants. The possibility to identify plant tissue committed to cell death (either pathogen-induced or spontaneous) by thermography would enable specific sampling. Early events could then be characterised biochemically and molecular-genetically, before the appearance of the resulting cell death.

The model system resistant tobacco - tobacco mosaic virus (2) was used for thermographic visualisation of a plant - pathogen hypersensitive response. In initial experiments, using a

random infection method and low-resolution thermal pictures, indications for a localised increase in temperature were observed. By optimising the infection method and the measuring conditions, the colocalisation between the thermal effect and the visual cell death symptoms was proved (3). The thermal response was characterised by a presymptomatic appearance of 'hot-spots' at the sites of infection (on average 8h before pinpoint cell-death lesions were visible by eye). A maximal temperature increase of 0.4 °C was measured at the centre of the thermal spots. The expansion of the thermal effect was very rapid, when compared with the visual symptoms. The maximum size of the thermal effect was reached after 2 days, whereas the visual symptoms needed on average 7days to expand to the same final size. When visual symptoms appeared, co-localised regions of lower temperature became visible at the centres of the 'hot-spots' in the thermal images. Cell death is known to be associated with bursting of cells and hence evaporation of their contents, explaining the lower local temperature.

When growing resistant tobacco plants infected with TMV at temperatures above 28°C, virus multiplication is not inhibited (4). When 'shifting' such plants back to 21 °C, a massive resistance response is mounted by the plant, resulting in both more rapid and more extended cell death, when compared with plants that were kept at 21 °C. Temperature-shifted plants displayed a more rapid expansion of the thermal effect and a shorter time span between thermal effect and visual cell death (3).

Two processes were studied that were thought to be responsible for the increase in leaf-surface temperature: respiration and transpiration. SA is known to induce the metabolic upsurge in flowers of thermogenic plants by increasing respiration, mediated by the non-energy conserving alternative respiration pathway (5). At the moment of visual lesion appearance, infected tobacco leaf tissue displayed higher levels of total and alternative respiration than control tissue. However, taking in account the small amount of heat produced and the physical characteristics of a leaf blade (6), metabolism was estimated to account for maximum 1% of the measured temperature increase. In general, metabolism is negligible in the energy balance of a plant leaf; on the other hand, transpiration has a major share in it. Plant leaves have tiny valves called stomata, which optimise CO_2-uptake for photosynthesis and minimise water loss by transpiration. SA is known to induce the closure of these leaf 'pores' in certain species (7). A decrease in transpiration causes a rapid increase in leaf-surface temperature. By using infrared gas analysis (IRGA) equipment, it was proven that the onset of the increase in leaf temperature after TMV-infection of resistant tobacco, as observed by thermography, coincided with a decrease in transpiration (3). The robotised thermography system permitted to visualise the response of several plants, allowing for the specific sampling of plant tissue during the early hypersensitive response. A time-course of accumulation of SA was obtained with a time-resolution of 1h. The start of accumulation of SA correlated with the emergence of the local increase in surface temperature. We therefore concluded that the observed local increase in leaf temperature was mainly due to a decrease in transpiration, presumably caused by the accumulation of SA or other induced compounds.

Tobacco plants transformed with a bacterial proton pump (bacterio-opsin, bO) were described to spontaneously form islands of cell death on their older leaves (8). When thermographically visualising these plants, several other cell-death 'phenotypes' were observed. In the case of isolated flecks, reminiscent of the TMV-infection loci, a thermal effect was visualised before appearance of cell-death. In most cases however, cell death was visualised as a front of higher temperature that moved from the leaf base to the leaf tip, ahead of the expanding cell death (9). When the propagating thermal front reached the measuring chamber of the IRGA-equipment, transpiration started to decrease. When cell death became apparent on the measured leaf area, transpiration increased again. Also in mutant Arabidopsis plants that spontaneously display lesions simulating disease (lsd) (10), a presymptomatic increase in leaf temperature was observed before the cell death lesions became clearly visible.

Methods

Plant material and infection

Tobacco plants (Nicotiana tabacum L.) cv. Xanthi NN (resistant to TMV infection) and cv. SR1 (cultivar susceptible to TMV) were grown in a growth room at 21 ± 1°C with 60 ± 10% RH and under a 16-h/8-h light/dark cycle.

TMV inoculum was prepared from SR1 plants infected with TMV strain U1. Leaf edges and

main veins of the selected leaf from Xanthi NN plants were copied on a parafilm sheet. The desired infection pattern was created by punching holes in the parafilm leaf outline. The parafilm sheet with infection grid was then placed on the leaf and the infection was carried out at each defined site by applying 0.5 µg of fine sand and 0.5µl of inoculum followed by gentle abrasion with a fine glass rod. As a control, mock-inoculation was similarly carried out with ground healthy SR1 tissue. Alternatively, regions between side- veins where infected as a whole by powdering with sand and applying several 0.5µl droplets before abrasion with a bent glass rod (the bent- over part matching approximately the width of the infected area).

After infection, the plants were placed in the custom-built measuring chamber set at 32 °C to allow virus multiplication. After 1 day temperature was decreased to 21 °C to induce the resistance response (temperature shift experiment). Experiments without temperature shift were carried out at 21 °C.

Robotised thermal imaging of plant leaves

At the start of this research project, it proved impossible to carry out reproducible measurements over long periods of time either due to diurnal or climatisation-induced temperature fluctuations. In addition, the resolution of the used FLIR/Agema THV900LW Stirling cooled thermography system proved insufficient to image several attached leaves from different tobacco plants simultaneously. To correlate the thermal signature of the leaf with visual symptoms of the infection, a customer-grade video camera was used. However, a fixed viewpoint combined with a short object distance introduced important parallax-deviations between thermal and video images.

To circumvent these 3 problems, a water-cooled cabinet with built-in Cartesian positioning system for both cameras was designed and installed. A manual teach-in procedure is used to enter the image-capture positions for the thermal camera. The system then captures thermal and video images at fixed intervals of 1 hour for the duration of the plant response to the infection (typically one week).

After each capture cycle, thermal and video images are transferred over a local network, and processed on a Solaris8 Intel workstation. Thermal images are converted into PC bitmap images, with a user-defined region of interest on the leaf surface as a reference. For each studied plant, thermal and video images are combined into a concatenated image, to allow for rapid detection of changes. This visualisation method proved ideal to guide sampling of tobacco tissue for SA-determination, before visual symptoms became apparent (thermography-aided tissue sampling). Each day, concatenated 'overview' images are converted into MPEG image sequences, allowing visualisation of the evolution of the thermal effect.

Results

TMV infection depends on small wounds at the surface of the tobacco leaf. To guarantee successful infection an abrasive as sand or carborundum is commonly used.

In experiments conducted to optimise the localised (spot wise) TMV infection method, the effect of superficial wounding through abrasion was visible as cold, dark regions in the thermal images. While no visual damage was apparent, the low temperature at the infection spots could remain visible up to the time of emergence of the thermal effect due to TMV infection. An example of successful infection with minimal abrasion damage is shown in figure 1.

The upper left panel of Fig. 1 shows a fast increase in temperature 30 minutes after infection, attaining 0.6 – 1 °C above the temperature of the surrounding leaf tissue. This effect reaches a maximum difference of 1.1 – 1.6 °C at 1 hour post infection (right panel on the top row). This effect passed undetected in previous experiments, due to later start-up of the imaging procedure. In both thermal images, the uninfected tissue has a heterogeneous 'patchy' appearance with surface temperature varying from 27.1 to 28.6 °C. Leaf margins and tip show a higher temperature of 28.5 to 30°C. The main vein also has a higher temperature ranging from 28 to 28.8 °C. The spots of higher temperature loose intensity very quickly thereafter. In the lower left panel taken 8 hours after infection, the commonly observed cold spots at the sites of infection are clearly discernable and are 0.3 – 0.4 °C colder than the unaffected tissue. Thereafter the pattern of lower temperature spots disappeared gradually. One day after infection (lower right panel) the leaf tissue between the major veins has again a uniform appearance. Comparing the 'TMV' pattern in the 3 first panels from Fig. 1 with the pattern in

the thermal images from Fig. 2 proves that the early temperature increase, the ensuing temperature decrease and the thermal effect preceding visual damage are precisely in the same place. An early higher-temperature effect due to tissue damage was visualised after both infection and mock infection of leaf areas (see Fig. 3), proving that this immediate thermal signature was due to local wounding. A higher level of abrasive wounding (lower temperature in upper right panel Fig. 3) correlates with a higher level of infection (higher temperature in lower right panel Fig. 3). Additional animations of this experiment are available at the PlantIR website:www.plantgenetics.rug.ac.be/PlantIR/Thermology.

TMV-infected tobacco plants show a faster expanding and more intense thermal effect when first grown at 32ºC and then shifted to 21ºC. The temperature shift was initiated 1 day after infection. Eight hours later, the already described thermal effect has reached its maximum extension and the temperature difference with the surrounding tissue amounts 0.3 – 0.4 °C. (Fig. 2, left panel). On the video image in the top right panel no effect is detectable. Five days after infection (lower panel) the extent of visual damage (necrosis) corresponds with the thermal signature of infection of the upper panel. In the accompanying thermal image, some ongoing necrosis is still visible as dark spots of 20 - 20.3 °C, which was 0.2 – 0.3 °C below the temperature of the unaffected surroundings. The dried regions at the infection sites range from 20.6 – 20.9 °C. At this stage of the resistance response, parts of the leaf blade are bent due to side-vein damage. These changes in orientation are reflected in the temperature distribution over the leaf blade. The upper central part of the leaf has temperatures of 20.9 - 21.1 °C, whereas the lower central part only reaches 20.3 –20.4 °C.

Discussion

The presymptomatic visualisation of TMV infection was proved by co-localisation of the early thermal effect and later necrotic cell death (3). A thermal effect was expected to occur at

Figure 1.
Thermal images of an attached tobacco leaf, infected with TMV using a localised infection method. Higher gray-scale intensity equals higher temperature. Upper left: 30' post infection (pi) an increase in temperature of 0.6 - 1 °C at the infection spots is visible ; Upper right: 1h pi the increase is maximal (1.1 - 1.6 °C); lower left 8h pi the infected loci are colder (0.3 - 0.4 °C) than the surrounding tissue; lower right 24h pi the infected spots are not discernable from the surrounding leaf tissue. The positioning system was programmed to capture three slightly overlapping images of the tobacco leaf. Time-lapse animations of this early response are

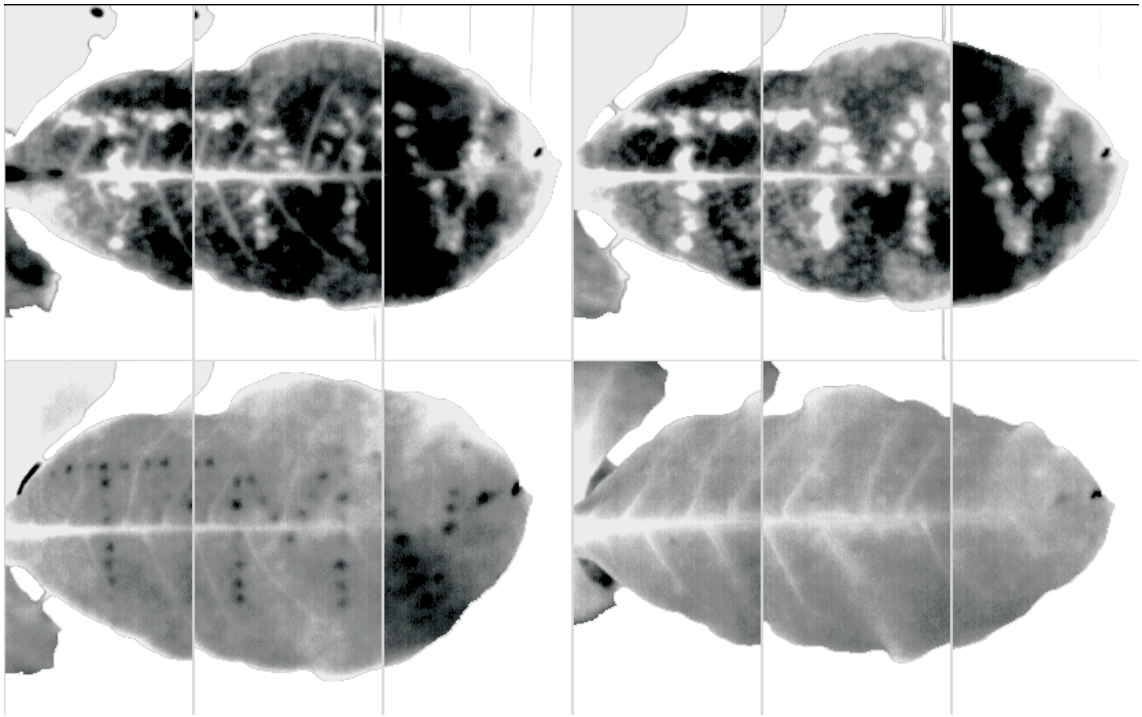


Figure 2.
Thermal (left) and visual spectrum (right) images of an attached tobacco leaf, infected with TMV using a localised infection method. After infection, the plant was grown at 32 °C, and then submitted to a temperature shift to 21 °C. In the upper images captured 8h post temperature shift (pTS), the thermal effect has reached its maximum extension and temperature increase (0.3 - 0.4 °C), while no visual effects are apparent. 5 days after the temperature shift (lower images), the visible pattern of cell death has reached its final extent. Each panel was composed from three adjacent images as shown in figure1. Animations of the whole infection process are available at the plantIR website.

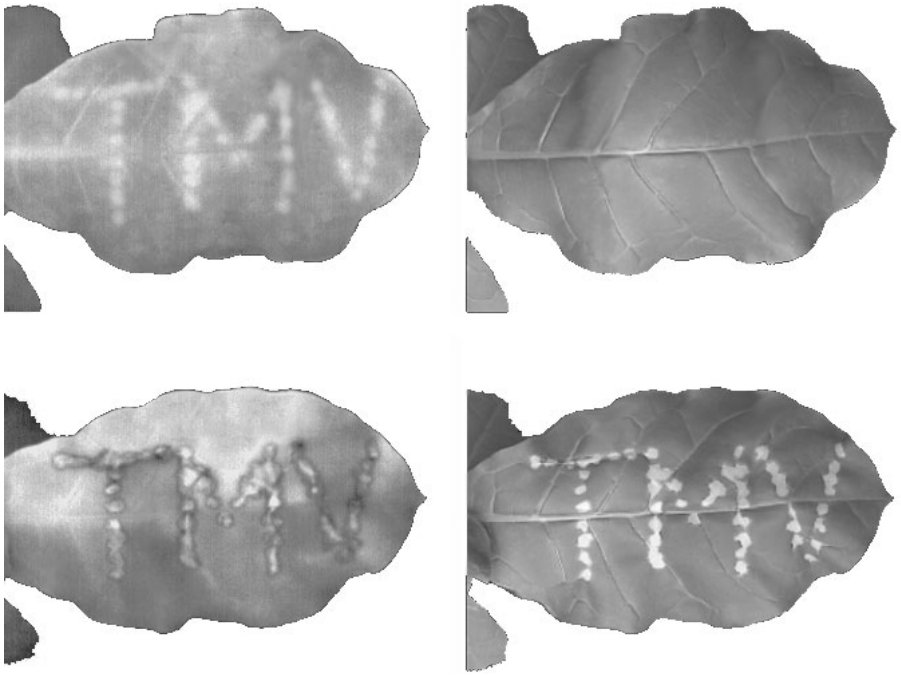
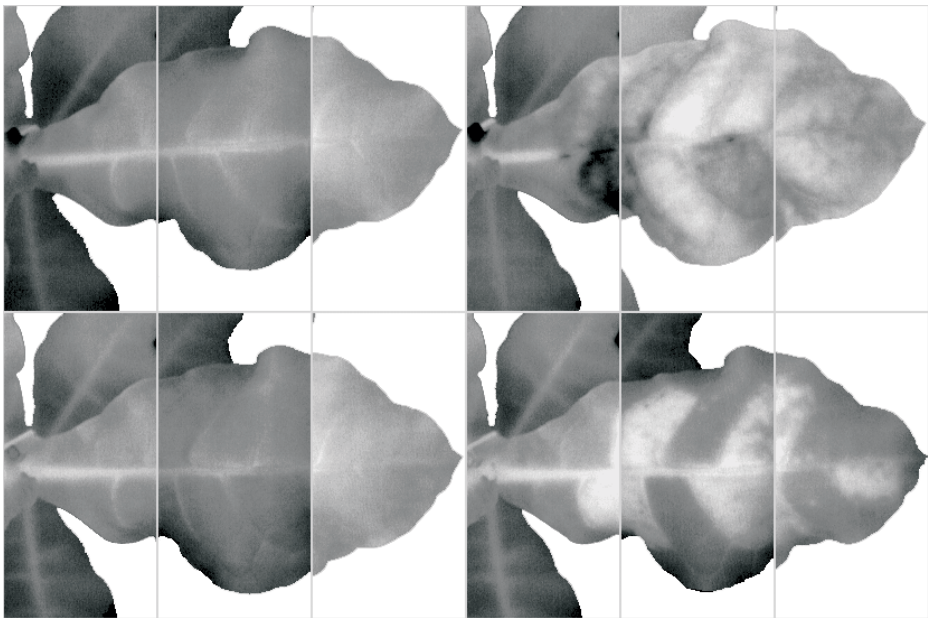


Figure 3.
Thermal images of an attached tobacco leaf, infected with TMV using a region infection method. Infection and imaging were carried out at 21 °C. The upper left image shows the leaf surface temperature distribution 30 minutes before infection (average temperature 21.2 °C). One hour after infection (upper right panel) the untreated basal part of the leaf has an average temperature of 20.8 °C. Some areas have a high temperature (0.5 °C higher), while others are considerably colder (0.4 °C lower). The leaf surface has again a pre-infection appearance 18.5 h post infection (pi), with the same average temperature of 21.2 °C. From the lower right panel (40.5h pi) infected regions can be discerned from mock-infected regions by their elevated surface temperature (0.3 – 0.5 °C difference). Animations of this experiment are available at the plantIR website.



the time of salicylic acid accumulation – as shown previously. In addition to this infection-specific local temperature increase, a local increase in temperature was apparent immediately after infection or mock-infection. The effect was only transient and gradually reverted, resulting in cold spots at the infection sites (Fig. 1). Tissue damage is expected to cause a drop in leaf surface temperature, since the contents of bruised cells will evaporate and locally cool the surface until the wound has dried. An analogous, yet more pronounced phenomenon occurs during pathogen-induced and spontaneous cell death (9). The thermal effect specifically associated with TMV-infection results from stomatal closure and associated decrease in transpirational cooling. Transpiration of regions of attached tobacco leaves was measured using infrared gas analysis (IRGA) equipment. Small measuring chambers, through which air is circulated, are clamped on the leaf. The measured increase in water vapour content indicates the amount of transpiration of the enclosed leaf region. By using this technique the process leading to the temperature changes was clearly identified.

Likely the local increase in leaf surface temperature immediately after infection also results from a decrease in transpiration. The infection method using abrasion probably disturbs stomata at the infection site causing a temporary closure. After a few hours the stomata could start function normally again, allowing the damage at the surface to be detected as smaller lower temperature spots. Since the early temperature increase was thermographically visualised in both infected and mock-infected regions (Fig. 3), a wounding-only explanation is prevalent. Direct transpiration measurements were not attempted immediately after infection since the leaf is still partly moist after rinsing. The plants were infected outside the measuring room at 21 °C, and then placed at 32 °C. As can be seen from the upper 2 thermal images in Fig.1, the uninfected leaf tissue has a heterogeneous appearance due to this temperature change and to the evaporation of residual rinsing water. The infected leaf regains its stable temperature distribution before 8h after infection (compare the upper 2 images in Fig1 with the 2 lower and the thermal images from Fig. 2). As can be seen from Fig. 3, a high level of infection is correlated with the amount of abrasive damage. At levels of abrasion that result in homogeneous infection of leaf areas, no temperature increase immediately after infection can be detected. This suggests that the wounding effect masks the stomatal closure response. The level of surface damage visible at the left in the upper right panel of Fig. 3 does not lead to later visual damage. This can be deduced from the 'recovery' visualised as a gradual disappearance of the temperature difference with the adjacent leaf regions.

Thermography clearly permits early and high-contrast visualisation of wounding, localised infection and cell death. In addition, the robotisation of combined thermal and video imaging allows to visualise the evolution of these responses over time. Given the importance of the cell-death response in the resistance to pathogens, robotised thermography could become a screening tool to search for mutants in the cell death response pathway.

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Thermographic findings of the lower extremity in Patients with Type II diabetes

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Summary

An increase of skin temperature of the feet has been repeatedly reported in patients with diabetes. This indicates that the normal temperature gradient from the knee to the toes was inverted and the forefoot at the same temperature level as the knee or the above knee temperature. However, the reason for this phenomenon is still unclear.

76 patients (29 male, 47 female) who had type II diabetes longer than 5 years were investigated. A physical examination of their feet was performed including range of motion measurement of the ankle joint, recording of the leg axis and the foot arches, toe deformities, skin changes such a callus formation or mycosis, occurrence of varicose veins, neurological examination. Nerve conduction tests were performed for both peroneal and sural nerves. Thermal images were taken from both legs in the anterior view, the foot from the anterior and plantar view.

An inverted temperature gradient was found in 36 patients on the right leg (mean of pathological gradient: -0.27 ± 0.68°K versus –1.84± 0.81°K) in 39 patients on the left leg (-0.77 ±1.15°K versus –1.49 ± 1.21 °K). The temperature readings of the sole (pathological gradient right side: 31.9 ± 1.1 versus 30.1 ± 1.0; pathological gradient left side: 32.1 ± 1.1 versus 30.1 ± 1.1) and the forefoot differed between patients with a normal and a pathological temperature gradient from the knee to the toes significantly. No correlations were found between nerve conduction and temperature measurements.

We could confirm that about half of type II diabetes patients present with a disturbed temperature gradient of the leg, but no clear explanation for this phenomenon was found.

Key words: infrared thermal imaging, type II diabetes, temperature gradient, neuropathy

Thermographische Befunde an den unteren Extremitäten von Typ II Diabetikern

Eine Erhöhung der Hauttemperatur an den Füßen von Diabetikern wurde wiederholt berichtet. Das bedeutet, dass der normale Temperaturgradient von den Knien zu den Zehen invertiert war und sich die Zehentemperatur auf dem gleichen oder einem höheren Niveau als die Knietemperatur befand. Der Grund für dieses Phänomen ist noch unbekannt.

76 Patienten (29 Männer, 47 Frauen), die seit mindestens 5 Jahren an einem Typ II Diabetes erkrankt waren, wurden in die Studie aufgenommen. Die klinische Untersuchung beinhaltete eine ausführliche Beurteilung der Füße mit Bestimmung des Bewegungsumfanges des Sprunggelenkes, der Beinachsen und der Fußgewölbe und eventueller Zehendeformitäten. Außerdem wurden Hautschäden wie Clavusbildungen, Mykosen und eventuelle Varizen registriert und eine neurologische Untersuchung vorgenommen. Die Nervenleitgeschwindigkeit beider Peroneal- und Suralnerven wurde bestimmt. Infrarotthermographien wurde von beiden Beinen von vorne, und von den Füßen von oben und von den Sohlen angefertigt.

Ein invertierter Temperaturgradient wurde bei 36 Patienten am rechten Bein (Mittel des pathologischen Gradienten -0.27 ± 0.68°K versus –1.84± 0.81°K) und bei 39 Patienten am linken Bein gefunden (Mittel des pathologischen Gradienten -0.77 ±1.15°K versus –1.49 ± 1.21 °K). Die Sohlentemperatur (bei pathologischen Gradienten rechts: 31.9 ± 1.1 versus 30.1 ± 1.0; pathologischer Gradient links: 32.1 ± 1.1 versus 30.1 ± 1.1) und die Vorfußtemperatur unterschieden sich signifikant bei Patienten mit pathologischem Gradienten im Vergleich zu normalen Temperaturgradienten vom Knie zum Fuß. Keinerlei Korrelationen wurden zwischen Nervenleitgeschwindigkeit und Temperaturwerten gefunden.

Wir konnten bestätigen, dass etwa die Hälfte der Untersuchten Typ II Diabetiker einen veränderten Temperaturgradienten an den Beinen zeigen, ohne dass eine klare Ursache für diese Veränderung gefunden wurde. Thermology international 2002; 12: 107-114

Schlüsselwörter: Infrarotthermographie, Typ II Diabetes, Temperaturgradient, Neuropathie

Introduction

An increase of skin temperature of the feet has been frequently reported in patients with diabetes [1-4]. Ring had described normal values for the temperature gradients from the knee to the toes [5]. In the case of hot feet this gradient will be inverted with the forefoot at the same temperature level as the knee or the above knee temperature. Autonomic neuropathy [1,2], increased local pressure due to poor footwear [3] and osteomyelitis [4] have been discussed as the reason for this phenomenon. The contribution of autonomic neuropathy is supported by reduced or absent responses of skin temperatures to thermal stimuli [6,7].

Constantly increased HBA1c levels are regarded as a risk factor for the development of neuropathy [8, 9], micro – and macrovascular changes [10,11] and skin ulcers [12]. The American Diabetes Association [13] recommends a goal of <7.0% for HbA1c in patients with type 2 diabetes, and therapeutic action is mandatory in concentrations above 8% .

The objective of our study was to investigate the occurrence of inverted thermal gradients in type II diabetics and to correlate the temperature gradient with the most recent blood glucose and HBA1c level, mal-alignment of the feet, restricted range of motion and nerve conduction measurements. Relationship between hot spots on the feet with callus formation, toe nail onycho- mycosis and foot arch and toe deformities have been reported elsewhere [14].

Method

Patients with Type II- diabetes, who had the onset of the disease at least 5 years ago, and who presented with their HbA1c level higher than 6,5% were recruited from the diabetes out-patient clinic of the Hanusch Hospital. Biographic data (age, gender, onset of diabetes), last recorded HbA1c and blood glucose level and actual drug treatment were recorded of each patient. The range of motion in the upper and lower ankle joint was measured with a gonio-m eter, the alignment of the feet at the subtalar joints, foot arches, toe deformities were investigated and skin changes such as superficial skin lesion, skin ulcers, varicose veins, callus formation and toe nail onychomycosis were recorded.

A clinical neurological examination was performed and light touch and a needle wheel assessed sensory nerve fibres to get information about hypo- or hyperaesthesia and hypo- or hyperalgesia or allodynia respectively. The vibration sense was tested using a tuning fork and the outcome was classified as normal, reduction or loss of vibration induced sensations. The knee and Achilles tendon jerks were also tested. Nerve conduction velocity was studied of both peroneal and sural nerves bilaterally. All studies were performed with surface electrodes for stimulation and registration. Nerve conduction velocity, distal latency and compound motor potentials were determined for the peroneal nerve [15] and antidromic sensory conduction velocity was measured for the sural nerve [16]. These values were classified as either normal or pathological using normal value charts [15, 16].

After acclimatisation for 20 minutes to a room temperature of 24°C thermal images were taken at fixed camera/object distance from both legs (patient standing) from the anterior view. The dorsal feet were imaged from the anterior aspect with the patient sitting on a chair and the feet resting on the floor. Thermal images of the soles were taken with the patient in a supine position on a bed.

Following the outline of the anatomical area, regions of interest were defined over the knee, the ankle, the forefoot, on the dorsal foot and the sole, bilaterally. A negative gradient greater than -1°K was regarded as normal [5] and a temperature difference less than 1°K as pathological finding. In addition, hot spots were defined on the soles as any area at least 0.5° warmer than the surroundings.

SPSS 10.0 for Windows was used for statistical analysis. Mean ± standard deviation (SD) and 95% confidence interval was used to describe the distribution of numerical findings. Due to HbA1c level above or below 8.0% were allocated into two groups and findings in those groups were compared with non-parametric tests. Another allocation was related to normal or pathological thermal gradient on the legs of the patient. Furthermore, clinical findings, results of the electrophysiological tests and of thermal imaging were correlated. A level of significance of $p<0.05$ was used.

Results

Mean age of 76 investigated patients (47 female / 29 male) was 67 ± 10 years. They had suffered an average of 16 ± 9 years from diabe-

tes. 42 patients were insulin dependent diabetics with mean values for HbA1c: 8,7 ± 1% and 163 ± 47 mg/dL for blood glucose.

Table 1 shows the findings after allocation into two groups due to the last HbA1c value. A significant difference of knee temperature was found in patients with HbA1c above 8%. Inversion of the forefoot temperature was significantly less in patients with higher HbA1c values and the motor compound potential of the right peroneal nerve was also significantly lower in this group.

A pathological temperature gradient of the lower leg was found in 36 patients on the right leg (mean of pathological gradient: -0.26 ± 0.68 °K versus –1.84 ± 0.81°K), and in 39 patients on the left leg (-0.77 ± 1.15°K versus –1.49 ± 1,21 °K). All temperature readings of the feet differed

Table 1
Comparison of findings in patients with HbA1c below and above 8,0% MNCV= motor nerve conduction velocity, SNCV= sensory nerve conduction velocity, DL= Distal latency MCP=Motor compound potential

	HbA1c <8.0 % Mean ± SD (95 % CI)	HbA1c ≥ 8.0 % Mean ± SD (95 % CI)	Mann Whitney U-Test 2-tailed p-value
Age (years)	62.5 ± 10.95 (56.4 to 68.5)	67.9 ± 9.54 (65.4 to 70.5)	0.07
Duration of disease (years)	13.5 ± 5.48 (10.5 to 16.6)	16.1 ± 9.27 (13.7 to 18.6)	0.30
Blood glucose (mg/dL)	160 ± 54 (130 to 190)	164 ± 47 (151 to 177)	0.48
HbA1c (%)	7,78 ± 1.0 (7,22 to 8,34)	8.90 ± 0.9 (8,67 to 9.13)	0.00
Plantarflexion right ankle (°)	42 ± 15 (34 to 50)	44 ± 19 (39 to 49)	0.50
Dorsalflexion right ankle (°)	17 ± 14 (10 to 25)	17 ± 16 (13 to 22)	0.74
Plantarflexion left ankle (°)	41 ± 14 (33 to 48)	42 ± 18 (37 to 47)	0.66
Dorsalflexion left ankle (°)	19 ± 12 (12 to 26)	18 ± 14 (14 to 22)	0.58
Eversion right ankle (°)	20 ± 7 (16 to 23)	21 ± 10 (18 to 23)	0.91
Inversion right ankle (°)	48 ± 7 (44 to 52)	41 ± 13 (38 to 45)	**0.04**
Eversion left ankle (°)	25 ± 7 (21 to 29)	24 ± 7 (23 to 26)	0.45
Inversion left ankle (°)	51 ± 9 (46 to 55)	44 ± 13 (40 to 47)	**0.02**
MNCV right peroneal nerve (m/s)	44 ± 4 (42 to 47)	43 ± 5 (42 to 44)	0.53
DL right peroneal nerve (ms)	4.6 ± 1.0 (4.0 to 5.1)	4.5 ± 0.9 (4.3 to 4.7)	0.78
MCP right peroneal nerve (µV)	6093 ± 3147 (4350 to 7836)	4470 ± 3194 (3622 to 5317)	**0.03**
MNCV left peroneal nerve (m/s)	44 ± 3 (42 to 46)	43 ± 5(42 to 45)	0.87
DL left peroneal nerve (ms)	4.2 ± 0.5 (3.9 to 4.5)	4.2 ± 0.8 (4.0 to 4.4)	0.50
MCP left peroneal nerve (µV)	5787 ± 3990 (3577 to7996)	4668 ± 2920 (3893 to 5442)	0.92
SNCV right sural nerve (m/s)	35 ± 16 (27 to 44)	27 ± 20 (21 to 32)	0.68
SNCV left sural nerve (m/s)	36 ± 16 (27 to 45)	28 ± 21 (22 to 33)	0.58
Mean temperature right sole (°C)	30.9 ± 1.6 (30.0 to 31.8)	31.1 ± 1,3 (30.8 to 31.5)	0.76
Mean temperature left sole (°C)	30.8 ± 1,7 (29.9 to 31.7)	31.2 ± 1.4 (30.8 ± 31.6)	0.69
Mean temperature right forefoot (°C)	31.4 ± 1.4 (30.5 to 32.2)	31.7 ± 1.1 (31.4 to 32.0)	0.15
Mean temperature left forefoot (°C)	31.1 ± 1.7 (30.2 to 32.0)	31.7 ± 1.2 (31.4 to 32.1)	0.21
Mean temperature right knee(°C)	31.1 ± 0.8 (30.7to 31.6)	31.8 ± 1.0 (31.5 to 32.0)	**0.01**
Mean temperature left knee(°C)	31.2 ± 1.1 (30.6 to 31.8)	31.8 ± 0.8 (31.6 to 32.0)	**0.02**
Mean temperature right dorsal foot (°C)	30.4 ± 2.0 (29.3 to 31.5)	30.9 ± 1.4 (30.5 ± 31.2)	0.28
Mean temperature left dorsal foot (°C)	30.2 ± 2.0 (29.1 to 31.3)	30.7 ± 1.6 (30.3 to 31.1)	0.39
Gradient right leg (°K)	-0.71 ± 1.61 (-1.6 to 0.2)	-0.92 ± 1.1 (-1,2 to – 0.6)	0.59
Gradient left leg (°K)	-0.99 ± 1.5 (-1.8 to - 0.2)	- 1.1 ± 1.2 (-1.4 to - 0.8)	0.62

Table 2
Comparison of findings in patients with normal and pathological thermal gradients at the right leg

MNCV= motor nerve conduction velocity, SNCV= sensory nerve conduction velocity,
DL= Distal latency MCP=Motor compound potential

	Normal Thermal gradient (<-1.0) Mean ± SD (95 % CI)	Pathologic Thermal gradient (>-1.0) Mean ± SD (95 % CI)	Mann Whitney U-Test 2-tailed p-value
Age (years)	68.4 (65.1 to 71.6)	65.7 (62.3 to 69.1)	0.21
Duration of disease (years)	15.1 (12.4 to 17.7)	15.8 (12.0 to 18.9)	0.87
Blood glucose (mg/dL)	156 (141 to 171)	170 (152 to 187)	0.55
HbA1c (%)	8.7 (8.4 to 9.1)	8.6 (8.2 to 8.9)	0.52
Plantarflexion right ankle (°)	43 (36 to 50)	45 (39 to 50)	0.89
Dorsalflexion right ankle (°)	20 (15 to 26)	14 (9 to 19)	0.03
Eversion right ankle (°)	22 (19 to 26)	18 (16 to 21)	0.06
Inversion right ankle (°)	43 (39 to 47)	41 (37 to 46)	0.67
MNCV right peroneal nerve (m/s)	43 (42 to 45)	43 (41 to 45)	0.89
DL right peroneal nerve (ms)	4.4 (4,2 to4,7)	4.6 (4.3 to 4.9)	0.16
MCP right peroneal nerve (µV)	4744 (3648 to 5841)	4760 (3703 to 5817)	0.78
SNCV right sural nerve (m/s)	24 (16 to 30)	32 (26 to 38)	0.04
Mean temperature right knee(°C)	31.6 (31.4 to 31.9)	31.6 (31.3 to 32.0)	0.51
Mean temperature right sole (°C)	30.2 (29.8 to 30.5)	31.9(31.6 to 32.3)	0.001
Mean temperature right forefoot (°C)	31.0 (30.6 to 30.9)	32.3 (31.9 to 32.6)	0.001
Mean temperature right dorsal foot (°C)	29.83 (29.4 to30.2)	31.7 (31.3 to 32.1)	0.001
Gradient right leg (°K)	-1.8 (-2.1 to -1.6)	-0.26 (-0.2 to 0.25)	0.001

Table 3 Comparison of findings in patients with normal and pathological thermal gradients at the left leg

MNCV= motor nerve conduction velocity, SNCV= sensory nerve conduction velocity,
DL= Distal latency MCP=Motor compound potential

	Normal Thermal gradient (<-1.0) Mean ± SD (95 % CI)	Pathologic Thermal gradient (>-1.0) Mean ± SD (95 % CI)	Mann Whitney U-Test 2-tailed p-value
Age (years)	66.9 (63.6 to 70.2)	66.6 (63.1 to 70.0)	0.80
Duration of disease (years)	14.8 (12.5 to 17.1)	16.3 (13.0 to 19.5)	0.90
Blood glucose (mg/dL)	159 (145 to 174)	165 (148 to 183)	0.77
HbA1c (%)	8.8 (8.4 to 9.1)	8.6 (8.2 to 8.9)	0.50
Plantarflexion left ankle (°)	18 (14 to 23)	18 (13 to 23)	0.34
Dorsalflexion left ankle (°)	44 (38 to 49)	40 (34 to 46)	0.54
Eversion left ankle (°)	25 (23 to 27)	25 (22 to 27)	0.85
Inversion left ankle (°)	48 (44 to 53)	43 (39 to 47)	0.13
MNCV left peroneal nerve (m/s)	42 (41 to 44)	44 (42 to 46)	0.21
DL left peroneal nerve (ms)	4.4 (4.1 to 4.6)	4.1 (3.9 to 4.4)	0.15
MCP left peroneal nerve (µV)	5423 (4531 to 6315)	4012 (2724 to 5300)	0.26
SNCV left sural nerve (m/s)	33 (28 to 39)	23 (15 to 30)	0.94
Mean temperature left knee(°C)	31.8 (31.6 to 32.1)	31.5 (31.2 to 31.9)	0.05
Mean temperature left sole (°C)	30.0 (29.7 to 30.4)	32.1 (31.7 to 32.5)	0.00
Mean temperature left forefoot (°C)	31.9 (31.5 to 32.4)	31.2 (30.7 to 31.6)	0.00
Mean temperature left dorsal foot (°C)	31.1 (30.6 to 31.5)	30.0 (29.4 to 30.6)	0.00
Gradient left leg (°K)	-0.8 (-1.1 to -0.4)	-1.5 (-1.9 to -1.0)	0.00

Table 4
Significant correlations between clinical findings
MNCV= motor nerve conduction velocity, DL= distal latency

Parameter	Correlation coefficient	2-tailed p-value
HbA1c-valueand blood glucose	0.230	0.048
HbA1c-value and eversion left food	-0.235	0.041
HbA1c-value and MNCV right peroneal nerve	-0.302	0.008
HbA1c-value and MNCV left peroneal nerve	-0.234	0.042
Duration of diabetes and plantar flexion left foot	0.235	0.041
Plantar flexion right foot and plantar flexion left foot	0.871	0.000
Dorsal flexion left foot and dorsal flexion left foot	-0.727	0.000
Dorsal flexion right foot and plantar flexion left foot	-0.755	0.000
Dorsal flexion left foot and plantar flexion right foot	-0.716	0.000
Dorsal flexion left foot and dorsal flexion right foot	0.916	0.000
Dorsal flexion right foot and plantar flexion right foot	-0.717	0.000
Dorsal flexion right foot and inversion right foot	0,320	0.005
Dorsal flexion right foot and eversion right foot	0.398	0.000
Plantar flexion right foot and inversion right foot	-0,298	0.009
Plantar flexion right foot and DL right peroneal nerve	-0.238	0.040
Dorsal flexion right foot and DL right peroneal nerve	0.340	0.003
Plantar flexion left foot and DL right peroneal nerve	-0.272	0.018
Dorsal flexion right foot and DL left peroneal nerve	0.340	0.003
Plantar flexion left foot and DL left peroneal nerve	-0.314	0.006

Table 5 Significant correlations between electrophysiologicalmeasurements
MNCV= motor nerve conduction velocity, SNCV= sensory nerve conduction velocity,
DL= Distal latency MCP=Motor compound potential

Parameter	Correlation coefficient	2-tailed p-value
MNCV right peroneal nerve and MNCV left peroneal nerve	0.676	0.000
MNCV right peroneal nerve and DL right peroneal nerve	-0.402	0.000
MNCV right peroneal nerve and MCP right peroneal nerve	0.383	0.001
DL right peroneal nerve and MCP right peroneal nerve	0.383	0.001
DL right peroneal nerve and DL left peroneal nerve	0.585	0.000
DL right peroneal nerve and MCP left peroneal nerve	-0.258	0.025
MNCV right peroneal nerve and DL left peroneal nerve	-0.493	0.000
MNCV right peroneal nerve and MCP left peroneal nerve	0.375	0.001
MNCV left peroneal nerve and DL left peroneal nerve	-0.501	0.000
MNCV left peroneal nerve and MCP left peroneal nerve	-0.298	0.009
MNCV right peroneal nerve and SNCV right sural nerve	0.455	0.000
MNCV right peroneal nerve and SNCV left sural nerve	0.480	0.000
MNCV left peroneal nerve and SNCV left sural nerve	0.393	0.000
DL left peroneal nerve and SNCV right sural nerve	-0.283	0.015
DL left peroneal nerve and SNCV left sural nervee	-0.290	0.012
MCP right peroneal nerve and SNCV right sural nerve	0.439	0.000
MCP right peroneal nerve and SNCV left sural nerve	0.480	0.000
MCP left peroneal nerve and SNCV left sural nerve	0.473	0.000

significantly between patients with normal and pathological temperature gradient from the knee to the toes .

Correlation

A number of weak to strong correlations was found between single clinical findings, single electrophysiological measurements and single temperature measurements. Only weak correlations were obtained between range of motion in the ankle and nerve conduction tests. Weak correlations exist also between HbA1c level, eversion of the right foot, MNCV of both peroneal nerves and, as expected, to recently measured blood glucose level (Table 4).

All electrophysiological measurements showed significant interrelation (Table 5). No correlations were detected between nerve conduction and temperature measurements (Table 6).

Discussion

We were unable to find any correlation between electrophysiological changes typically for neuropathy and elevated skin temperature of the feet. However, there was a weak correlation between restricted motion of the ankle and motor nerve conduction velocity. But it remains unclear whether this finding contributes to elevated skin temperature, because we found in a previous study, that either peroneal palsy or restricted range of motion of the ankle joint was significantly correlated with low skin temperatures of the lower leg [17].

A detailed analysis of hot spots on the feet with callus formation, foot arch and toe deformities [14] could not reveal any relationship. Although radiographic imaging of the feet was not part of our examination protocol, the absence of pain and foot ulcers make undiagnosed

Table 6
Significant correlations between temperature measurements
MNCV= motor nerve conduction velocity, SNCV= sensory nerve conduction velocity,
DL= Distal latency,CP=Motor compound potential

Parameter	Correlation coefficient	2-tailed p-value
Temperature right sole and temperature left sole	0.938	0.000
Temperature right forefoot and temperature right sole	0.816	0.000
Temperature right forefoot and temperature left sole	0.757	0.000
Temperature left forefoot and temperature right sole	0.787	0.000
Temperature left forefoot and temperature left sole	0.846	0.000
Temperature right knee and temperature right sole	0.500	0.000
Temperature right knee and temperature left sole	0.520	0.000
Temperature right knee and temperature right foot	0.613	0.000
Temperature right knee and temperature left knee	0.867	0.000
Temperature right knee and temperature right sole	0.500	0.000
Temperature right knee and temperature left sole	0.520	0.000
Temperature right foot and temperature right sole	0.837	0.000
Temperature right foot and temperature left sole	0.776	0.000
Temperature left knee and temperature right sole	0.541	0.000
Temperature left knee and temperature left sole	0.536	0.000
Temperature left foot and temperature right sole	0.823	0.000
Temperature left knee and temperature right forefoot	0.665	0.000
Temperature left knee and temperature left forefoot	0.643	0.000
Temperature left knee and temperature right foot	0.705	0.000
Temperature left knee and temperature left foot	0.682	0.000
Temperature left foot and temperature left forefoot	0.905	0.000
Temperature left foot and temperature right forefoot	0.834	0.000
Temperature left foot and temperature right foot	0.905	0.000

osteo- mylitis as the reason for the elevated skin temperature of the foot highly unlikely.

Conventional nerve conduction testing cannot assess the function of the autonomic nerve system. However, a comprehensive asssessment of autonomic nerve fibers can only be achieved by the combined testing for sympathetic skin reflex and RR interval variation [18]. Because the latter method is not applicable in the age above 70years, we did not include the evaluation of autonomic nerve fibers in our examination protocol.

High skin temperature is highly correlated with dilated skin microvasculature and a high blood pool in the superficial skin layer. These vessels and also the sweat glands are under the control of the autonomic nerve system. Therefore increased skin temperature was often interpreted as loss of sympathetic nerve function. Any reduction in sweat production, reported as an early sign of autonomic dysfunction in type 1 diabetics [19], may result in high skin temperature in the same way as the loss of vasoconstriction. This is obvious in the case of fully dissected peripheral nerves [20], but might not be the case in metabolic disorders, when adrenergic nerve transmitters [21] and short acting metabolites such as NO [22, 23] interact with glucose induced structural changes of the vessel wall [24] and the endothelium cells [25] in the control of the blood vessel width.

In addition, macroangiopathy can contribute to skin temperature, because low perfusion will be followed by low skin temperature even in the case of fully dilated microvessels. Thermal imaging has been successfully used for the determination of the level of amputation in diabetics with occlusive angiopathy [26]. However, none of the patients investigated complained of any symptoms typically for macroangiopathy.

In conclusion, we could confirm that about half of type 2 diabetes patients present with a disturbed temperature gradient of the leg. This phenomenon might be caused by dilation of superficial skin vessels, but no clear cause for that condition was detected.

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Thermal Responses of the Chest Region to Wearing Protective Barrier Suit During Recovery in a Warm and Neutral Environment

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Summary

The purpose oft his investigation was to examine the mean chest skin temperatures during warm and neutral environment recovery after wearing a protective barrier suit (PBS) during a work task. Eight male subjects completed four trials: PBS/ warm recovery, NO PBS/ warm recovery, PBS/Neutral Recovery, NO PBS/Neutral Recovery. The work task consisted of walking on a treadmill for 30 minutes (1.65 m/s, 6% grade) in a heated chamber (30°C, 50% relative humidity) while wearing either running shorts or a Tyvec™ Protective Barrier Suit. The environmental conditions during recovery were neutral room (25°C, 50% humidity) or warm (30°C, 50% relative humidity) for 30 minutes. Recovery temperatures were obtained using a Bales TIP 50™ infrared thermographic processor. Core temperatures were obtained using a rectal probe. On removing the PBS, a decrease in temperatures (0.57°C) of the chest region indicative of cutaneous vasoconstriction was observed. This decrease in mean skin temperature was most prominent in the neutral/PBS trial recovery in which an elevation of the core temperature coincided with the vasoconstriction of cutaneous blood flow to the chest region.

Key words: Thermoregulation, cutaneous blood flow, heat stress

Temperaturverhalten der Brustregion in der Erholungsphase in einem warmen bzw. neutralen Umfeld nach dem Tragen eines Schutzanzugs

Das Ziel der Untersuchung war die Erfassung der mittleren Temperatur des Brustkorbes während der Erholung in einer warmen oder einer Temperatur neutralen Umgebung, nachdem eine definierte Arbeit bei gleichzeitigem Tragen eines Schutzanzuges geleistet worden war. Acht männliche Probanden führten jeweils folgende Versuche durch: Arbeit im Schutzanzug/Erholung in warmer Umgebung, Arbeit ohne Schutzanzug/Erholung in warmer Umgebung, Arbeit im Schutzanzug/Erholung in Temperatur neutraler Umgebung und Arbeit ohne Schutzanzug/Erholung in Temperatur neutraler Umgebung. Die Arbeitsleistung war 30 minütiges Gehen auf einem Laufband(1.65 m/s., 6% Steigung) in warmer Umgebung (30°C, 50% relative Feuchte), wobei entweder kurze Hosen oder ein Tyvec™ Schutzanzug getragen wurde. Die Umfeldbedingungen während der Erholung waren entweder als thermoneutral (25°C, 50% rel. Feuchte) oder warm (30°C, 50% rel.Feuchte) definiert. Die Oberflächentemperaturen wurden in der Erholungsphase mit einer Bales TIP 50™ Infrarot-Kamera erhoben; die Kerntemperatur wurde mit einer Rektalsonde gemessen. Nach Ablegen des Schutzanzuges wurde am Brustkorb eine Verringerung der Temperatur (0.57°C) als Ausdruck einer Vasokonstiktion beobachtet. Diese Abkühlung war besonders ausgeprägt in der Versuchsanordnung "Arbeit im Schutzanzug/Erholung in Temperatur neutraler Umgebung", wobei gleichzeitig mit der vasokonstriktorisch bedingten Verminderung der Hautdurchblutung im Bereich des Thorax eine Erhöhung der Kerntemperatur beobachtet wurde.

Schlüsselwörter: Thermoregulation, Hautdurchblutung,Wärmebelastung

Thermology international 2002; 12(3):115-120

Introduction

The ability or inability to thermoregulate our core temperature can have dire consequences on our ability for survival or capability to perform thermally challenging work tasks. Approximately 75% of all energy expended during work is in the form of heat. Reducing the

potential for heat storage is accomplished through a favorable balance between heat production and heat boss. Our bodies dissipate heat through radiation, conduction, convection, and evaporation. At rest, in a comfort- able environment, 60% of the heat boss can occur via radiation, as radiant heat moves down the thermal gradient towards a cooler heat source (1). Clothing provides an insula- tive barrier that serves as a protective covering and often impedes heat loss which results in greater heat storage.

During exercise, the major means of dissipating heat shifts to evaporative cooling. Again, clothing can impede this cooling process by creating a micro-environment between the clothing and skin in which the temperature and moisture may rise to the point where evaporation is severely impaired (2). Similar to clothing, protective barrier suits further isolate the individual from the external environment. This may be potentially harmful to the worker if body heat cannot be adequately dissipated. Protective barrier materials are avail- able along a continuum that provides increasing levels of protection. However, the more effective these barrier suits are in isolating individuals from the external environment, the greater the potential for heat gain and heat storage in the micro-environment created between the protective barrier suit and the body. With an increase in body core temperature there is an increased risk of heat injury (heat illness, heat exhaustion, heat stroke). Therefore, the individual who wears a protective barrier outfit is subjected to both a threat from the outside and from the heat created within.

Numerous types of protective barrier suits are currently used by firefighters, pilots and astronauts, oil rig workers, hazardous waste workers, metal/glass foundry workers, body armor, infectious material and medical isolation suits, military combat gear for biological and chemical warfare, etc. The assault of the external environment can be a thermal (hot or cold), radiological (UV, X-ray, etc.), biological threat (bacteria, germs, disease, agricultural insecticides, manufacturing chemicals). In essence, everything you wear between your skin and the environment, including daily clothing, can be considered a protective barrier outfit.

To compensate for these thermal problems, research has been directed towards determining appropriate work/rest ratios and the potential monitoring of individuals using either skin or core temperature. In this investigation, skin and core temperature will be measured throughout a 30 minute work cycle in a warm environment (30°C, 50% relative humidity) with and without wearing protective barrier suit (PBS) and during a 30 minute passive recovery in a thermal neutral (25°C, 50% relative humidity) and warm environment (30°C, 50% relative humidity) without the PBS.

Methods

Eight healthy male (21-48 years) recreationally fit subjects participated in this research project after signing a University approved informed consent. Each subject completed four trials in a random order: NO PBS/Warm recovery, NO PBS/Neutral Recovery, PBS/ Warm Recovery, and PBS/Neutral Recovery. At least 24 hours were allowed between each trial. During the PBS trials, the subjects wore a protective barrier suit (Tyvec™) which covered their legs, chest, arms, and head with a hood. In the NO PBS trials the subjects wore running shorts and no shirt. Before participating in any trial, the subjects were requested to drink at least one liter of water 1 1/2 hours prior to the exercise bout in an attempt to have the subjects euhydrate. No water was provided during the trial or recovery.

Prior to exercise bouts, the subjects dressed in their running shorts stood in a thermo- neutral room (25°C, 50% humidity) for at least 15 minutes to allow for equilibration ofs kin temperatures of the chest region. After equilibration, a pre-exercise thermal image (Bales Thermal Imaging Processor 50™) was obtained. Pre-exercise images were com- pared to images obtained during recovery. Prior to imaging in the post exercise conditions, sweat was removed by softly pressing a towel against the skin surface area. During the exercise trials, core temperature (rectal) and suit temperature and humidity were recorded via thermistors and a data logger (Grant 1250™). The subjects walked for 30 minutes (1.65 m/s, 6% grade) on a treadmill in a heated chamber (30°C, 50% relative humidity). Upon completing the work task, the subjects removed the protective barrier suit and remained standing in their running shorts for 30 minutes while chest skin surface temperatures were obtained by electronic infrared thermography. During this same time period, core temperature was continuously recorded.

Data Analysis

The data were analyzed using a repeated measures ANOVA in which the conditions (PBS, NO PBS) were compared over time (Pre-trial, immediate post exercise, 3,6, 9, 12, 15, 18, 21, 24, 27, 30 minutes post exercise). The level of significance was set at $p<0.05$. Significant differences between multiple comparisons were determined using a Tukey post hoc analysis.

Results

The work environment for the exercise bouts was kept constant at 30°C, 50% relative humidity. However, during the trials in which the subjects wore the PBS, the micro-environment between the skin and barrier suit demonstrated a significant increase in temperature and humidity when compared to the environmental testing conditions (Table 1).

Table 1
Work/Rest temperatures within a Protective Barrier Suit in the Heat Chamber (30°C, *50%* relative humidity)

	Pre Trial	Post Trial
Temperature within Suit	33.10°C	34.90°C
Humidity within Suit	78.2%	98.1%

Thermograms of the mean skin temperature (32.05 ± 0.05°C) and rectal core temperatures (37.2 ± 0.2°C)) were not significantly different for the two treatment groups in the pre-exercise analysis. In the post trial analysis, the rectal temperature in the Neutral/No PBS trial was significantly different when compared to Warm/ PBS, Neutral/PBS, and Warm/ NO PBS. This significant difference remained throughout the 30 minutes recovery for both PBS trials (Warm and Cool), but only for the first 15 minutes comparison with the Warm/NO PBS trial (Figure 1). Only in the cool PBS trial demonstrated a rise in the core temperature during the first five minutes ofthe recovery. After 30 minutes of recovery, the subjects were able to return the core temperature back to a pre-exercise value only during the NO PBS trials.

The mean skin temperatures immediately post exercise were significantly different for the warm (32.27 ± 0.16 °C) versus neutral recovery (31.64 + 0.10°C) conditions only during the initial three minutes. Thereafter, only the mean skin temperature for the neutral/PBS trial remained significantly lower than the other trials during recovery from 6 to 21 minutes. Mean skin temperature never returned to a pre-exercise value during neutral/PBS trial.

Figure 1
Rectal temperature response during recovery

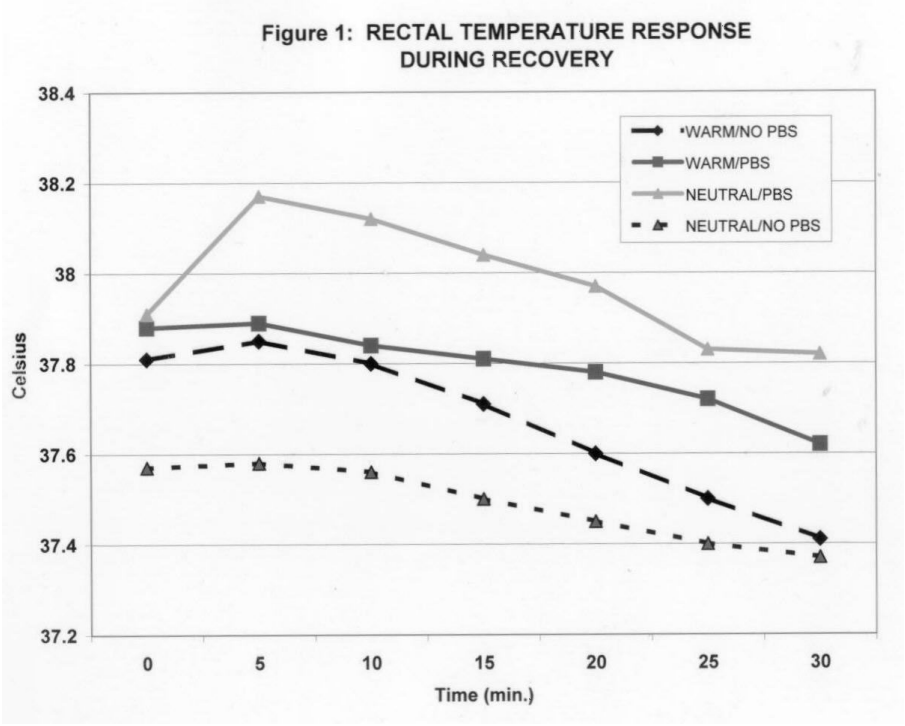


Figure 2
Mean skin temperature during recovery

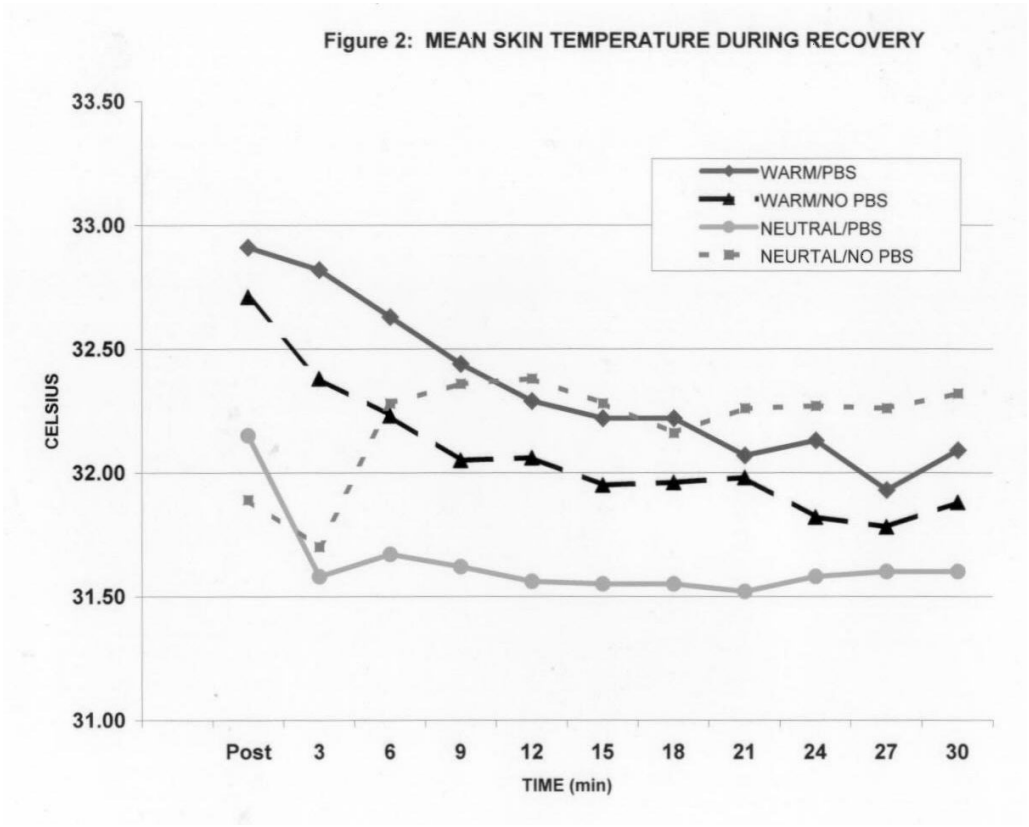
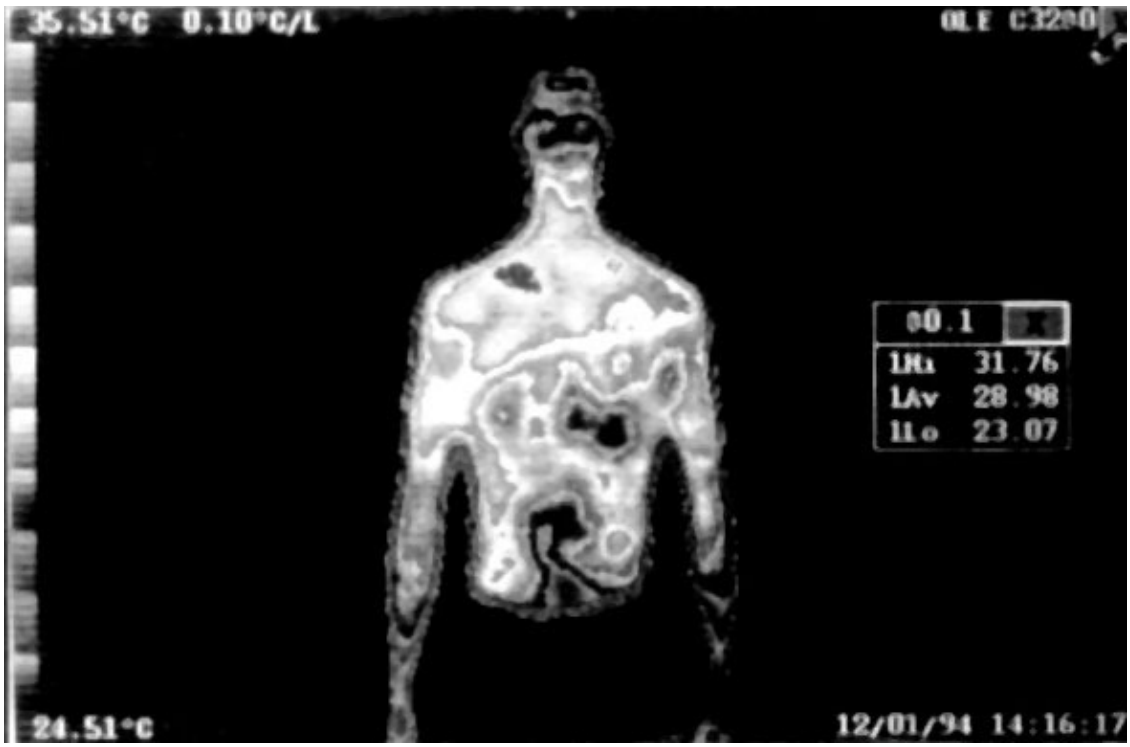


Figure 3
Neutral/PBS trial: Thermal image immediately after removal of protective barrier suit



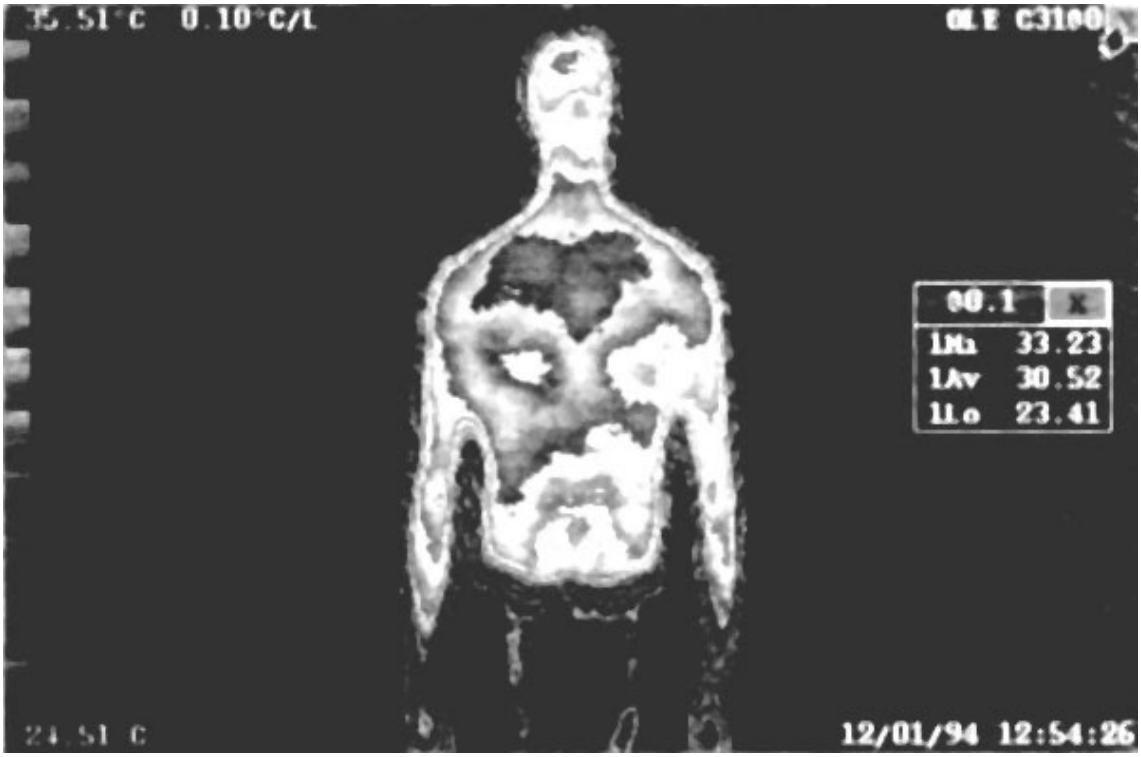
The decrease in skin temperature during recovery after the removal of the PBS (-0.57 °C) is most evident in the Neutral/PBS trial. This immediate drop in skin temperature can be clearly visualized in thermograms provided (see Figure 3 Pre & Figure 4 Post).

Discussion

Working in warm-hot, humid conditions challenges the body‘s mechanisms for maintaining thermoregulatory control. This regulatory control may be impaired when one wears a protective barrier suit which limits one‘s ability to dissipate heat, especially due to the reduction in evaporative cooling. In the Neutral/PBS trial, the core temperature continued to rise after the cessation of work and continued to remain elevated throughout the recovery. This increase in core temperature coincides with a dramatic decrease in mean skin temperature. A decrease in skin temperature is indicative of a reduced cutaneous blood flow (cutaneous vasoconstriction). Similar vasoactive changes were also noted for the legs and arms. In this investigation, no attempt was made to quantify the role of the chest versus arm and leg skin surface areas in the dissipation of stored heat. This decrease in blood flow at the skin reduces the removal of heat from the core to the periphery to the environment. As the muscles continue to dissipate their metabolically produced heat to the blood, the core temperature rises. Under these conditions, further recovery time is required to decrease the heat storage and core temperature. This reduction in skin temperature is not the result of enhanced evaporative cooling due to a sharp decrease in temperature and humidity as the individual came out of the PBS, as this cooling effect would have resulted in a decrease in core temperature. This decrease in skin temperature is due to the peripheral vasoconstriction that resulted from now uncovered subject being exposed to the cooler air temperature. In contrast, the Neutral/NO PBS trial resulted in a very slight increase in core temperature (0.4 °C.) suggesting that heat loss mechanisms were able to dissipate most of the heat production. The difference in these two trials (Neutral/PBS and Neutral/NO PBS) illustrates the impact of a PBS on heat loss mechanisms, heat dissipation, and heat storage.

In the warm trials, the mean skin temperature and core temperature decreased throughout the

Figure 4
Neutral/PBS trial: Thermal image after recovery



recovery period. Again, the skin temperature demonstrated a modest vasoconstriction which the subjects perceived as a cooler sensation of the skin. This reduction in cutaneous blood flow will lessen the body‘s ability to dissipate heat and may demonstrate the body‘s attempt to titrate the thermal responses to reduce large fluctuations in core temperature.

The textile development of protective barrier suits for various work applications are often directed towards the assault from the external stressor (fire, bacteria, radiation, etc.) without much consideration for the potentially fatal internal environment resulting from heat storage. To compensate for this potential problem, many work/exercise situations incorporated work/ rest ratios to allow the subjects to reduce their internal core temperature. These recommendations are made according to the known/calculated heat production related to a specific work task. Determinations of the thermal load at the work site may incorporate the use of skin temperature probes, but often ignore core temperature because of the invasive nature of the probe. These skin temperatures will provide isolated skin temperatures measure which maynot represent the mean skin surface area. Furthermore, the method of attachment of these probes to the skin surface area often influences the thermal measure. The use of thermography for deter- minations of skin surface temperatures can provide valuable information related to the blood flow response after a thermal stress. Our data suggest that the time for recovery from a thermal stress may be increased as a result of this decrease in cutaneous blood flow to the chest region. Despite the feeling of being cool, these subjects have significantly elevated core temperatures due to vaso- constriction.

Conclusions

For the test conditions described above, the following conclusions can be made.

1.Immediately post trial/recovery, the chest region thermograms were cooler than pre- trial and decreased substantially upon removing the PBS.

2.The cooler chest temperature without a reduction in core temperature during the recovery period suggests cutaneous vasoconstriction of the chest region.

3.The cooler chest temperature resulting from cutaneous vasoconstriction are not indicative of a reduced thermal stress to the body core temperature.

4.The use of skin temperature alone to determine an adequate recovery prior to resuming work will provide an inaccurate assessment ofthe thermal bad on the individual.

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Thermography: Its role in functional evaluation of mammalian testes and scrotum

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The disruption of the normal thermal patterns of the scrotum is directly related to testicular degeneration, spermiogenesis, and male infertility. In animal studies conducted in our department over the last 20 years on scrotal thermoregulation of bulls, stallions, bucks, dogs, and llamas are presented. This study manuscript also reviews some of the infrared thermography works in human subjects. The normal infrared thermographic patterns of the scrotum in all species studied thus far are characterized by having right and left symmetrical patterns and a constant decrease in thermal gradient from the base to the apex. In bulls and bucks the temperature gradients of 4°C to 6°C from the base to the apex is considered normal. Where as in human and stallions may have a gradient of 3°C to 4°C. A gradual decrease in temperature from the base to the apex was demonstrated by concentric bands representing a gradual cooling pattern, which demonstrated the function of a vascular counter current heat exchange mechanism in the testis. Normal infrared thermal gradient and patterns in dogs and llamas are unique to their own species and may vary from other animals.

Various clinical conditions and factors can cause disruption of thermoregulatory mechanisms in the scrotum and testes, thus causing depression of spermatogenesis. Lack of thermal symmetry and change in temperature gradients were seen in both acute and chronic testicular degeneration. Increase in scrotal temperature (>2-3°C), and reduction of temperature gradient from base to apex were also seen in testicular degeneration. Thermography, a non invasive technique, has been efficacious in diagnosing acute and chronic testicular, which may cause transient or permanent infertility in the male.

Key words: thermography, thermoregulation, scrotum, testis, infertility

Die Bedeutung der Thermographie zur Beurteilung der Funktion des Hodens und Skrotums von Säugetieren

Die Veränderung der normalen Temperaturverteilung am Hodensack steht im direkten Zusammenhang mit Hodendegeneration, Spermiogenese und männlicher Infertilität. In den letzten 20 Jahren wurden Untersuchungen über die Thermoregulation des Skrotums an Stieren, Hengsten, Bocken, Rüden und Lamas durchgefiihrt und deren Resultate werden berichtet. Auch einige Ergebnisse der InfrarotThermographie des menschlichen Hodens werdendargestellt. Bei allen bislang untersuchten Arten zeigt das Infrarotmuster des Hoden eine symmetrische Temperaturverteilung an beiden Seiten und eine konstante Verringerung des Temperaturgradienten von der Basis zum Scheitel hin. Bei Stieren und Böcken gilt ein Gradient von der Basis zum Apex im Ausmaß von 4 bis 6°C als normal. Hingegen beträgt dieser Temperaturabfall beim Menschen und bei Hengsten lediglich 3-4°C. Eine allmähliche Temperaturverminderung von der Basis zum Scheitel wurde durch konzentrische Temperaturzonen demonstriert, die einen auf Gegenstrom basierenden Kühlungsmechanismus des Hodens anzeigen. Das normale lnfrarotmuster bei Hunden und Lamas sind typisch für ihre Art und unterscheiden sich vom Muster anderer Tiere.

Verschiedene Erkrankungen und Umstände können zu einer Störung der Wärmeregulation des Skrotums und des Hodens führen mit der Konsequenz einer verminderten Spermiogenese. Ein Verlust der symmetrischen Temperaturverteilung und ein veränderter Temperaturgradient wurden sowohl bei akuten und chronischer Hodendegeneration beobachtet. Auch eine Erhöhung der Hodentemperatur um 2-3°C und die Verminderung des Temperaturgradienten von Basis zum Scheitel des Hodens wurde beobachtet. Die nicht invasive Methode der Thermographie kann wirksam akute und chronische Hodenveränderungen diagnostizieren, die eine vorübergehende oder dauernde männliche Unfruchtbarkeit bedingen können.

Schlüsselwörter: Thermographie, Wärmeregulation, Skrotum, Hoden, Unfruchtbarkeit
Thermology international 2002; 12: 125-130

Thermoregulation of the Testes and Scrotum

The testicular temperature must be below body temperature for normal spermatogenesis [1,2, 3,4]. The testes of most domestic mammalian species migrate out of the abdomen and are retained in the scrotum which provides the appropriate thermal environment for normal spermatogenesis. The thermoregulatory mechanism by which testicular temperature is controlled consists of a balance between the heat carried into the testes by arterial blood, the metabolic heat generated, and the heat loss by the scrotum [5,6,7,8]. The major components of scrotal thermoregulation include heat loss regulation by the tunica dartos and the cremaster muscle, sweat glands in the skin, and vascular counter current heat exchange.

The scrotum contains temperature receptors that initiate a series of reflexes influencing local and general thermoregulation in. response to changing skin temperature [9,10,11]. The local regulation consists of blood flow changes in the scrotal skin. The scrotal skin contains arteriovenous anastomoses that allow maximum blood flow to the skin and blood flow to the scrotum doubles when the difference between scrotal and body temperature decreases by 50%. Thus increased skin temperature results in almost doubled blood flow to the scrotum and causes vasodilation to enhance heat loss [12,13,14,15]. In ruminants and many other animal species sweat glands are present in the scrotal skin and under sympathetic enervation increased sweating occurs in response to higher environmental temperature. Thermally induced scrotal sweating can be abolished by perineal nerve block or sympathetic denervation. Scrotal sweating begins in calves and rams when skin temperature reaches 35 to 36 C. The degree of scrotal extension or relaxation is dependent upon the skin temperature and is independent of ambient air temperature between 9 and 47°C [15]. On the other hand, general body temperature regulation can be influenced by scrotal skin temperature similar to that in other skin region. Thermal polypnea was induced in rams by increasing scrotal temperature above a threshold value of 35 to 360C [12]. When scrotal temperature exceeds 38 °C marked respiratory efforts result in decreased body temperature, as much as 2 degree C in one hour. The marked increase in respiratory frequencies of more than 200 breaths/ min was observed by scrotal heating, whereas heating equivalent areas of flank skin only caused a small increase in respiratory rate. The response from the scrotum can be abolished by local anesthesia. Similarly, cooling of the scrotum caused shivering when core body temperature was normal. This relationship between scrotal temperature and respiratory rate and core body temperature does not occur in the bull. Perhaps this phenomenon can be explained by the fact that the scrotum represents a greater percentage of surface area in rams than in bulls.

The scrotum varies in appearance and size; cold causes it to become contracted and wrinkled, whereas heat causes it to relax. Receptors in the scrotum initiate the impulses via sensory afferent fibers in the superficial perineal nerves, and lumbar sympathetic outflow provides tonic control to the tunica dartos muscles. The degree of extension of the scrotum depends on the scrotal skin temperature in rams; when skin temperature exceeds 35°C, the tunica dartos muscles are fully relaxed [14,16]. The cremaster muscle inserts on the tunica vaginalis parietals. The cremaster, although incapable of sustained contraction observed in the tunica dartos muscles, retracts testes in response to extremely cold environments [17]. When the cremaster muscle contracts the spermatic cord veins are compressed thereby reducing blood flow away from the testes. On the other hand relaxation of the cremaster muscle lowers the testes and the blood flow in the testicular veins increases, thus allowing warm blood to leave the testicle with resultant lowering of testicular temperature [5,6,7,17].

The testicular artery has a convoluted structure with arterial coils enmeshed in the pampiniform plexus of the testicular veins providing a counter current mechanism by which arterial blood entering the testes is cooled by the venous blood leaving the testes. In the ram the temperature of the blood in the testicular artery decreases 4° C in its course from the external inguinal ring to the surface of the testes. To function effectively the mammalian testes must be maintained at a lower temperature than the body temperature [1,6,7]. The testicular artery is elongated and becomes extremely convoluted between the inguinal canal and the testis. Within the pampiniform plexus the spermatic vein closely surrounds the artery and in many places only the vessel walls separate arterial and venous blood. The temperature of testicular

venous blood is always similar to the temperature beneath the scrotal skin and regulates the temperature of arterial blood entering the testis. Pre-cooling of arterial blood by countercurrent heat exchange between the testicular artery and veins in the pampiniform plexus and spermatic cord ensures the deep testicular temperature is uniform and quickly follows any variation in scrotal temperature. In the bull, the long course of the testicular artery around the testis surface in the tunica vasculosa may allow for further heat loss [8]. This countercurrent heat exchange is not autoregulatory and is strictly dependent upon the magnitude of the temperature gradient between the body and the scrotum. This mechanism becomes inoperable if testicular heat is not lost through the scrotum thereby causing venous blood to be inadequately cooled.

Thermographic evaluation of the testes and scrotum

Infrared thermography, a non invasive, non contact imaging technique is used to determine normal and abnormal thermal patterns in bulls, stallions, bucks, and other animal species. The normal thermogram of the scrotum in all species studied is characterized by a right and left symmetrical pattern with a constant decrease in the thermal gradient from the base to the apex [1,2,3].

In bulls, stallions, and bucks the temperature gradient of 4°C and 6° C from the base to the apex is considered normal (Figure 1,2,5). A gradual decrease in temperature from the base to the apex is demonstrated by concentric bands representing a gradual cooling pattern consistent with the function of a vascular coun-

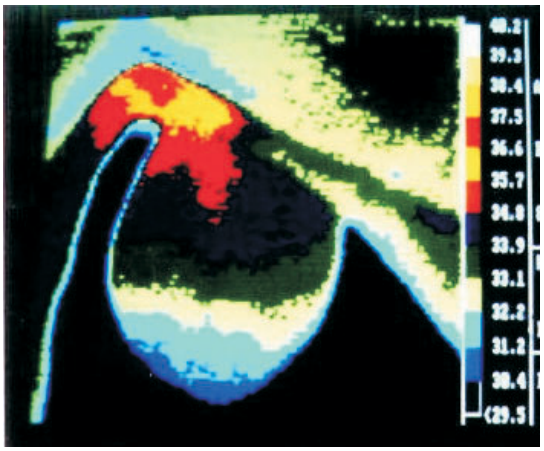

Figure 1
Normal Bull

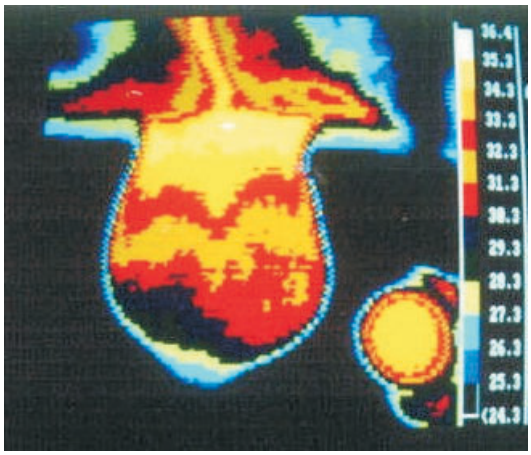

Figure 2
Normal Goat

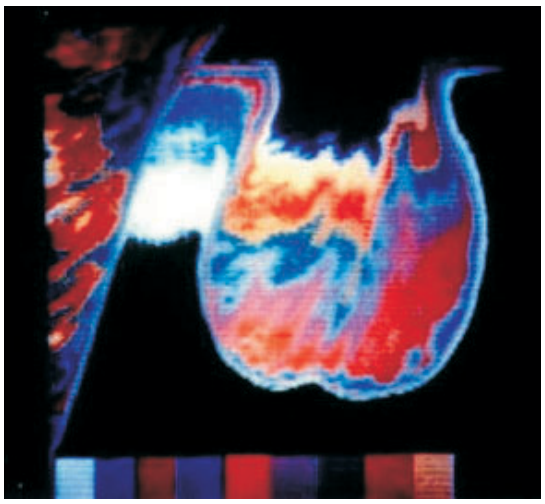

Figure 3
Acute Degeneration

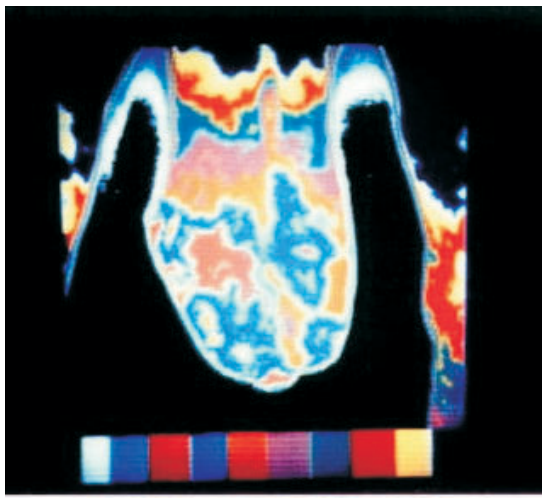

Figure 4
Chronic Degeneration

ter-current heat exchange mechanism in the testis. Normal infrared thermal gradients and patterns in dogs (Figure 7,8) and llamas (Figure 6) are unique to their own species and may vary from other animals [1,2,3].

In the bull in the normal temperature form the base to the apex (34.9 ± 0.6°C to 30.11 ±0.91°C) is significantly different ($P<0.05$). Lack of thermal symmetry and change in temperature gradients were seen in both acute (Fig 3) and chronic (Fig 4) testicular degenerations. Lack of thermal symmetry was seen in bulls with unilateral lesions [1,2]. Inflammation of one testicle increased ipsilateral scrotal infrared emission temperature 2.5 °C to 3°C above that in the contralateral side. If both testes were inflamed and hyperaemic, there was an overall increase in scrotal temperature of at least 3°C, and a reduction in temperature gradient of 2 to 3°C from the base to the apex of the scrotum. Area temperatures in bulls with chronic testicular degeneration with fibrosis were reduced.

Inflammatory processes of the bull testicle are a common clinical entity. It is difficult to predict the long-term effects of conservative (non surgical) treatment of orchitis, periorchitis, and epididymitis. When one testicle is inflamed, the heat thus generated affects the contralateral testicle. Reversibility of degenerative change depends upon the severity and duration of the insult [18,19]. Abnormal morphologic changes of spermatozoa can appear as early as 2 days after the onset of acute inflammation. The magnitude of the added insult of surgery in the past has been unknown [19]. Thus, we performed unilateral orchiectomies in bulls with satisfactory semen quality to study the effect of the procedure on quality of semen form the

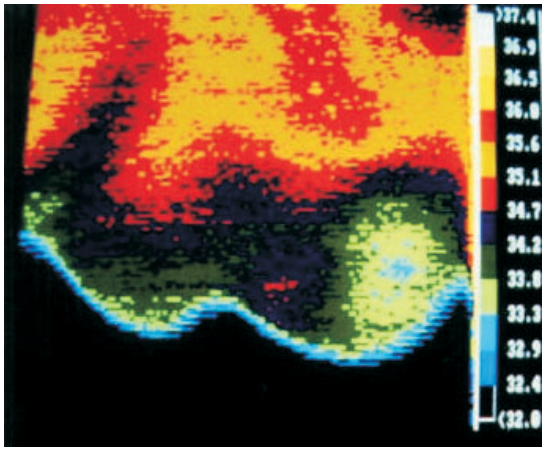

Figure 5
Normal Stallion

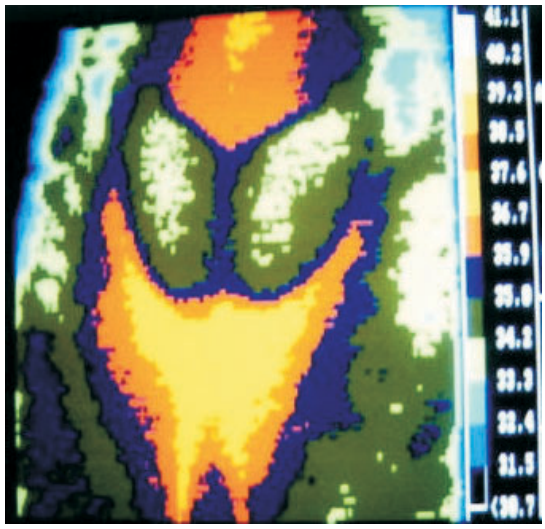

Figure 6
Normal Llama

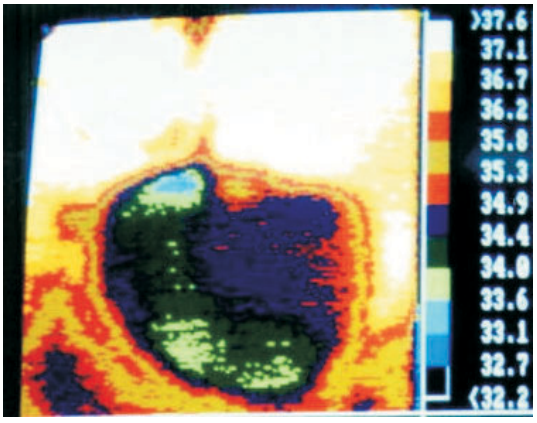

Figure 7
Normal Dog

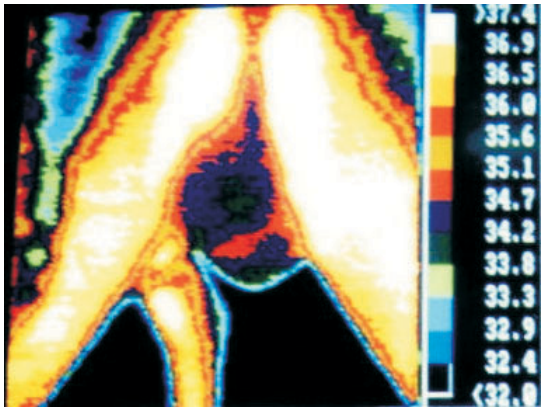

Figure 8
Normal Dog

contralateral testicle. Semen was collected by electroejaculation before surgery and on alternate days for 2 weeks, then once weekly for 8 weeks. Each sampling day, progressive motility and morphologic features of spermatozoa were determined, and scrotal thermograms were taken. The percentage of normal spermatozoa decreased significantly ($P<0.05$) only on postoperative day 6. Progressive motility scores vaned but at the end of the study there was no significant difference from preoperative values. Scrotal thermography revealed inflammation in the contralateral side of the scrotum, beginning 3 days after surgery, but the thermograms were normal in most bulls by 3 weeks after and all thermograms were normal by 4 weeks [19].

Conclusions

Various clinical conditions and other factors can cause disruption of thermoregulatory mechanisms in the scrotum and testes, thus causing depression of spermatogenesis. Even though the causes of infertility in several mammalian species are well defined, very little is known about the effects of the disruption of neurogenic mechanism of scrotum and testes [14]. Studies were done to determine the effects of unilateral transections of the superficial perineal and the caudal scrotal nerves on thermoregulation of the bull scrotum. Unilateral neurectomies were done so that the non-neurectomised side of the scrotum may serve as control for evaluation of thermal patterns and testicular functions. Thermographic examinations and semen evaluations were done periodically in all bulls until return of normal testicular function. Even with the unilateral neurectomised, thermal pattern disruption and abnormal increases in thermal gradient occurred on both sides of the scrotum. This increase in thermal heat due to loss of vasomotor tone resulted in testicular degeneration of greater than 3 months in all six bulls used in this study. Most bulls returned to normal testicular function in about 5 to 8 months. The present studies in bulls provide the evidence that a nerve injury associated with disruption of thermoregulation may cause testicular degeneration and infertility in mammals with scrotal testes.

In human beings, thermography of normal testes is characterized by symmetric and constant thermographic patterns, with the temperature staying between 32.5°C and 34.5°C [20,21,22, 23,24]. An ipsilateral increase in scrotal IR emission, which leads to a difference of at least 2.8°C between the abnormal and the normal testicle, is associated with intrascrotal tumor, inflammation, and varicoceles [25,26]. Thermography has also been used in man to diagnose subclinical varicoceles and lack of symmetry caused by unilateral lesions. In man, testicular thermoregulation was disrupted when the oral body temperature reached 37.7°C [27].

Testicular degeneration could be induced by disruption of the thermoregulatory mechanism, localized or systemic infection, vascular lesion, obstructive lesion in the head of the epididymis and noxious agents such as chemicals, metals, and ionizing radiation [28,29,30]. Various causes of orchitis, neoplastic diseases (i.e., interstitial cell tumors, sertoli cell tumors, seminoma), spermatoceie, spermatic granuloma, torsion of the spermatic cord, and other abnormalities will alter the unilateral or bilateral infrared emission.

While obtaining testicular thermograms, environmental effects should be minimized by taking thermograms in a room kept at a temperature cooler than that of the body and free from air drafts [31,32]. Animals should be held in these surroundings for 10 to 15 minutes before obtaining thermograms. Because various sedatives, tranquillizers, vasoactive drugs, and general anaesthesia may affect thermographic patterns and temperature gradients, animals should not be given general anaesthesia and tranquillizers before thermographic evaluation.

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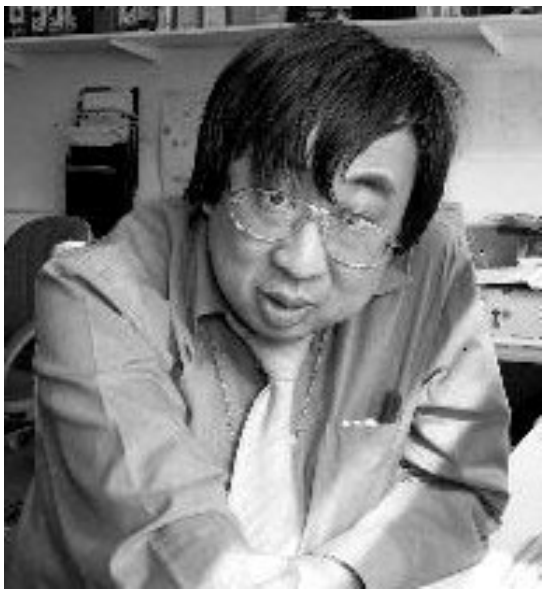
Brian Chu, 1961-2002

Graham Machin

National Physics Laboratory, Teddington, Middlesex, TW11 0LW, United Kingdom

Brian graduated from Brunel University in 1983 with a 2:1 in Physics and joined the Temperature Standards Section of NPL that year. He soon made his mark working with colleagues on the development of the world's first cryogenic radiometer, a device that went on to revolutionise the measurement of thermal and optical radiation around the world reducing measurement uncertainties by more than a factor of 10. Later he carried out research on the performance of a new type of thermocouple (the Nicrosil/Nisil, type N) work for which he and other colleagues were awarded the Honeywell Prize in 1985.

From the late 1980s Brian worked in non-contact thermometry standards and it is for this work that he will be most remembered. He undertook research into improving blackbody calibration sources, (culminating in the development of a suite of heat-pipe blackbody sources with sub 0.1 °C uncertainty) as well as research into understanding sources of uncertainty and calibration consistency. He published 11 scientific papers on many aspects of low temperature IR thermometry both in the literature and in conference proceedings. He also ran an ISO 17025 UKAS accredited calibration service for IR thermometers, thermal imagers, radiometers and blackbody sources. In addition he has helped other National Measurement Institutes like NPL set up their own calibration facilities, such as the Finnish and Malaysian laboratories. In more recent times Brian had become interested in traceability issues in medicine and was busy seeking to develop calibration sources for tympanic thermometers and thermal imagers.



Besides his technical prowess Brian demonstrated considerable flair with computing and wrote many of the programs currently in use in the Temperature Standards Group at NPL.

I personally had known Brian for 17 years and we worked closely together on many developments in Radiation Thermometry. I always admired his depth of technical knowledge, his commitment to his work as well as his tenacity and his sense of humour.

Brian had served in the Temperature Community for 19 years and will be sorely missed by colleagues both at NPL and from around the globe.

News in Thermology

5th International Congress of Thermology

The authorities of the City of Vienna have expressed their appreciation for the successful organisation of the 5th International Congress of Thermology, held last April in Vienna. The organising committee received a diploma and was informed that conferences bring both scientific merit and high commercial impact on the city. According to figures published by the Vienna Convention Bureau, 1,403 conferences, corporate meetings and incentives were hosted by the Austrian capital in 2001 (-1%), generating 682,000 overnight stays (± 0%). Conference tourism accounted for an unchanged 8.9% of Vienna's overall bednights.

Vienna hosted 265 international conferences in 2001, representing a year-on-year rise of 26%. Such events are not only a vital image factor for Vienna, but also the form of tourism generating the highest revenues. In 2001, turnover per delegate and night averaged EUR 352. The most frequent specialist fields were business and politics (25%), followed by human medicine (23%) and scientific disciplines (15%). The economic impact of Vienna's domestic and international conferences in 2001 was analysed in a study performed at the Vienna University of Economics and Business Administration. The total tax revenues generated by this sector were as much as 46.3 million Euro.

3rd Asian-Pacific Federation of Thermology Meeting

Professor Ye Chul Lee, M.D., Ph.D.President of APFT (Asian Pacific Federation of Thermology), and KAMT (=Korean Academy of Medical Thermography) has sent an invitation to participate in the 3rd Asian Pacific Congress of Thermology to be held from September 6 to 7, 2002 in Seoul, Korea. He mentioned , that since the last second APFT meeting at Nagaoka in Japan three years ago, great advances have been made in thermographic techniques, equipments and the applications of thermography to the detection and evaluation of various physiological processes in different diseases.

Prof. Yong-Eun CHO, M.D., Ph.D, is the Secretary General, of the 3rd Congress of APFT. The conference topics will include clinical application of thermology such as CNS& neuromusclo-skeletal diseases, pain medicine, Oriental Medicine, autonomic nervous system disorders, orthopaedics, peripheral vascular diseases, rheumatic diseases, oncology, sports medicine, dermatology and veterinary medicine. Physiology and temperature regulation, infrared technology and standardization of thermal imaging are other important themes. The conference language is English. Experts in the field of thermology from Europe, USA, and Japan have already confirmed their participation. The final programme will appear on the website of the Korean Academy of Thermology at http://www.kamt.org in due course.

The weather in early September in Seoul is warm at daytime (average 23°C,73°F) and cool in the evening (10°, 50°F).

Venue: Seoul Olympic Parktel 88-8,
Bangyeedong, Songpagu, Seoul 138-749,
KoreaPhone : +(82-2) 421-2111
Fax: +(82-2) 410-2101
E-mail : parktel@sosfo.or.kr
http://www.parktel.co.kr/eng.htm

Hotel Reservation: The meeting will be held at the Olympic Parktel in Seoul. A block of rooms has been preserved for registrants and their guests at this hotel. Conference room rates are 115,500Won for single/double occupancy. Room rate is specially reduced for this meeting. The above rate includes 10% service charge and 10% VAT, but it does not include breakfast. In order to receive the special conference room rate, registrants must be specify that they are with the Asian-Pacific Federation of Thermology Meeting when making reserva-

tions. Hotel reservations can be made by phone, fax, e-mail or home page of Olympic Parktel. Please note that after 1, August 2002, reservations will be taken on a space available basis only.

QIRT 2002 in Dubrovnik

The 6th International conference on quantitative infrared thermography will be held in Dubrovnik, Croatia, in September 24-27, 2002 The QIRT Congresses are traditional meetings of experts in the field technical and industrial applications of infra red thermal imaging. However, biomedical applications are regularly presented and discussed at QIRT Meetings. This years´QIRT provisional programme scheduled 47 oral presentations and 27 posters, of which 7 papers and 5 posters are dedicated to biomedical application of quantitative thermal imaging. The authors of these contributions originate from 5 European countries.

Call For Abstracts - American Academy of Thermology Annual Meeting

Date: **NOVEMBER 7th to 10th, 2002**

Location

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Individuals directly to call the hotel at 407-313-3600. Guarantee first night room deposit with a major credit card. To get reasonable room rates, please reserve your room as soon as possible directly with Marriott Residence Inn, Sea World International Center.

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Deadline for the abstract October 1, 2002.

All abstracts are to be sent by e-mail to Dr. Hooshmand and Dr. Goldberg.

Guidelines for abstract:

1.Microsoft Word or Windows 98.

2.Title, authors, and text 200 words. All accepted abstracts will be published in Thermology international.

3.All abstract submissions should accompany registration fees.

Registration fee: can be paid by check, money order or credit card.

Checks made payable to
American Academy of Thermology.

Mail the checks to Dr. Hooshmand along with the Registration Form.

American Academy of Thermology Member M.D., D.O., PH.D - $250.-

Technologist Member - $l50.-

Student and Resident and Fellows - $100.-

International Members and Attendees - $100.-

Non-members add additional $25.00 for registration (students & fellows excluded)

All registrations after October 1st, please add $25 more to registration amount.

9th European Congress of Thermology

Prof.Dr.Anna Jung is preparing the organisation of the 9th European Congress of Thermology, which will take place in Krakow May 30 to June 1, 2003. The conference will be combined with 6th National Congress of the Polish Association of Thermology. Thermal physiology, thermoregulation, clinical applications of thermal imaging, advances in thermal imaging technology, thermal image processing and telemedicine are the main topics of this important meeting.

Deadline for abstracts is February 1st 2003, please use the form on page 134 of this issue.

Registration fees are 350 US$ before February 1st, 2003 or 450 US$ after February1st, 2003.

All payment has to be made to
10201156-202693-270-1,
Polskie Towarzystwo Diagnostyki Termograficznej W Medycynie.

The venue of the congress is the Cultural Institute, located in the centre of the old university city of Kraków in Zyblikiewicza 1 Str. Kraków has an International airport, with a number of direct flights from the major European cities. A regular service is provided from Warsaw International airport.

A convenient hotel, Campanile, is located 100 meters from the conference hall in Sw. Tomasza 34 Str.

Prizes are for a single room: 75 USD (breakfast included) and 85 USD (breakfast included) for a double room. Deadline for hotel reservation is April 15th 2003. Please use the form printed in this issue (page 133). Reservations can also be made by e-mail:
ajung@cskwam.mil.pl

13th THERMO in Budapest

Another regular international conference will be held in **Budapest the 18th to 20th of June, 2003**. Prof Benkö has issued the invitation to the 13th conference to experts in the field of thermology to report and discuss recent advances in temperature measurement in industry, physics, medicine and biology.

The conference is hosted by the OSSKI Center (Törley Palace, Budapest, XXII. (Budafok), Anna u. 5.) located in the vicinity of the famous Budafok wine cellars. More information about the conference place and hotel accommodation will be sent after the arrival of the Registration Form.

The photocopy-ready abstracts of six A4 format pages to be presented on the conference are to be submitted **before 15 October, 2002.** To assist the work of the Scientific Committee the authors are kindly requested to point out the aim, method and results of their work.

Notification of the acceptance will be forwarded to the authors until 28 February, 2003. The abstract of all accepted papers will be included the Proceedings to be presented to the participants at the Conference.

For any further information please contact the following address:

Dr.Imre BENKÕ,
Budapest University of Technology and Economics (BME), Department of Energy (DoE),
H-1111 Budapest, Mûegyetem rkp. 7. D.208.,
Hungary.

Office phone: +361-463-2183.

DoE Phone/fax: +361-463-3273 or -310-0999.

BME Fax: +361-463-1110

E-mail: benko@eta.enrg.bme.hu

Veranstaltungen (MEETINGS)

September 6-7, 2002

3rd Asian-Pacific Congress of Thermology in Seoul, Korea

Venue: Olympic Parktel, Seoul,

*Information:*Yong-Eun CHO, M.D., Ph.D.
Secretary General,
The 3rd Congress of APFT
Tel : (82-2) 3497-3393, FAX: (82-2) 3461-9229
E-mail: ydnscho@yumc.yonsei.ac.kr

September 24-27, 2002

6th International Conference on Quantitative Infrared Thermography, QIRT´2002, in Dubrovnik, Croatia

Organized by: University of Zagreb (Croatia), Faculty of Mechanical Engineering and Naval Architecture

Venue: Collegium Ragusinum
C. Carica 4, Dubrovnik

*Information:*http://www.fsb.ht/Qirt2002

Conference secretary: QIRT´2002

Igor Sindov, Faculty of Mechanical Engineering and Naval Architecture

I-Lucica 5, 10000 Zagreb,Croatia

Phone: +385 1 616 8174 Fax: +385 1 616 5940

September 28-29, 2002

5thConference of the Polish Society of Thermology

Venue: "HYRNY" Guest-House
Zakopane, Pilsudskiego Str. 20

Information: Prof Dr.Anna Jung
Paediatric and Nephrology Clinic
Central Clinical Hospital MMU,
Szaserow Str 128, 00-909 Warsaw, Poland

Phone +48 22 681 7236/Fax +48 22 681 6763
email: ajung@cskwam.mil.pl

November 7-10, 2002

Annual Meeting of the American Academy of Thermology in Orlando

Venue:
Marriott Residence Inn.

Seaworld International Center,

11000 Westwood Boulevard,

Orlando, Florida 32821.

Phone: 407-313-3600, Fax; 407-313-3611.
www.residenceinnseaworld.com

Information:

Dr.Hooshmand at hoosh@prodigy.net

or Dr.Goldberg at JerryGol@aol.com

December 4-8, 2002

EMBEC´02 , 2nd European Medical & Biological Engineering Conference in Vienna, Austria

Venue: Vienna International Congress Centre

Special Session:
Developments in Infrared Thermal Imaging, organised by
Prof Dr.EFJ Ring & Prof.DDr K.Ammer

Information about EMBEC:
Prof. Dr. Helmut Hutten

Institute for Biomedical Engineering
University of Technology
A-8010 Graz (Austria), Inffeldgasse 18
tel: ++43-316-873-7390 fax: ++43-316-46 53 48
email: hutten@ibmt.tu-graz.ac.at

Information about the infrared session:

Prof Dr Francis Ring, email:efring@glam.ac.uk
Prof DDr Kurt Ammer.
Email:kammer1950@aol.com

May 30th – June 1st, 2003

9th European Congress of Medical Thermology in Krakow, Poland

Venue: Cultural Institute, Kraków, Zyblikiewicza 1 Str

Abstract deadline: February 1st 2003

Registration fee

Before February 1st, 2003 350 USD
After February 1st, 2003 450 USD

The payment has to be made to

10201156-202693-270-1

POLSKIE TOWARZYSTWO DIAGNOSTYKI TERMOGRAFICZNEJ W MEDYCYNIE

Hotel accommodation

A convenient hotel, Campanile, is located 100 meters from the conference hall in Sw. Tomasza 34 Str.

Single room: 75 USD (breakfast included)
Double room: 85 USD (breakfast included)

Deadline for hotel reservation April 15th2003
Reservation e-mail: ajung@cskwam.mil.pl

June 18.-20, 2003

13th International Conference on Thermal Engineering and Thermogrammetry (THERMO)

in the OSSKI Center (Törley Palace).
Budapest, XXII. (Budafok), Anna u. 5.

Chairman: Prof.Dr. I. Benkõ, BME, DoE, Hungary (EAT, HST, President of TE & TGM)

Secretary: I. Kovacsics, Msc.

EGI-Contracting/Engineering Co. Ltd., Budapest, Hungary (HST, TE & TGM)

Information: Dr.Imre BENKÕ
Budapest University of Technology and Economics (BME),
Department of Energy (DoE),
H-1111 Budapest, Mûegyetem rkp. 7. D.208., Hungary.

Office phone: +361-463-2183.
DoE Phone/fax: +361-463-3273 or -310-0999.
BME Fax: +361-463-1110

E-mail: benko@eta.enrg.bme.hu

REGISTRATION FORM ANNUAL MEETING OF THE AMERICAN ACADEMY OF THERMOLOGY 2002

NAME _____

SOCIAL SECURITY #_____

DEGREE_____SPECIALITY _____

OCCUPATION _____

HOSPITAL OR ORGANIZATION_____

HOME OR BUSInESS ADDRESS (INCLUDING DEPARTMENT)_____

_____

CITY_____STATE _____ZIP_____

DAY TELEPHONE (_____)_____FAX#(_____)_____

E-MAIL ADDRESS_____

PAYMENT SHOULD BE ENCLOSED WITH THIS FORM.

RECEPTION 11-8-02 $15.00 $ _____

REGISTRATION FEE $ _____

$_____ TOTAL

Credit Card #: _____ Expiration date: _____

Signature _____

METHOD OF PAYMENT:

Send money order, personal check or credit card # with the Registration Form.

MC / VISA / AMERICAN EXPRESS / OTHERS

9th European Congress of Medical Thermology

6thNational Congress of the Polish Association of Thermology

Kraków / Poland – May 30th – June 1st, 2003

REGISTRATION AND HOTEL ACCOMMODATION FORM

PARTICIPANT

First Name .....

Family Name .....

Address .....

ZIP Code .....

City .....

Country .....

Phone .....

Fax ..... E – mail .....

REGISTRATION FEE

Paid until	February1st, 2003	350 USD
Paid after	February1st, 2003	450 USD

HOTEL ACCOMODATION

I would like to make the following reservation in Campanile Hotel:

.....	Single room	75 USD
.....	Double room	85 USD

Deadline for hotel reservation – April 15th, 2003

Date of arrival ..... Date of departure ..... no. .....of nights

PAYMENT

The payment has been made to: 10201156-202693-270-1

POLSKIE TOWARZYSTWO DIAGNOSTYKI

TERMOGRAFICZNEJ W MEDYCYNIE

Signature ..... Date.....

Return to:
Organising Comittee:
Pediatric and Nephrology Clinic MSM
Szaserów Str 128 00 909 Warsaw 60, POLAND
Fax (48 – 22) 6816763 E – mail ajung@cskwam.mil.pl

9th European Congress of Medical Thermology

6thNational Congress of the Polish Association of Thermology

Kraków / Poland – May 30th – June 1st, 2003

Last Name.....First Name..... Title .....

Institution .....
Street .....

ZIP Code .....City.....Country .....

Phone..... Fax .....E – mail.....

Title

Autors

Abstract

Return this form not later than February 1st, 2003 to: Prof. Anna Jung
Pediatric and Nephrology Clinic MSM
Szaserów Str 128 00 909 Warsaw 60, POLAND
Fax (48 – 22) 6816763 E – mail ajung@cskwam.mil.pl