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Literatur

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Inhaltsverzeichnis (CONTENTS)

Editorial

Originalien (ORIGINAL ARTICLES)

Berichte (REPORTS)

News in Thermology

Mitteilung der ÖGT

Veranstaltungen (MEETINGS)

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Thermal imaging and stroke prevention

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Blood flow measurements of facial areas based on thermal images, recently attracted the interest of the scientific community, when Pavlidis et al used thermal imaging as alternative techniques to polygraph testing to detect deceptive subjects with high accuracy (1, 2).

In this isssue of "*Thermology international*" B.Gratt et al. revive thermal imaging as diagnostic test for carotid occlusion (3). This application of infrared thermography was described at first in 1964 (4), was used and promoted by centres in Europe (5), Japan (6) and US (7) and became finally part of the technical guidelines of the American Academy of Thermology (8). With advanced developments of Doppler sonography and magnetic resonance imaging, thermal imaging became almost forgotten as a test for carotid occlusion during the nineties.

Gratt et al report promising results of a pilot study, which found a diagnostic accuracy of 95%. However, it is necessary to ask the question - is thermal imaging really a cost effective breakthrough as a screening method for stroke prevention ? There are more limitations for this application than the authors mention in their paper. Most are related to epidemiologic features and the usual methods of staging occlusive carotid disease.

A clear relationship does exist between the degree of stenosis and the risk of stroke. Several studies have proved that only patients with 60-70% occlusion will benefit from surgical treatment (9). Unfortunately the prevalence of high- grade stenosis (i.e. 60 or more percent occlusion) is not known. Occlusion <50% has a prevalence in elderly subjects of 5-7% in females and 6-11% in males (10). Screening for high-grade carotid occlusion may only be cost effective if the possibility of finding carotid stenosis is at least 20%. Screening should therefore be restricted to patients with either occlusive peripheral artery disease <grade 1 and / or coronary heart disease (10).

Another important requirement for the use of thermal imaging as screening test of carotid occlusion is to establish if there is any relationship between temperature readings and the grade of stenosis. There is already a lack of standard for defining the grading of stenosis. Staging occlusive carotid disease can vary from one imaging technique to the next (11), and adding thermal imaging to this procedure will increase the confusion. Infrared thermography cannot image the stenosis of the carotid artery itself and must conclude from temperature changes related to the blood flow in the artery. Several conditions such as all changes in circulation, local changes of temperature regulation and skin lesions of any kind will effect this non-direct assessment of the carotid blood flow.

Standardising the technique of thermal imaging of the face is both difficult yet very important. Room temperature should be kept within a narrow range, because slight changes of the room temperature may result in big changes of the surface temperature of the face (12). The cooling rate varies a great deal between different regions of the face, although the mean temperature difference from one half of the face to the contra-lateral side does not exceed 0.2 °C. A standard position of the head during image capture and standard positioning of regions of interest are other important points required to increase the reliability of facial temperature measurements. Regions of interest should always be related to anatomical landmarks of the face to achieve reproducible

measurements even when there are individual anatomical variations.

Finally, a well-planned study in patients with different grades of carotid stenosis is needed, to establish a clear relationship between temperature readings and grade of occlusion. If such a relationship exists, thermal imaging might in fact become an important screening tool in the prevention of stroke.

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A Pilot Study of Facial Infrared Thermal Imaging Used as a Screening Test for Detecting Elderly Individuals at Risk for Stroke

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Summary

Background: Stroke is a major cause of death. Finding a noninvasive, inexpensive method for the identification of asymptomatic stroke-prone individuals would be a significant advance in public health.

Methods: Twenty subjects underwent facial telethermography with single-blind temperature analysis. Ten patients had carotid occlusive disease confirmed by Doppler ultra-sound; while 10 gender and age matched individuals were used as controls.

Results: Facial infrared thermal imaging demonstrated an overall correct classification rate of 95%, with a sensitivity = 100% (true-positive rate, 10/10) and a specificity = 90% (true-negative rate, 9/10).

Conclusions: Infrared thermal imaging has promise as a noninvasive, painless, inexpensive screening test of asymptomatic elderly adults at risk for stroke. However, clinical studies are needed before infrared thermal imaging may be accepted for routine clinical applications. *Thermol international 2002; 12: 7-15*

Keywords: Infrared thermal imaging, stroke, diagnostic test, carotid occlusal disease, pilot study.

Eine Pilotstudie zum Einsatz der Gesichtsthermographie als Screening Test bei älteren Personen mit einem Risiko einen Schlaganfall zu erleiden

Hintergrund: Der Schlaganfall ist ein bedeutende Todesursache. Das Auffinden einer kostensparenden, komplikationslosen Methode, um für einen Schlaganfall anfällige Personen zu identifizieren, würde einen bedeutenden Fortschritt für das öffentliche Gesundheitswesen darstellen.

Methode: Bei 20 Personen wurde mit einer Infrarot-Thermografie des Gesichts untersucht . 10 Personen hatten eine mit der Ultraschall-Doppler-Untersuchung nachgewiesene Karotisverengung, und weitere 10 Personen entsprechenden Alters und Geschlechts dienten als Kontrolle. Die Analyse der Thermografiebilder wurde einfach-blind durchgeführt.

Ergebnis: Die Gesichts-Thermografie zeigte eine korrekte Klassifikationsrate von insgesamt 95%, die Sensitivität betrug 100%; und die Spezifität 90%.

Schlussfolgerung: Infrarot-Thermografie bietet sich als nicht invasive, schmerzlose und kostsparende Methode an, um symptomfreie, ältere Leute, die vom Schlaganfall gefährdet sind, zu untersuchen. Weitere Studien sind aber notwendig, bevor die Infrarot-Thermografie als eine Routinemethode akzeptiert werden kann.
Schlüsselwörter: Infrarot-Thermographie, Schlaganfall, diagnostischer Test, Carotisverschluss, Pilotstudie.

Introduction

Stroke, also called cerebrovascular accident, or CVA, is the third leading cause of death in the United States. Approximately 500,000 senior citizens are victims of stroke each year. Currently in the United States, there are more than 3,000,000 disabled stroke survivors all requi-

ring long-term care with costs amounting to more than 18 billion dollars per year (1, 2). Finding methods of decreasing stroke mortality and morbidity is therefore of enormous health and economic importance. The identification of asymptomatic stroke-prone individuals by a quick, noninvasive, painless, inexpensive test would be a great advance in health care.

Atherosclerosis producing plaques, along the intima of blood vessels, is the most common cause of stroke. These plaques may fracture-off forming emboli, which can travel within vascular blood channels, lodging in the brain, obstructing the brain's blood supply, resulting in a cerebral vascular accident. The most common origin for emboli is in the region where the common carotid artery bifurcates into the internal carotid artery and the external carotid artery (3, 4).

For many years, contrast arteriography has been the accepted imaging technique or "gold standard" for detecting atherosclerosis. However, contrast arteriography is an expensive procedure associated with patient morbidity and on occasion patient mortality. Therefore, contrast arteriography is prohibited as a screening test. Currently, the method of choice for diagnosing atherosclerosis associated with impaired blood flow is Doppler ultrasound imaging, performed in a vascular imaging laboratory, under guidance of a vascular surgeon or an ultrasound radiologist. This technique is an improvement over contrast arteriography, as it has not demonstrated patient morbidity or mortality (5-7). Because of costs, Doppler ultra-sound is prohibited as a mass-screening test for asymptomatic patients.

It has been documented that intraluminal carotid plaques, which restrict and reduce blood flow, result in decreased facial skin temperature (3-15). Facial skin temperature is quick and simple to measure, as it can be accomplished by an assortment of devices (3, 4, 16). Infrared thermal imaging techniques have been demonstrated to detect a reduction of 30% or more of blood flow within the carotid arteries (7).

It is the intent of this pilot study to assess the potential of a pre-selected temperature zone, found on the face above the eyes, for the objective detection of blood flow reducing carotid plaques; assessing thermography's potential as a public health mass-screening device for detecting elderly individuals at risk for stroke.

Patients and Methods

Study population

Under IRB authorization, 20 subjects, all over 64 years of age, volunteered to participate in this pilot study. A health history was obtained, limited to information of previous stroke, diabetes, hypertension, and/or heart attack, as well as information about smoking and the use of alcohol, to check that both study groups were similar (Table 1).

Table 1.

Demographics of targeted study populations consisting of ten "normal" control subjects and ten "abnormal" subjects.

	"Normal"	"Abnormal"
Age in years, mean (s.d.)	71.6 (4.2)	73.1 (3.3)
Male: Female	7 : 3	7 : 3
Smokers	50 %	60%
Heavy drinkers	30%	40%
Previous heart attack	40%	60%
Hypertension	50%	40 %
Diabetes	40%	40%

Mean (s.d.) carotid obstruction as measured by Doppler ultrasound	
Most affected side	56.6% (8.4)
Least affected side	31.9% (6.2)

Targeted "abnormal" study population

Our "abnormal" population consisted of 10 subjects with proven carotid occlusal disease. Each patient had at least 50% occlusion of their carotid vessels on at least one side of the neck (termed most affected side), as measured by color flow Doppler ultra-sound imaging. All carotid artery ultra-sound examinations were performed in a vascular imaging laboratory using duplex scan with CDI (5 MHz for Doppler and 7.5 MHz for B-scan imaging; HP SONOS 1000; Hewlett Packard, CA).

Patients with prominent scarring over either eyebrow or with a history of stroke were not included in this study.

Targeted "normal" control population

Our "normal" population consisted of 10 subjects being "normal" or "negative" for calcified carotid plaques as judged from dental head and neck radiographs. These were panoramic, lateral skull, and frontal skull radiographs taken for other purposes, diagnosed by two board certified oral radiology specialists (17, 18). The control subjects did not undergo ultrasound imaging.

Subjects with prominent scarring over either eyebrow were not included in this study.

Infrared thermal imaging

Equipment

Facial infrared thermal imaging was conducted with an Agema 870 thermovision unit, including an infrared scanner, control unit, thermal image computer (TIC-8000) and Meds 1.02 software, with a color monitor (Agema Infrared Systems, Sacaucus, N.J.). Room conditions for infrared thermographic examinations included a draft- limited environment, no windows, closed doors, temperature ranging from 20 to 23°C, variable lighting, a comfortable patient chair, and a small hand-held electric fan.

Facial imaging

Facial infrared thermal imaging was performed using a rapidly obtained frontal projection (Figure 1). Before infrared imaging, each patient's face was cleared of hair, tied back using an elastic headband. The forehead was wiped with a cloth and then air dried with the use of a small hand-held electric fan. Each subject was seated for at least 15 minutes in the waiting room, allowing for facial temperature equilibration (taking 5 minutes) before thermal imaging was conducted. All thermal images were coded, and stored on a computer disk, allowing for a blinded analysis at a later date.

Thermal image analysis

Four blinded objective thermal measurements were made on each of the 20 research subjects (Figure 1).

Measurement 1: A mean temperature measurement was made of a rectangular area over the right forehead region on "normal" subjects and over the "most affected side" on "abnormal" subjects, approximately 1 cm x 2 cm, just above Measurement 2.

Measurement 2: A mean temperature measurement was made of a rectangular area over the right supra-orbital region on "normal" subjects and over the "most affected side" on "abnormal" subjects, just above the right eyebrow, a rectangle approximately 1 cm x 2 cm.

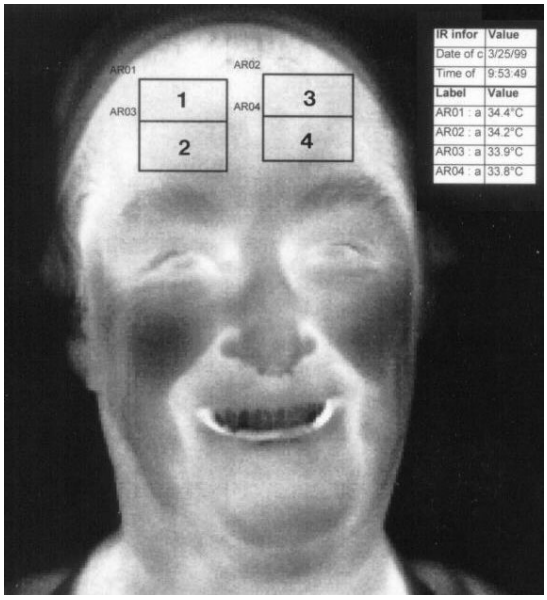

Figure 1.
Black-and-white mode facial telethermogram showing the four (4) thermal regions measured on 20 subects.

Region 1: A rectangular area on the forehead, approximately 1 cm x 2 cm, located just above Region 2.

Region 2: The supra-orbital area above the right eyebrow, a rectangular area approximately 1 cm x 2cm.

Region 3: A rectangular area on the forehead located just above Region 4, approximately 1 cm x 2 cm.

Region 4: The supra-orbital area, located above the left eyebrow, a rectangular area approximately 1 cm x 2 cm.

Similar measurements were made on the left side of each subject's face, indicated as Measurement 3 and Measurement 4. While a similar method, one employing conventional ΔT measurements, had been used in a previous study of 100 normal subjects and demonstrated normal thermal symmetry in a normal adult population (16), a new method using vertical differences in temperature regions (termed small delta T, or δT) was employed.

Data analysis.

From the 4 temperature measurements, on each of our 20 subjects, δT calculations were obtained by subtracting Measurement 2 from Measurement 1; and by subtracting Measurement 4 from Measurement 3 (Figure 1). The t-test for paired differences was used for statistical evaluation. In addition, in a blinded analysis, if at least one side of a subject's face (either the right, left, or both sides) had a δT value *below* -0.4 °C then the patient was classified as "abnormal." Likewise, if both sides of a subject's face had a δT value *equal to or above* -0.2°C, then the patient was classified as "normal". If measured δT values were found to be at -0.3 °C the subject was to be classified as "equivocal" for the presence of carotid plaques (15, 16). Finally, we conducted a correct classification analysis (sensitivity and specificity) on the 10 patients from the "abnormal " group and the 10 subjects from the "normal" control group.

Results

The results of our blinded objective temperature analysis are shown in Table 2. In our "normal" control population the mean temperature for the right supra-orbital region was 33.9 °C, as was the mean temperature over the right forehead region. The mean temperature for the left side was 33.8 °C over the supra-orbital region and 33.7 °C over the forehead region. The mean δT values for the control "normal" groups were close to 0.0 °C on the right sides of the face. There were no statistically significance differences between sides of the control group.

On the other hand, in our "abnormal" population the mean temperature for the most affected side of the face was 33.4 °C over the supra-orbital region and 34.0 °C over the forehead region. The mean temperature for the remaining side of the face was 33.6 °C over the supra-orbital region and 33.8 °C over the forehead region. The mean δT values for the "abnormal" groups were -0.6 °C on the most affected side of the face and -0.2 °C on the remaining side of the face.

There was a highly significant difference between δT measurements on the sides of the control group (δT= 0.0 °C) and the most affected side of the abnormal group (δT= -0.6 °C) ($p < 0.000$, t-statistic =7.01).

The results of our correct classification analysis indicated an overall correct classification rate of 95%, with a sensitivity = 100% (true-positive rate, 10/10) and a specificity = 90% (true-negative rate, 9/10).

Discussion

The correct classification rate using facial infrared thermal image for detecting carotid occlusal disease was found to be diagnostically useful at an overall accuracy of 95% (19/20). The correct classification rate demonstrated a sensitivity of 100% (10/10). This high sensitivity rate can be easily explained. Blood flow can be looked upon as heat transfer through a pipe. In the human body, heat is generated mainly in the liver. As warmed blood flows to an anatomic region, the region becomes relatively "warm". If the flow of warmed blood to an area decreases, then the region becomes relatively "cold". If a calcified or non-calcified plaque is present within the lumen of an artery it may obstruct or restrict blood flow and the resultant heat flow to a region. The region will then become relatively "cold". As seen in this study, skin temperature cooling around the orbit, is a function of restricted carotid arterial blood flow.

While the sensitivity of our screening test was 100%, the specificity was found to be 90% (9/10). While 90% is still a clinically acceptable detection rate, it is not unblemished. Under normal conditions, arterial blood flow is controlled locally by the autonomic nervous system and centrally by the hypothalmus. Even when all of the arteries in a region, on both sides of the body, are clear or unobstructed, the *absolute* skin temperature of a selected region will vary by: the time of day, the type and amount of food intake, exercise, ambient temperature, the use of clothing, the menstrual cycle, smoking, drugs, etc. However, the skin temperature difference between opposing sides of the body (ΔT skin temperature of oppo-

sing sides) will remain nearly zero, imaged as a symmetrical thermal pattern. The temperature difference of the skin (ΔT skin temperature) remains balanced from side-to-side, ΔT skin temperature having a normal range of 0.0 °C to ± 0.2°C (19, 20, 16). On normal subjects, this balanced temperature from side-to-side has been demonstrated on the arms, the legs, and the trunk of the body (18), and in areas of the face (16, 20).

Our 10% error rate in specificity (1/10), found in this study, may be due to our lack of an ideal "gold standard." When obtaining our targeted "abnormal" population, we used Doppler ultra-sound, an excellent "gold standard" for obtaining and defining an "abnormal" population. On the other hand, we only used conventional head and neck radiographs taken for other purposes to avoid inconvenience or unnecessary risk to our patients. We believed that this method of using different "standards" was an ethical method of dealing with control subjects. This being the case, there was a chance that we used a subject (1/10) having a non-calcified plaque as a "normal" subject.

In most arm, leg, and trunk thermography studies, investigators use ΔT as a diagnostic measure to determine abnormalities. While this works for unilateral conditions, it does not for bilateral conditions. In this study we used the

Table 2. Mean supra-orbital and forehead skin temperature measurements (in ^{o}C), same as in Table 1.

	Patient Number	Measure 1	Measure 2	Right δT		Measure 3	Measure 4	Left δT	Over-all Temp Rating
Normals	1	34.3	34.5	0.2		34.4	34.6	0.2	normal
	2	33.5	33.7	0.2		33.5	33.7	0.2	normal
	3	34.4	34.5	0.1		34.1	34.0	-0.1	normal
	4	34.2	34.6	0.4		34.3	34.5	0.2	abnormal
	5	34.4	34.2	-0.2		34.1	34.0	-0.1	normal
	6	34.5	34.5	0.0		34.3	34.4	0.1	normal
	7	34.5	34.6	0.1		34.3	34.3	0.0	normal
	8	29.9	30.1	0.2		29.9	30.0	0.1	normal
	9	34.4	34.2	-0.2		34.2	34.0	-0.2	normal
	10	34.4	34.3	-0.1		34.2	34.1	-0.1	normal
Mean		**33.89**	**33.88**	**0.07**	*	**33.73**	**33.76**	**0.03**	
s.d.		1.43	1.35	0.21		1.37	1.35	0.15	
		Most	affected	side		Least	affected	side	
Abnormals	1	34.4	33.9	-0.5		34.2	33.8	-0.4	abnormal
	2	34.4	34.0	-0.4		34.3	34.3	0.0	abnormal
	3	32.7	32.1	-0.6		32.4	32.0	-0.4	abnormal
	4	34.5	34.0	-0.5		34.3	33.9	-0.4	abnormal
	5	34.5	34.0	-0.5		34.3	34.3	0.0	abnormal
	6	32.7	32.3	-0.4		32.9	32.4	-0.5	abnormal
	7	34.4	33.9	-0.5		34.2	33.8	-0.4	abnormal
	8	34.5	34.0	-0.5		34.3	34.5	0.2	abnormal
	9	34.7	34.0	-0.7		34.3	34.6	0.3	abnormal
	10	32.7	32.2	-0.5		32.6	32.1	-0.5	abnormal
*	**10**	**33.95**	**33.44**	**-0.51**	*	**33.78**	**33.57**	**-0.21**	
		0.87	0.86	0.09		0.80	1.01	0.30	

* = Statistically significant using student t-test, t = 7.01, at p> 0.000.

difference between two regions on the same side of the face as a diagnostic measure; we call this temperature measurement "small delta T " or δT. The advantage of this measure is that it is independent of the patient having a unilateral or a bilateral carotid occlusion, in either case a significant δT will be measured indicating a patient at risk, and the patient can be referred for further follow-up. If, on the other hand, the patient is measured as being normal on both sides, they do not require follow-up to rule out carotid occlusal disease.

It should be noted that this project was only a pilot study of supra-orbital infrared thermal imaging and not a clinical trial. This pilot study was carefully designed toward arriving at a signal-to-noise-ratio for supra-orbital diagnostic infrared thermal imaging, allowing for careful experimental design in future, larger, clinical trials. Applying state-of-the-art human experimental ethics, we only conducted procedures on humans that were absolutely necessary for the conduct of this pilot study. We determined that 10 control and 10 "affected" subjects, while too small of a number for a clinical trial, was more than an adequate number of subjects for this pilot investigation. In addition, we determined that the "affected" group must undergo a Doppler ultra-sound examination, as a "gold standard" for carotid occlusal disease. On the other hand, it was thoughtfully considered and finally determined that the control group did not have to undergo this procedure (previously taken radiographs were adequate), since volunteers would have no benefit from the Doppler ultra-sound examination, and the results of the procedure would not affect the net outcome of this pilot study. If this project had been designed as a clinical trial, both groups would have been larger and both groups would have been required to complete all diagnostic tests.

Based on the results of this pilot study, we see infrared thermal image as promising for the screening of asymptomatic elderly adults at risk for stroke. In the future, computerized infrared scanners might be set-up at locations frequented by senior citizens and operated by rapidly trained technicians. If necessary, thermal images obtained *en masse* may be analyzed at a later date. We estimate as many as 8 to 12 adults could be screened per hour per infrared thermal image unit. Elderly subjects having positive thermographic findings would be referred to a physician for a clinical assessment to rule/out the presence of carotid occlusal disease. Those found to be positive on physical examination by a physician might then be referred for Doppler ultra-sound to rule/out carotid occlusal disease. Those found to have carotid occlusal disease, confirmed by clinical examination and Doppler ultra-sound, may be considered for carotid endarterectomy, a procedure with proven medical benefit (21).

Negative aspects and limitations of facial infrared thermal imaging

The negative aspects of using infrared thermal image in this study, and in general, are rather few. The infrared scanning and computing equipment is initially expensive (approximately $60,000 US), but the cost per examination would be minimal (direct costs well under $5 US, per patient).

Scarring on the forehead, just over the eyebrow, may result in a decreased blood flow to the region, and falsely appear "cold" on the telethermogram. However, in conducting over 1,200 clinical facial telethermographic examinations we have found only 5 subjects (less than 1%) with sufficient scarring on the forehead to disqualify them as candidates for the procedure. A more common problem occurs when female patients are wearing heavy make-up, cosmetics, or thick ointments on their skin, covering their forehead. These must be removed prior to conducting facial infrared thermal imaging. After gentle scrubbing and washing it is best to wait an additional 15 minutes prior to infrared imaging.

Marked facial sunburn will also disqualify a subject and must be given two weeks to heal before facial infrared thermal image. Patients with a history of migraine headache have been reported to have "vascular cold patches" on their foreheads (22, 23), which may be a problem for some subjects having the potential to produce "false" positive readings. Similarly, patients with a history of cluster headache are reported to have "hot spots" on their foreheads (24, 25). While we have not been troubled by these conditions in the conduct of over 1,200 facial thermograms, more clinical research will help define the magnitude of these conditions and the role of a medical history in the prescreening of patients.

Most future projections on the scientific development of infrared thermal imaging are very

promising, with continuing advances in high-technology electronics resulting in equipment with improved precision and increasing accuracy. Electronic thermal imaging equipment and advanced computing systems both continue to become easier to operate and less expensive. More research will help to clarify the role of thermal analysis in medicine and dentistry.

In conclusion, the results of this pilot study give promise for the diagnostic potential of infrared thermal image as a simple, non-invasive, screening test for elderly asymptomatic adults at risk for stroke. More research is required before facial infrared thermal image can be clinically applied on a daily basis for the detection of carotid occlusal disease.

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Infrared Imaging in Cardiology: Evaluating the Dual Nature of Endothelin-1

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Summary

Endothelin-1 (ET-1) is a potent vasoconstrictor peptide, which may also elicit severe ventricular arrhythmias. The aims of our study were apply infrared imaging to compare the effects of total left anterior descending coronary artery (LAD) occlusion to intracoronary (ic.) ET-1 administration and to investigate the pathomechanism of ET-1 induced arrhythmias in 3 groups of anesthetized, open-thoracic mongrel dogs. In group A (n=10) a total LAD occlusion was carried out for 30 min, followed by a 60 min reperfusion period. In groups B and C ET-1 was administered into LAD for 30 min at a rate of 30 pmol/min (n=6) and 60 pmol/min (n=8). Infrared imaging was applied to follow the epimyocardial heat emission changes. Significant epimyocardial temperature decrease indicating ischemia was observed only in group A (ΔT_{30MIN}: -1.44±0.21°C, $p<0.01$). At the end of the ET-1 infusion period coronary blood flow (CBF) was reduced significantly in groups B and C (ΔCBF_{30MIN} B: 21±2%, $p<0.05$; C: 35±2%, $p<0.05$), paralleled by a significant epimyocardial temperature decrease in group C (ΔT_{30MIN}: -0.65±0.29 °C, $p<0.05$). Two dogs died of ventricular fibrillation (VF) in the reperfusion period in group A. Ventricular premature contractions and ventricular tachycardic episodes appeared in group B, where six dogs died of VF in group C. In conclusion, although ET-1 reduced CBF significantly in groups B and C, cardiothermography did not indicate ischemic changes. ET-1 induced major ventricular arrhythmias appeared before signs of myocardial ischemia developed, though reduced coronary blood flow presumably contributed to sustaining the arrhythmias. *Thermology international 2002: 12: 14-22*

Key words: infrared imaging, endothelin-1, ventricular arrhythmias

Infrarot Thermographie in der Kardiologie: die Beurteilung der Doppelnatur von Endothelin-1

Endothelin-1 (ET-1) ist ein hochwirksames vasokonstringierendes Peptid, das auch schwere ventrikuläre Arrhythmien auslösen kann. Das Ziel unserer Studie war mittels Infrarot-Thermographie den Einfluss eines völligen Verschlusses des vorderen absteigenden Astes der linken Koronararterie (LAD) mit der intrakoronaren (ic) Infusion von ET-1 zu vergleichen un den Pathomechanismus von ET-1 ausgelösten Arrhythmien im Tierexperiment an 3 Gruppen von narkotisierten Bastardhunden mit geöffnetem Brustkorb zu untersuchen. In Gruppe A (n=10) wurde der vollständige LAD-Verschluss 30 Min. lang aufrecht erhalten, anschließend wurde 60 Min. lang reperfundiert. In den Gruppen B und C wurde ET-1 in die LAD 30 Min. in einer Konzentration of 30 pmol/min (n=6) bzw. 60 pmol/min (n=8) infundiert. Mittels Infrarot-Thermographie wurden die Veränderungen der epimyokardilen Wärmeabstrahlung registriert. Ein für eine Ischämie typischer, epimyokardiale Temperaturabfall signifikanten Ausmaßes wurde nur in Gruppe A (ΔT_{30MIN}: -1.44±0.21°C, $p<0.01$) beobachtet. Am Ende der ET-1-Infusion fand sich die Koronardurchblutung(CBF) in den Gruppen B und C (ΔCBF_{30MIN} B: 21±2%, $p<0.05$; C: 35±2%, $p<0.05$) signifikant reduziert, bei gleichzeitigen signifikantem epimyokardialen Temperaturabfall in Gruppe C (ΔT_{30MIN}: -0.65±0.29 °C, $p<0.05$). In Gruppe A verendeten 2 Tiere in der Reperfusionsphase an ventrikulären Fibrillationen (VF). Unzureichende ventrikuläre Kontraktionen und ventrikuläre Tachycardien wurden in Gruppe B beobachtet, während in Gruppe C sechs Hunde an VF verstarben. Zusammenfassend kann gesagt werden, dass die Thermographie des Herzens keine ischämischen Veränderungen entdecken konnte, obwohl ET-1 den CBF in den Gruppen B und C signifikant reduziert hatte. Deutliche ET-1 induzierte ventrikuläre Arrhythmien entstehen bevor sich Zeichen einer myokardialen Ischämie entwickeln, auch wenn die reduzierte Kotronardurchblutung mit großer Wahrscheinlichkeit am Fortbestand der Arrhythmien beteiligt ist.
Schlüsselwörter: Infrarot-Thermographie, Endothelin-1, ventrikuläre Arrhythmie

Introduction

Endothelin-1 (ET-1) is an endothelium-derived potent vasoconstrictor, consisting of 21 amino acids. ET-1 secretion is stimulated by vascular stretch, endothelial injury, sympathetic activation, impaired release of nitric oxide and hypoxia (1). Raised levels of ET-1 have been detected in myocardial infarction, chronic heart failure and cardiogenic shock. Besides being a vasoconstrictor, ET-1 may also exhibit a primary arrhythmogenic property that is not solely attributable to myocardial ischemia. Earlier studies have shown the direct arrhythmogenic effect of ET-1 on isolated cardiac tissues (2) and the development of severe ventricular tachyarrhythmias on low dose intracoronary (ic.) infusion of ET-1 (3-5). The data of these investigations suggest that the arrhythmogenic effect of ET-1 may differ from the ischemic arrhythmogenesis, however direct evidence is still lacking. Therefore, we employed infrared imaging to detect possible myocardial ischemia.

The aims of our study were (1) to compare a 30 min total left anterior descending coronary artery occlusion to two different doses of ic. administered ET-1 nfusion and to determine the hemodynamic and thermographic changes during the observation period and (2) to investigate the ischemic or direct pathomechanism of ET-1 ar- rhythmias.

Materials and Methods

General Preparation

Acute experiments were conducted on three groups of mongrel dogs (weight: 22.8±0.5 kg). The investigation conformed with the *Guide for the Care and Use of Laboratory Animals* published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996). Each animal was initially anesthetized with sodium pentobarbital (30 mg/kg iv.), and additional anesthesia was given as needed to maintain a constant level. After endotracheal intubation the dogs were ventilated with room air (Cape Ventilator CV2424, Cape Engineering Co. Ltd., U.K.). The right femoral artery was catheterized for monitoring arterial blood pressure and the left great saphenous vein was catheterized for intravenous infusion of fluids. Standard ECG leads were recorded throughout the experiment. Trans-sternal thoracotomy was performed in the 5th intercostal space and the heart was suspended in a pericardial cradle. The left anterior descending coronary artery (LAD) was isolated and an electromagnetic flow probe (Gould SP 7515) was placed around the vessel and connected to a flowmeter (Statham SP 2201) for measurement of coronary blood flow. In Group A a snare occluder was placed around the LAD under its first diagonal branch. In Groups B and C a 24G catheter was positioned in LAD to allow ic. infusion of ET-1 and saline. Continuous recordings of coronary blood flow (CBF), blood pressure (BP), heart rate (HR) and ECG were performed on a computer using Dasylab 4.0 software.

Infrared Imaging

Thermographic measurements were performed with the Detector Unit TH 1101 and the Thermo Tracer TH 1100 (NEC San-ei Instruments Ltd., Japan, NBN Elektronik, Austria). On-line visual analysis of the thermograms was followed by an off-line evaluation with a tailor-made computer program called PicWinIris (EBS Automatisierte Thermographie und Systemtechnik GmbH, Germany). Two measurement areas were fitted on the obtained thermal images: one area covered the anterior surface of the left ventricle, whereas the other (laying over an indifferent myocardial region) was taken as control. The software calculated the minimum, maximum and mean temperatures in both areas.

Experimental Protocol

After surgical instrumentation each animal underwent a 20 min equilibration period to ensure stability of the preparation, which was followed by a 20 min baseline period. At the end of the baseline period coronary blood flow, blood pressure, heart rate and ECG were recorded, two thermographic images were taken.

Group A: Effects of 30 min LAD occlusion and 60 min reperfusion were studied in 10 dogs.

Groups B and C: Effects of 30 min intracoronary ET-1 infusion were examined in 14 dogs. ET-1 was administered into LAD at 30 pmol/min (Group B, n=6) and 60 pmol/min (Group C, n=8) doses, respectively.

Thermographic images were obtained in 1 min intervals in all 3 groups throughout the experiment.

Statistical Analysis

Data are expressed as mean±SEM. Repeated measure ANOVA was used on the dependent variables, whereas one way ANOVA was applied on the independent variables. Newman-Keuls post hoc test was used when appropriate. The level of statistical significance was achieved as $p<0.05$.

Results

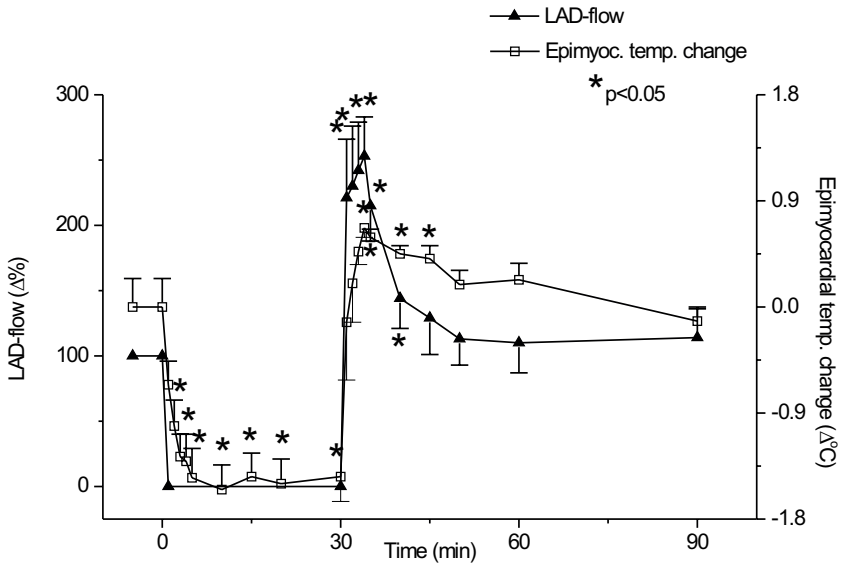
Effects of 30 min ischemia of the left anterior descending artery and 60 min reperfusion

Coronary blood flow ceased during the 30 min occlusion, a reactive hyperemic response (control vs. maximum: 34±3 vs. 85±12 ml/min, $p<0.01$) was observed on release of the occlusion. Coronary blood flow returned to normal value at 20 min of reperfusion (Fig. 1). Blood pressure and heart rate did not change significantly. In the occlusion period some ventricular premature contractions (VPC) were observed in all dogs. Upon reperfusion VPC-s were noticed in all of the ten dogs and ventricular tachycardia (VT) occurred in two instances. Two dogs died of ventricular faliure in the 6th and 8th min of the reperfusion, respectively. Significant ST-segment elevation (greater than 0.2 mV) was observed in all but one case. Epimyocardial temperature of the LAD-area decreased continuously and significantly during the ischemic period (ΔT_{30MIN}: -1.44±0.21°C, $p<0.01$). The highest epimyocardial temperature increase (ΔT_{34MIN}: +0.67±0.08°C, $p<0.01$) was noticed with the peak coronary blood flow value in the 4th min of the reperfusion (Fig. 4). A good correlation was found between the temperature increase of the LAD-area and the elevated coronary blood flow throughout the whole reperfusion period ($r=0.73$).

Effects of 30 pmol/min intracoronary ET-1 infusion

Coronary blood flow gradually decreased during the ic. ET-1 infusion (control vs. 30 min: 38±4 vs. 30±3 ml/min, $p<0.05$), but returned to 90% of the control value at 60 min after terminating the infusion (Fig. 2). Blood pressure and heart rate remained unaltered. Significant ST-segment elevation was observed in one dog only. All dogs exhibited VPC-s in increasing numbers during the ET-1 infusion (Fig. 7), two dogs had multiple VT-s. No VF was observed in the first 30 min. In the reperfusion period multiple VT-s occurred in two dogs, while one animal died of VF in the 9th min of reperfusion. In that dog coronary blood flow was virtually unaffected (control vs. 39 min: 55 vs. 53 ml/min) at the onset of ventriculat fibrillation. The tem-

Figure 1.
Coronary blood flow and epimyocardial temperature changes during 30 min LAD occlusion and 60 min reperfusion.



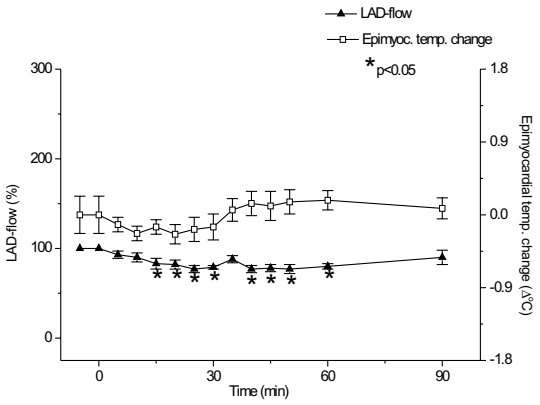


Figure 2
Coronary blood flow and epimyocardial temperature changes during the 30 pmol/min ET-1 infusion.

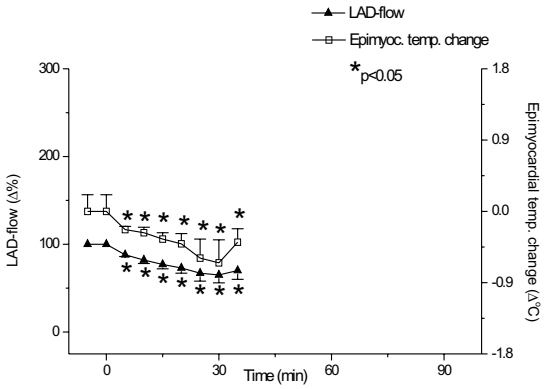
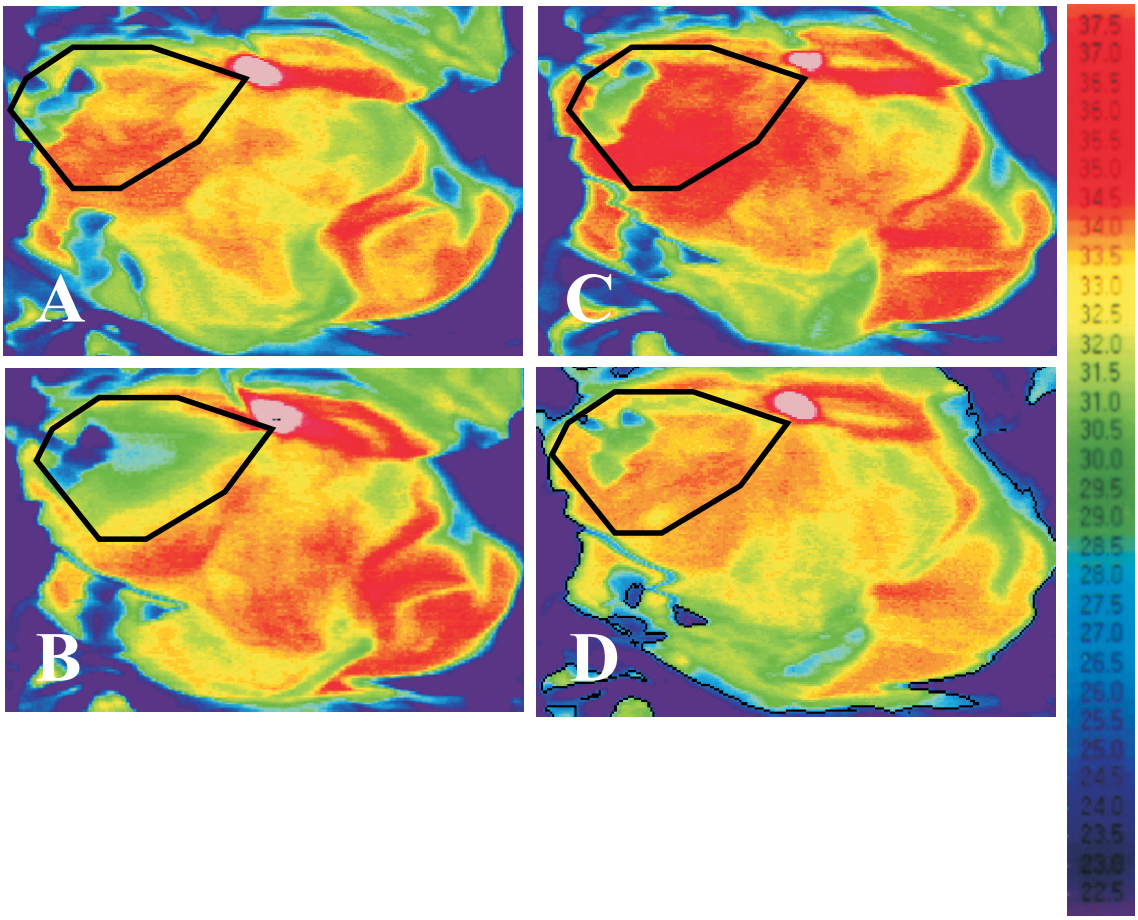


Figure 3
Coronary blood flow and epimyocardial temperature changes during the 60 pmol/min ET-1 infusion. Good correlation was found between the LAD- flow decrease and the epimyocardial temperature decline (r=0.91).

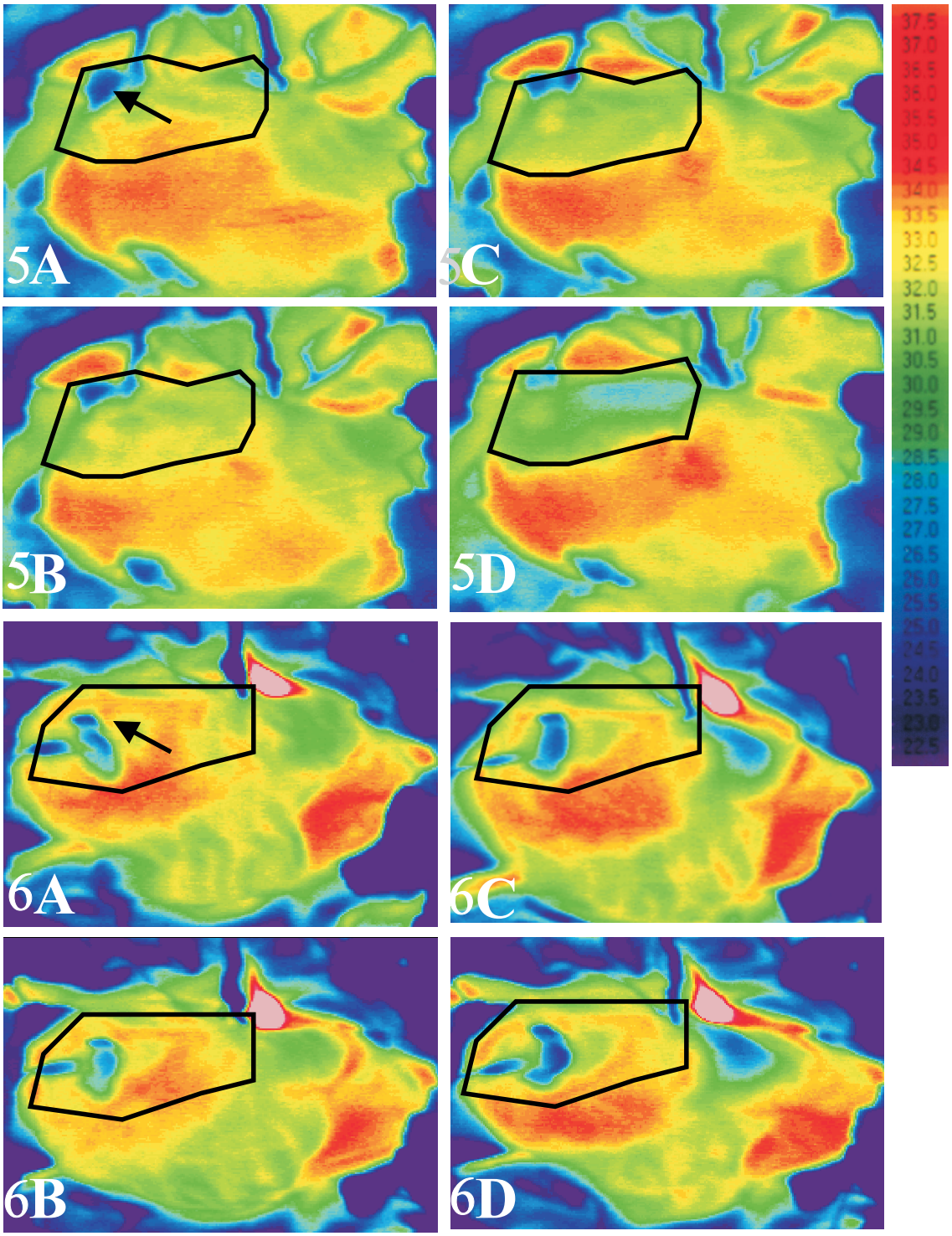
Figure 4
IR thermograms obtained during 30 min LAD occlusion and 60 min reperfusion.
A: control image, B: 30 min LAD occlusion, C: 4 min reperfusion, D: 30 min reperfusion. The LAD area is shown by the solid line. The ischemic area (green spot on B) and the reactive hyperemic response (red spot on C) can clearly be seen



Figures 5 and 6

Thermograms demonstrating the dual nature of endothelin-1 The minority of the animals displayed a homogeneous, circumscribed cold spot (Fig 5/D) that corresponded to the LAD-area (solid line), whereas cooling of the epimyocardium was non-significant in most of the cases (Fig 6/D) despite the observed malignant ventricular arrhythmias.

(Solid arrows indicate left ventricular epicardial monophasic action potential electrodes.)

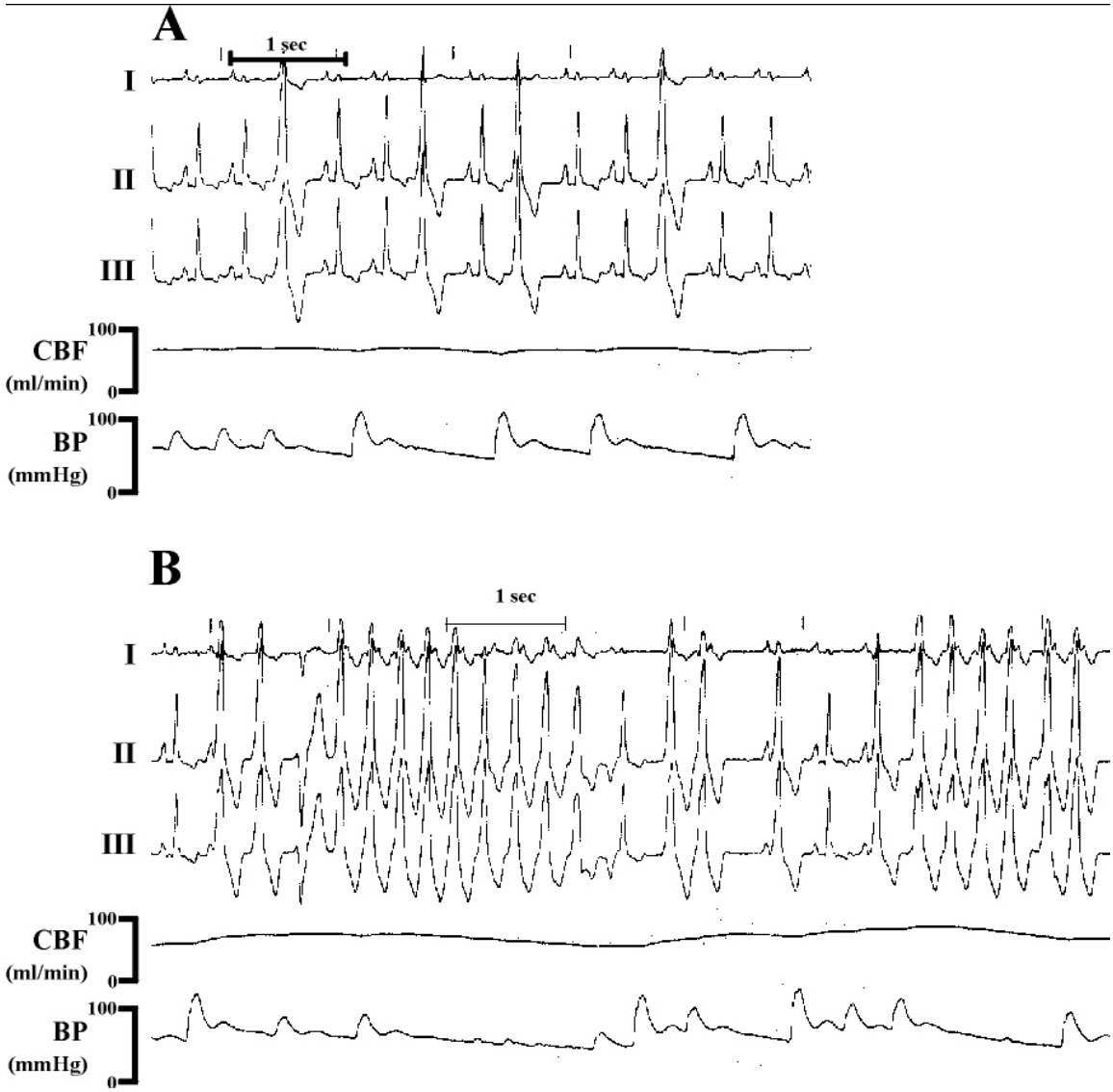


perature of the LAD-area decreased slightly in the first 30 min (ΔT_{30MIN}: -0.15±0.16 °C, p=ns.), but returned to control at the 5th min after terminating the infusion.

Effects of 60 pmol/min intracoronary ET-1 infusion

Coronary blood flow gradually decreased during the ic. ET-1 infusion (control vs. 30 min: 40±3 vs. 27±4 ml/min, $p<0.05$) and increased only slightly after terminating the infusion (Figure 3). In two instances coronary blood flow did not change throughout the ET-infusion despite the observed severe ventricular arrhythmias. Blood pressure and heart rate were unaltered. Significant ST-segment elevations were observed in three instances. Multiple VPC-s were observed in all dogs, VT-s were detected in six cases (Figure 7), Ventricular fibrillation appeared once. In the post ET-1 phase the arrhythmias worsened in all of the remaining seven animals. Five dogs fibrillated in the first 20 min after discontinuing the infusion. Comparing cardiac deaths in the two groups, 16%

Figure 7
30 pmol/min intracoronary ET-1 induced ventricular premature contractions (Panel A).
60 pmol/min ic. ET-1 induced incessant non-sustained ventricular tachycardia (Panel B).



(1/6) of the animals were lost as a result of 30 pmol/min ET-1 infusion, whereas 75% (6/8) exhibited fatal arrhythmias in the 60 pmol/min ET-1 group.

The temperature of the LAD-area decreased significantly during the 60 pmol/min ET-1 infusion (ΔT_{30MIN}:-0.65±0.29 °C, $p<0.05$) and remained at that level until the onset of major and fatal arrhythmias. The epimyocardial cooling of the LAD-area correlated well with the decreasing coronary blood flow values ($r=0.91$, Figures 5, 6).

Discussion

In our study we aimed to compare the effects of a total occlusion of the left anterior descending artery to low dose ic. ET administration and to differentiate between the ischemic and proposed direct arrhythmogenic effect of ET-1. We showed that in the LAD-occlusion group the ar- rhythmias were the consequences of primary LAD ischemia, whereas the development of major ventricular arrhythmias preceded the ischemic coronary blood flow and epimyocardial temperature changes in both ET groups.

The action potential duration (APD) increasing effect of endothelin-1 has been described on canine (2), rabbit (6), and guinea pig (7) cardiomyocytes. ET-1 was found to inhibit ATP-sensitive K^+ current (7), moreover it partially reversed nicorandil and cromakalin induced decreases in action potential duration (8). Therefore, under ischemic conditions, ET-1 may augment myocardial injury via the increase in transmembrane Ca^{++} influx. On the other hand, ET-1 stimulated Na^+/H^+ exchange (9) may also lead to Ca^{++} overload and to the development of afterdepolarizations causing severe ventricular arrhythmias.

The direct proarrhythmic effect of low-dose ic. ET-1 on canine hearts was postulated by Tóth, however the degree of accompanying myocardial ischemia was not clarified (4). In a recent study severe ventricular arrhythmias occurred after intrapericardial infusion of ET-1 (5). In spite of significantly reduced coronary blood flow, no ischemic ECG changes were detected after ic. ET-1 infusion in open-chest mongrel dogs. On the other hand, onset of sudden ventricular faliure was observed after high dose bolus ET-1 despite prior hemodynamic recovery, whereas ET-1 infusion resulted in an increasingly severe decrement in LAD-flow and elevation of left ventricular end-diastolic pressure (10).

Conflicting results concerning the proarrhythmic dose of ET-1 have been reported in earlier studies. Neither escalating ic. bolus doses of ET-1 (1-30 pmol/kg), nor constant ic. ET infusions (5-15 pmol/kg/min) were sufficient to elicit sustained ventricular arrhythmias in pigs (10). In our experiments 1/8-1/4 of the aforementioned ic. ET-1 doses were capable of inducing ventricular fibrillation. The total ET-doses in our study (900 pmol and 1800 pmol) are comparable to those administered by Salvati, when ventricular tachycardia and ventricular fibrillation was observed after cumu-l ative doses of 300, 700 and 1500 pmol ET-1, respectively. ET-1 administered into LAD caused maor ventricular arrhythmias, whereas infusion into the circumflex artery was less arrhythmogenic (11).

In our study, ventricular premature contractions occurred in the first 10 min after starting the ET-infusion followed by VT-s between 20 and 30 min. Severe VT-s and ventricular fibrillation appeared after discontinuing the infusion. Although, 60 pmol/min ET-induced arrhythmias were more severe and of earlier onset then the rhythm disturbances of Group B, neither of them were accompanied by ischemic hemodynamic alterations. These arrhythmias may have been the result of ET-1 accumulation.

The high blood flow rate and the considerable metabolic activity render the myocardium a possible candidate for IR imaging. The first cardiothermographic experiment was performed in 1971 by Senyk, who observed a myocardial surface temperature drop after ligation of the lower descending coronary artery in open-thoracic dogs (12). Some years later Robicsek stated, that the emitted IR radiation of the heart was directly proportional to its temperature, which was directly proportional to the coronary blood flow. He described the thermogram of the healthy heart as a homogeneous warm image and noted that the temperature of the ischemic myocardial scars was significantly lower than that of the normal myocardium (13). It was in our laboratory, where Papp established the quantitative correlation between epimyocardial temperature and coronary perfusion (14). Tzivoni compared epicardial ECG-changes to thermograms and found that a 25% reduction of the coronary blood flow did not af-

fect either the epicardial temperature or ECG, while 50-100% coronary perfusion reduction decreased the surface temperature and resulted in marked ST-elevations (15). Adachi found a significant correlation between the myocardial surface temperature and the regional myocardial blood flow after 90 min of ischemia. By the end of the reperfusion, the myocardial injury (ratio of infarcted myocardium to ventricular wall thickness) appeared to correlate signi- ficantly with surface temperature (16). Mohr applied infrared imaging in 520 patients undergoing coronary artery bypass surgery and a detected 4% early graft failure rate (17). He also performed semiquantitative internal mammary artery (IMA) flow measurements with the method, comparing postoperative digital subtraction angiography to thermography and found an excellent (100%) correlation between these two methods (18). Thermal imaging was also applied during minimally invasive direct coronary artery bypass (MIDCAB) operation, where the patency of an IMA-LAD anastomosis was documented (19).

In our study clear ischemic circumstances were created by total LAD occlusion. Despite being the strongest vasoconstrictor, the epimyocardial cooling caused by 60 pmol/min ET-1 was only half of the temperature decrease caused by LAD-occlusion, moreover the decrease of the surface temperature in the 30 pmol/min ET-1 group was negligible. Two patterns of ET-1-induced thermal response could be observed. All animals in Group B and the majority of the animals in Group C displayed a non- significant epimyocardial temperature decrease during ic. ET-1 infusion. On the other hand, a circumscribed cold spot –strongly reminiscent to LAD-occlusion as seen in Group A- were detected in two cases. This dual thermal response further emphasizes the dual nature of ET-1, namely: vasoconstrictor (cold spot) and direct arrhythmogenic effects.

In summary, the hemodynamic and thermographic data of our study support the notion that the mechanism of ET-induced arrhythmias differs from the pure ischemic arrhythmia-pathogenesis. The most characteristic features at the onset of ET-arrhythmias were: slightly decreased coronary blood flow and epimyocardial temperature. Low dose ET-1 administration precipitated severe ventricular arrhythmias before signs of myocardial ischemia appeared. At the onset of arrhythmias neither ischemic ECG signs nor significantly reduced coronary blood flow with significant epimyocardial cooling were present. The ET-1 induced major ventricular arrhythmias were not proportional to the observed -comparatively moderate- coronary blood flow changes. However, reduced coronary blood flow and focal endocardial ischemia presumably did contribute to sustaining and worsening of the arrhythmias. The fundamentally different characteristics of the observed parameters do not support the ischemic arrhythmogenesis in the two ET groups, and suggest a direct arrhythmia mechanism, however ET-1 induced and ischemic arrhythmias may complement each other under certain pathologic conditions. Therefore ventricular arrhythmias caused by ET-1 could be the prototypes of rhythm disturbances leading to sudden cardiac death.

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The Determination of Normal Temperature Values of Finger Joints

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Summary

Background: Bilateral involvement of body regions occurs frequently in inflammatory rheumatic diseases. In this case, the knowledge of normal temperature values of the affected area is required for correct interpretation of thermal images.

Aim of the study: to determine the possibility of a normal temperature range of the finger joints.

Methods: The hands of 140 subects were investigated by thermal imaging.. 37 patients presented with symptoms of painful osteoarthritis, 21 patients were diagnosed as arthritis. 22 patients suffered from carpal tunnel syndrome (confirmed by nerve conduction studies in all cases), 8 patients presented with symptoms of thoracic outlet syndrome and 10 patients showed Raynaud´s phenomenon in individual fingers. The remaining 42 subjects did not have painful sensations in their hands.

Joint swelling and tenderness of the metacarpophalangeal (MCP), and the proximal (PIP) and distal interphalangeal (DIP) joints were recorded. With the exception of 42 non-symptomatic subjects; a cold water test was performed in all patients and the pattern of temperature recovering was observed. The normal range of temperatures was defined by mean value ± standard deviation and alternatively by the median and the interquartile interval. Temperature readings of each finger joint were related to clinical symptoms such as tenderness and swelling.

Results: In non-symptomatic joints the highest temperature values have been found over the joints of the thumb and the lowest readings on the little finger. The standard deviations were in the range of 1.5 to 2.0 °K. The longitudinal temperature gradient from the MCP to the DIP joint was between -0.4 and 0.8 °K.

Most of the tender joints showed higher temperature readings than non-tender joints. However, the tender interphalangeal-joints of the ring-finger and little finger presented with slightly lower temperatures than non-tender joints. The temperature of all swollen joints and some of the tender joints recovered faster than the other joints after a mild cold challenge..

Conclusion: The definition of the normal range for temperature readings from thermal images of finger joints has not been shown to be clinically useful. Although an overlap in temperatures of tender and non-tender joints exists, hyperthermic changes can be detected by either the disturbance of symmetric temperature distribution from side-to side or by changes of the temperature gradients along individual fingers.

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Die Bestimmung normaler Temperaturwerte der Fingergelenke

Hintergrund: Ein beidseitiger Gelenksbefall ist bei entzündlichen Gelenkerkrankungen häufig. In diesem Fall ist die Kenntnis normaler Temperaturwerte der erkrankten Region für eine richtige Interpretation von Thermogrammen hilfreich.

Ziel der Studie war die Bestimmung des normalen Temperraturbereichs über den Fingergelenken

Methodik: Die Hände von 140 Personen wurden thermographisch untersucht. 37 Patienten boten Symptome einer schmerzenden Fingerpolyarthrose, 21 Patienten litten an einer Arthritis. 22 Patienten hatten ein elektroneurographisch verifiziertes Karpaltunnesyndrom, 8 Patienten hatten ein mildes pseudoneurogenes

Die übrigen 42 Personen gaben keine Schmerzen im Handbereich an.

Gelenkschwellungen und Druckempfindlichkeit der Metakarpophalangealgelenke, der proximalen unddistalen Interphalangeal- (DIP)-Gelenke wurde vermerkt. Mit Ausnahme der 42 beschwerdefreien Personen wurde bei Allen ein milder Kaltwassertest durchgeführt und dabei das Muster der Wiedererwärmung beobachtet. Der Normalbereich der Gelenktemperatur wurde durch den Mittelwert ± Standardabweichung und alternativ als Median und Interquartilabstand definiert. Die Temperaturwerte jedes Fingergelenks wurden mit den klinischen Symptomen Druckempfindlichkeit bzw. Schwellung in Beziehung gesetzt.

Results: An asymptomatischen Gelenken wurden die höchsten Temperaturewerte and den Daumengelenken und die Niedrigsten am Kleinfinger gefunden. Die Standardabweichung bewegte sich zwischen 1.5 to 2.0 °K. Der longitudinale Temperaturgradient vom MCP- zum DIP-Gelenk zeigte Werte zwischen -0.4 und 0.8 °K.

Der gro0ßteil der druckempfindlichen Gelenke war wärmer als nicht druckempfindliche Gelenke.Allerdings druckempfindliche Interphalangealgelenke des Ring - und des Kleinfingers erschienen kühler als nicht symptomatische Gelenke.

Nach einem milden Kaltwassertest erfolgte die Wiederwärmung aller geschwollenen und der meisten druckdolenten Gelenke rascher als bei den anderen Gelenken.

Schlussfolgerung: Die Definition eines normalen Temperaturbereichs der Fingergelenke in Wärmebildern hat sich nicht bewährt. Obwohl eine Überlappung der Temperaturwerte von druckempfindlichen und nicht druckempfindlichen Gelenken besteht, können dennoch Hyperthermien auf Grund der Symmetriestörung von einer Hand zur Gegenüberliegenden bzw. durch Veränderung des typischen Temperaturverlaufs entlang einzelner Finger erkannt werden..

Schlüsselwörter: Normalwert, Thermographie, Arthritis, Arthrose, Raynaudphänomen

Introduction

Symmetric involvement of body regions occurs frequently in inflammatory rheumatic diseases (1). As heat is one of the five principal clinical signs of inflammation, the knowledge of normal temperature values of the affected area is considered to be necessary for the correct interpretation of thermal images. However, reports of normal values of the hands or finger joints are scarce.

Goodman et al. (2) and Uematsu (3) determined the side-to-side temperature difference of the wrist, but both publications did not provide absolute temperature readings of designated areas of the body. Ring reported thermal indices of hands in healthy subjects (4), patients with osteoarthritis and subjects suffering from rheumatoid arthritis, but the regions of interest did not included the fingers. Dieppe et al.(5) published correlations between the thermal index measured over finger joints and degenerative changes in radiographs, but temperature readings were not reported. Acciarri et al. (6) described temperature gradients from the carpus to the fingertips, and similar to Uemetsu (2) the temperature difference between the index and the little finger, but temperature values of individual finger joints cannot be found in their papers. Engel published normal values for the wrist joint and preliminary values for metacarpophalangeal joints (7). In previous studies, mean temperatures of the index and the little finger were determined in healthy subjects and patients suffering from mild pseudo- neurogenic thoracic outlet syndrome (8), and in patients with carpal tunnel syndrome (9). To close the gap in knowledge about normal temperature of finger joints, a retrospective study was conducted with the aim to determine mean temperature values of finger joints.

Method

At the physical examination, joint swelling and tenderness of the metacarpophalangeal (MCP), and the proximal (PIP) and distal interphalangeal (DIP) joints were recorded. Nerve conduction test were performed in all patients suspected of either carpal tunnel or thoracic outlet syndrome.

Thermal images of both hands were recorded after the subjects had acclimatized with bare arms to a room temperature of 24°C for 15 minutes. With the exception of 40 non-symptomatic subjects, a mild cold water test (hands covered with plastic gloves and immersed for 1 minute in water of 20°C) was performed in all patients and the pattern of temperature recovering was observed. Temperature recovery from the finger tips towards the dorsum of hands

Table 1 Biographic data

Diagnosis	Number	Gender		Age mean ±std.dev; median; range
		Female	Male	
All subjects	140	115	25	55.1 ± 17.1; 54.0; 19-91
Healthy subjects	42	32	10	47.7 ± 16.9; 47.5; 19-90
Osteoarthritis	37	35	2	62.9 ± 12.6; 57.0; 46-90
Inflammatory Arthritis	21	16	5	55.1 ± 17.9; 47.0; 29-91
Carpal Tunnel Syndrome	22	14	8	64.2 ± 15.2; 64.5; 30-85
Thoracic Outlet Syndrome	8	8	0	47.5 ± 15.8; 47.0; 24-77
Raynaud Phenomenon	10	10	0	43,4 ± 15.9; 41.5; 20-70

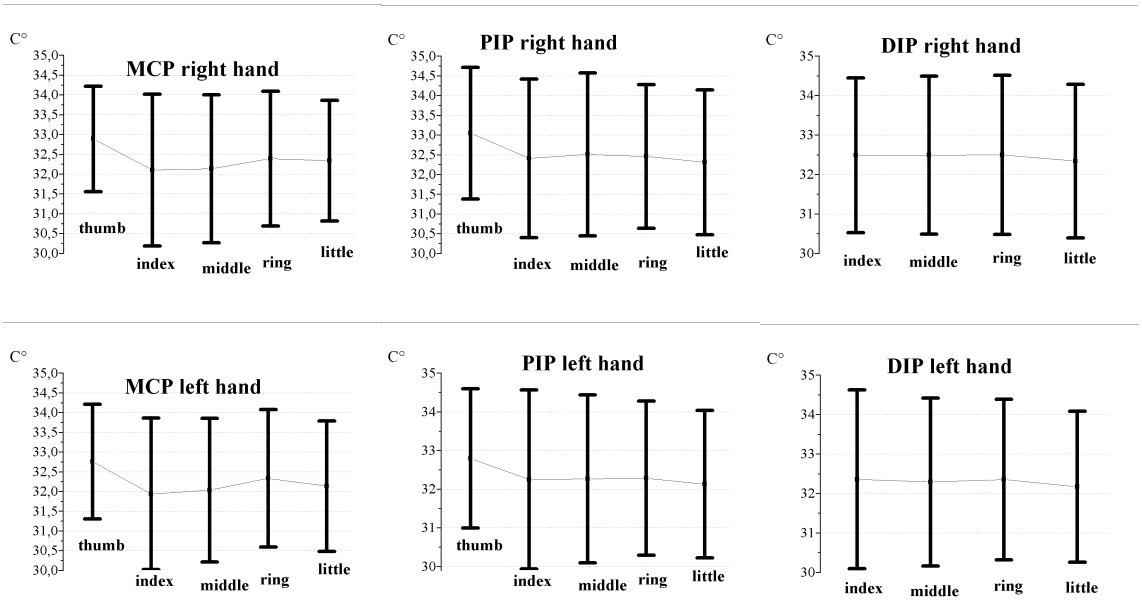
were regarded as normal pattern. Any temperature changes, that did not follow the normal time course of recovering, were interpreted as being the result of local inflammatory heat production.

Rectangular regions of interest were defined over each metacarpophalangeal, the proximal and the distal interphalangeal joints and the mean temperature of this area were recorded for each individual joint. Longitudinal temperature gradients were calculated by subtracting the temperature value of the MCP-joint from the temperature of the DIP-joint. In the group with tender joints, gradients were only calculated if both the MCP and the DIP-joint were recorded as tender.

After the allocation of subjects to six diagnostic groups, the distribution of temperature values of each joint was checked for normal distribution using the Kolmogorov-Smirnov-test. Median value and the inter-quartile interval, mean value, standard deviation (SD) and 95% confidence intervals of the mean were calculated. As not all hand joints are usually involved in degenerative or inflammatory arthritis, temperature readings were assigned either to a group of tender or a group of non-tender joints. Distribution of the values in these two new groups was also checked for normality with the Kolmogorov-Smirnov-test.

Temperatures of each individual joint were analysed with the Mann-Withney test between the six diagnostic groups and also between tender and non-tender joints. All statistical procedures were performed with SPSS for Windows, Version 10.0.

Figure 1
Mean ± standard deviation of temperature of the metacarpophalangeal, proximal and distal interphalangeal joints of healthy subjects



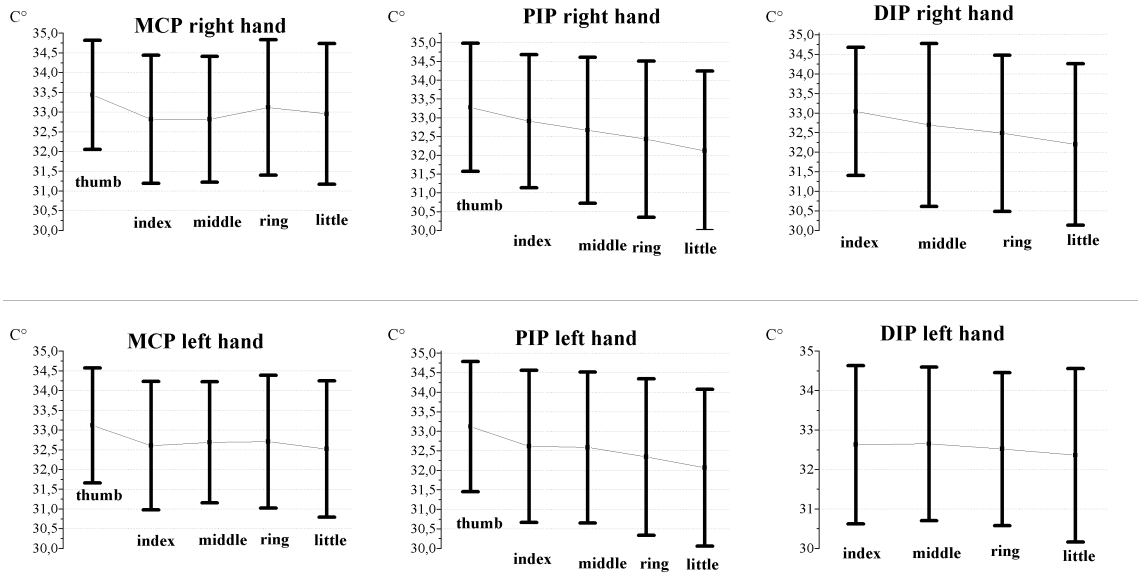


Figure 2
Mean ± standard deviation of temperature of the metacarpophalangeal, proximal and distal interphalangeal joints of patients with osteoarthritis

Results

We investigated the hands of 140 subjects by thermal imaging. 37 patients presented with symptoms of painful osteoarthritis, 21 patients were diagnosed as arthritis. 22 patients suffered from carpal tunnel syndrome (confirmed by nerve conduction studies in all cases), 8 patients presented with symptoms of thoracic outlet syndrome and 10 patients showed Raynaud´s phenomenon in individual fingers. The remaining 42 subjects did not present with clinical signs of affected joints. Biographic data of the investigated sample are shown in table 1.

Healthy subjects

No significant deviation from normal distribution of temperature values of any joint was de-

Figure 3
Mean ± standard deviation of temperature of the metacarpophalangeal, proximal and distal interphalangeal joints of patients with inflammatory arthritis

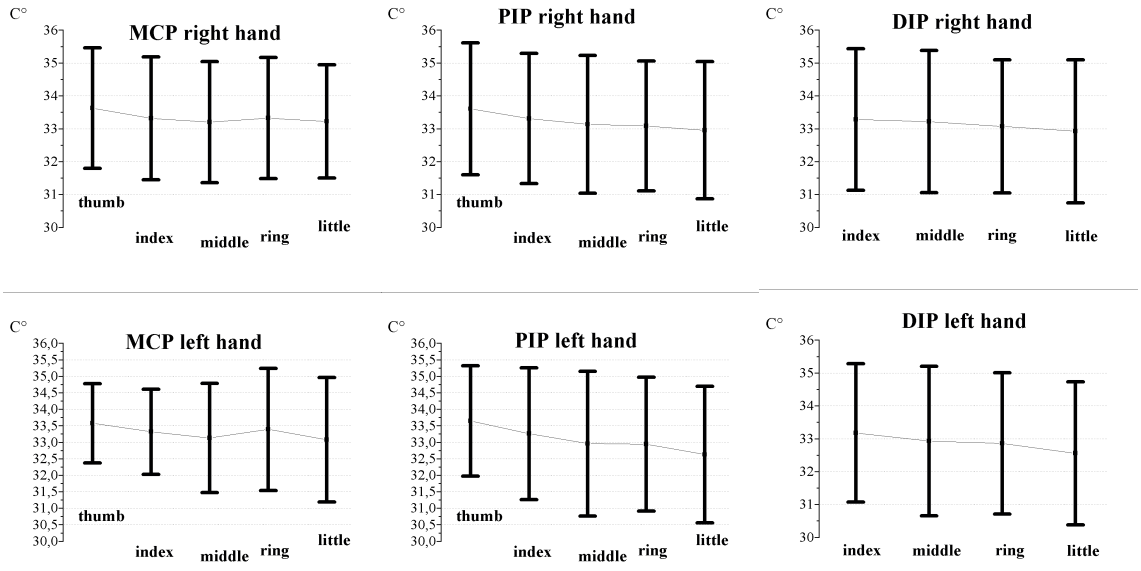


Table 2 Metacarpophalangeal joints,
mean± standard deviation, (95%confidence intervalof mean), median, (interquartile interval)

Joint	Healthy	Osteoarthritis	Inflammatory arthritis	Carpal Tunnel Syndrome	Thoracic Outlet Syndrome	Raynaud`s phenomenon
MCP 1 right	32.89 ±1.33 (32.47 - 33.30) 32.90 (1.88)	33.40 ± 1.39 (32.93 - 33.87) 33.60 (1.68)	33.63 ± .184 (32.79 - 34.46) 34.10 (1.80)	32.98 ±1.68 (32.23 - 33.72) 33.20 (2.48)	33.80 ± 0.75 (33.17 - 34.43) 34.05 (1.25)	31.81 ± 1.47 (30.76 - 32.86) 31.90 (2.05)
MCP 2 right	32.10 ± 1.92 (31.50 -32.70) 32.25 (2.43)	32.81 ± 1.65 (32.25 - 33.37) 33.25 (2.38)	33.32 ± 1.87 (32.47 - 34.17) 33.90 (1.70)	32.48 ± 1.87 (31.65 - 33.31) 32.55 (2.20)	33.43 ± 1.04 (32.56 - 34.29) 33.65 (1.10)	30.86 ± 2.10 (29.36 - 32.36) 30.85 (2.33)
MCP 3 right	32.13 ± 1.87 (31.55 ± 32.72) 32.25 (2.48)	32.81 ± 1.62 (32.26- 33.36) 33.15 (2.48)	33.20 ± 1.84 (32.36 - 34.04) 33.90 (2.00)	32.34 ± 1.76 (31.59 - 33.15) 32.50 (2.08)	33.43 ± 0.92 (32.66 - 34.20) 33.60 (1.25)	31.09 ± 1.94 (29.71 - 32.48) 30.75 (2.68)
MCP 4 right	32.39 ± 1.70 (31.86 - 32.92) 32.40 (2.55)	33.13 ± 1.74 (32.54 -33.71) 33.40 (2.33)	33.32 ± 1.84 (32.48 - 34.16) 33.80 (2.20)	32.65 ± 1.79 (31.85 - 33.44) 32.80 (2.15)	33.44 ± 0.85 (32.73 - 34.15) 33.65 (1.23)	31.74 ± 1.83 (30.43- 33.06) 31.45 (3.00)
MCP 5 right	32.34 ± 1.53 (32.81 - 32.44) 32.40 (2.00)	32.95 ± 1.81 (32.34- 33.56) 33.05 (2.65)	33.22 ± 1.73 (32.44 - 34.01) 33.90 (1.85)	32.51 ± 2.01 (31.61 - 33.40) 32.50 (2.80)	33.29 ± 0.92 (32.52 - 34.05) 33.50 (1.13)	31.48 ± 2.2 (29.89 - 33.07) 31.30 (3.18)
MCP 1 left	32.76 ± 1.45 (32.30 - 33.21) 32.95 (1.48)	33.09 ± 1.47 (32.59 -33.58) 33.35 (1.83)	33.58 ± 1.20 (33.03 - 34.12) 33.90 (1.90)	33.39 ± 1.46 (32.73 - 34.04) 33.75 (2.00)	33.83 ± 0.81 (33.15 - 34.51) 34.10 (0.90)	31.80 ± 1.78 (30.52 - 33.08) 31.50 (2.68)
MCP 2 left	31.94 ± 1.92 (31.34 - 32.54) 32.10 (2.40)	32.58 ± 1.64 (32.02 -33.13) 32.70 (2.10)	33.32 ± 1.29 (32.73 - 33.91) 33.30 (2.20)	32.70 ± 1.53 (32.02 - 33.38) 32.75 (1.68)	33.18 ± 1.15 (32.21 - 34.14) 33.25 (2.35)	30.93 ± 1.89 (29.58 - 32.28) 30.75 (2.90)
MCP 3 left	32.03 ± 1.82 (31.46 - 32.60) 32.20 (2.52)	32.66 ± 1.55 (32.14 - 33.19) 32.85 (1.93)	33.13 ± 1.66 (32.37 - 33.88) 33.50 (2.15)	32.68 ± 1.52 (32.01 - 33.36) 32.70 (1.43)	33.23 ± 1.25 (32.18 - 34.27) 33.60 (2.30)	31.00 ± 1.85 (29.68 -32.32) 30.75 (3.05)
MCP 4 left	32.33 ± 1.74 (31.79 -32.87) 32.40 (2.00)	32.68 ± 1.70 (32.10 - 33.25) 32.80 (2.35)	33.39 ± 1.85 (32.55 - 34.23) 34.10 - 2.40)	32.84 ± 1.66 (32.10 - 33.58) 33.05 (1.70)	33.29 ± 1.28 (32.22 - 34.36) 33.55 (2.45)	31.46 ± 1.70 (30.24 - 32.68) 31.25 (2.65)
MCP 5 left	32.14 ± 1.65 (31.62 - 32.65) 32.35 (2.38)	32.50 ± 1.75 (31.91 - 33.09) 32.75 (2.88)	33.08 ± 1.89 (32.22 -33.93) 33.50 (2.30)	32.73 ± 1.63 (32.01 - 33.45) 32.90 (1.95)	33.03 ± 1.18 (32.04 - 34.01) 33.20 (1.98)	31.20 ± 1.81 (29.91 - 32.50) 31.00 (2.53)

Table 3 Proximal interphalangeal joints
mean ± standard deviation, (95%confidence intervalof mean), median, (interquartile interval)

Joint	Healthy	Osteoarthritis	Inflammatory arthritis	Carpal Tunnel Syndrome	Thoracic Outlet Syndrome	Raynaud`s phenomenon
PIP 1 right	33.05 ± 1.67 (32.53 - 33.57) 33.30 (1.65)	33.22 ± 1.70 (32.65 - 33.79) 33.89 (2.00)	33.61 ± 2.00 (32.69 - 34.52) 34.50 (1.9)	33.02 ± 1.98 (32.14 - 33.90) 33.6 (3.00)	33.85 ± 0.97 (33.04 ± 34.66) 34.05 (1.83)	31.74 ± 1.67 (30.55 - 32.93) 32.20 (1.95)
PIP 2 right	32.41 ± 2.01 (31.78- 33.04) 32.60 (2.18)	32.89 ± 1.79 (32.29 -33.50) 33.50 (2.65)	33.31 ± 1.98 (32.41 - 34.21) 34.10 (2.00)	32.38 ± 2.41 (31.31-33.44) 32.7 (3.65)	33.24 ± 0.85 (32.52 - 33.95) 33.35 (1.28)	30.89 ± 2.55 (29.07 - 32.71) 31.95 (3.65)
PIP 3 right	32.51 ± 2.06 (31.86 - 33.15) 32.70 (2.25)	32.64 ± 1.97 (31.98 - 33.31) 33.15 (3.03)	33.13 ± 2.10 (32.18 - 34.09) 33,80 (2.10)	32.40 ± 2.33 (31.37 - 33.43) 33.3 (3.25)	33.18 ± 0.94 (32.39 - 33.96) 33.40 - (1.65)	31.38 ± 1.71 (30.16 - 32.60) 31.45 (2.63)
PIP 4 right	32.46 ± 1.82 (31.89 - 33.02) 32.60 (2.00)	32.39 ± 2.10 (31.68 - 33.11) 33.15 (2.80)	33.09 ± 1.98 (32.19 - 33.99) 33.80 (2.50)	32.47 ± 2.10 (31.54 - 33.40) 32.95 (3.43)	33.24 ± 1.09 (32.33 - 34.15) 33.50 (1.43)	31.38 ± 1.54 (30.28 - 32.48) 31.15 (2.38)
PIP 5 right	32.31 ± 1.83 (31.74 - 32.88) 32.50 (1.85)	32.08 ± 2.14 (31.35 - 32.80) 33.00 (3.15)	32.95 ± 2.09 (32.00 - 33.90) 33.6 (2.55)	32.33 ± 2.18 (31.36 - 33.30) 32.95 (3.05)	32.98 ± 0.97 (32.17 - 33.78) 33.35 (1.55)	31.02 ± 1.90 (29.66 - 32.38) 31.00 (3.55)
PIP 1 left	32.80 ± 1.80 (32.23 - 33.36) 33.10 (1.85)	33.09 ± 1.68 (32.53 -33.66) 33.55 (2.85)	33.64 ± 1.67 (32.89 - 34.41) 34.20 (1.80)	33.30 ± 1.93 (32.43 - 34.15) 33.75 (2.45)	33.96 ± 0.76 (33.32 - 34.60) 34.15 (1.55)	31.63 ± 1.45 (30.60 - 32.66) 31.70 (2.15)
PIP 2 left	32.25 ± 2.31 (31.53 ± 32.97) 32.65 (2.70)	32.61 ± 1.95 (31.95-33.27) 33.00 (3.20)	33.26 ± 2.00 (32.35 - 34.17) 34.20 (2.45)	32.92 ± 1.91 (32.07 - 33.76) 33.15 (2.43)	33.64 ± 0.81 (32.96 - 34.32) 33.75 (1.28)	31.30 ± 1.68 (30.10 - 32.50) 31.15 (2.38)
PIP 3 left	32.26 ± 2.17 (31.59 - 32.94) 32.80 (2.60)	32.55 ± 1.95 (31.89 - 33.21) 33.00 (2.63)	32.96 ± 2.19 (31.96 - 33.96) 34.00 (3.00)	32.87 ± 1.90 (32.03 - 33.71) 33.25 (2.33)	33.24 ± 1.33 (32.13 - 34.35) 33.40 (2.18)	31.33 ± 1.73 (30.09 - 32.57) 30.90 (2.85)
PIP 4 left	32.28 ± 1.99 (31.66 - 32.90) 32.60 (2.40)	32.32 ± 2.03 (31.63 - 33.00) 32.70 (2.80)	32.94 ± 2.02 (32.02 -33.86) 33.90 (2.00)	32.84 ± 1.91 (32.00 - 33.69) 33.20 (2.45)	33.11 ± 1.33 (32.00 - 34.23) 33.35 (1.93)	31.40 ± 1.60 (30.26 - 32.54) 31.05 (2.98)
PIP 5 left	32.13 ± 1.90 (31.54 - 32.72) 32.50 (2.35)	32.03 ± 2.03 (31.35 - 32.72) 32.65 (2.95)	32.63 ± 2.07 (31.69 - 33.57) 33.40 (2.55)	32.62 ± 1.94 (31.76 - 32.77) 33.35 (2.33)	32.98 ± 1.23 (31.95 -34.01) 32.95 (2.23)	31.18 ± 1.66 (29.99 - 32.37) 30.75 (3.15)

Table 4 Distal interphalangeal joints,
mean ± standard deviation, (95%confidence interval of mean), median, (interquartile interval)

Joint	Healthy	Osteoarthritis	Inflammatory arthritis	Carpal Tunnel Syndrome	Thoracic Outlet Syndrome	Raynaud`s phenomenon
DIP 2 right	32.49 ± 1.96 (31.87 -33.10) 32.60 (1.80)	33.02 ± 1.66 (32.46 - 33.58) 33,50 (2.45)	33.28 ± 2.15 (32.30 -34.26) 34.10 (2.6)	32.52 ± 2.34 (31.48-33.56) 33.10 (3.20)	33.38 ± 1.02 (32.52 - 34.23) 33.90 (1.52)	30.95 ± 2.34 (29.28 - 32.62) 31.70 (4.15)
DIP 3 right	32.49 ± 2.00 (31.87 - 33.12) 32,70 (1.75)	32.66 ± 2.10 (31.95 - 33.37) 33.35 (2.48)	33.22 ± 2.16 (32.23 - 34.20) 34.00 (2.45)	32.57 ± 2.23 (31.58 - 33.56) 33.35 (3.93)	33.08 ± 0.91 (32.31 - 33.84) 33.45 (1.73)	31.27 ± 1.88 (29.93 -32.61) 31.45 (2.63)
DIP 4 right	32.50 ± 2.02 (31.87 - 33.13) 32.75 (2.05)	32.45 ± 2.01 (31.77 - 33.13) 33.15 (2.50)	33.07 ± 2.02 (32.15 - 33.99) 33.80 (2.40)	32.54 ± 2.27 (31.53 - 33.54) 33.05 (3.40)	33.16 ± 1.03 (32.30 - 34.02) 33.55 (1.58)	31.21 ± 1.83 (29.90 - 32.52) 31.5 (2.75)
DIP 5 right	32.34 ± 1.94 (31.73 -32.94) 32.45 (1.94)	32.15 ± 2.08 (31.45 - 32.85) 32.95 (2.90)	32.92 ± 2.18 (31.93 - 33.91) 33.70 (2.55)	32.38 ± 2.11 (31.44 - 33.31) 32.95 (2.88)	32.88 ± 1.05 (32.00 - 33.75) 33.40 (1.75)	30.89 ± 1.94 (29.50- 32.28) 31.05 (3.30)
DIP 2 left	32.36 ± 2.27 (31.65 - 33.06) 32.85 (2.43)	32.63 ± 2.01 (31.95 - 33.31) 33.15 (2.40)	33.18 ± 2.10 (32.22 - 34.13) 34.20 (2.70)	32.98 ± 2.10 (32.00 - 33.86) 33.45 (2.20)	33.39 ± 0.92 (32.62 - 34.16) 33.60 (1.48)	31.33 ± 1.79 (30.05 - 32.61) 31.45 (2.08)
DIP 3 left	32.29 ± 2.13 (31.63 - 32.95) 32.65 (2.70)	32.61 ± 1.96 (31.95 - 33.28) 33.20 (2.80)	32.93 ± 2.27 (31.89 - 33.96) 33.9 (2.80)	32.91 ± 1.96 (32.04 - 33.78) 33.15 (2.35)	33.11 ± 1.22 (32.10 - 34.13) 33.15 (2.08)	31.31 ± 1.77 (30.05- 32.57) 31.25 (1.83)
DIP 4 left	32.35 ± 2.04 (31.72 - 32.99) 32.55 (2.28)	32.48 ± 1.95 (31.82 - 33.14) 32,70 (2.30)	32.86 ± 2.15 (31.88 - 33.84) 33.30 (2.30)	32.95 ± 1.93 (32.10 - 33.81) 33.20 (2.25)	33.10 ± 1.24 (32.06 ± 34.14) 33.20 (1.88)	31.38 ± 1.85 (30.06 - 32.70) 31.35 (3.05)
DIP 5 left	32.17 ± 1.92 (31.58 -32.77) 32.65 (2.13)	32.33 ± 2.22 (31.57 -33.08) 32.70 (2.65)	32.56 ± 2.18 (31.57 - 33.55) 33.30 (2.25)	32,68 ± 1.57 (31.85 - 33.51) 33.15 (2.43)	32.96 ± 1.12 (32.03 - 33.90) 32.90 (1.73)	31.20 ± 1.62 (30.04 - 32.36) 31.35 (2.30)

Table 5 Longitudinal gradient,
mean± standard deviation, (95% confidence interval of mean), median, (interquartile interval)

finger	healthy	osteoarthritis	Inflammatory arthritis	Carpal Tunnel Syndrome	Thoracic Outlet Syndrome	Raynaud`s phenomenon
1st right	-0.16 ± 0.90 (-0.44 - 0.12) -0.25 (0.6)	0.18 ± 0.74 (-0.07 - 0.43) 0.15 (0.93)	0.02 ± 0.61 (-0.26 - 0.30) 0.00 (0.80)	-0.04 ± 0.63 (-0.32 - 0.24) -0.15 (0.80)	-0.05 ± 0.76 (-0.69 - 0.59) 0.20 (0.48)	-0.07 ± 1.15 (-0.75 -0.89) 0.00 (1.98)
2nd right	-0.39 ± 1.41 (-0.82 - 0.05) -0.40 (1.18)	-0,21 ± 0,78 (-0.47 - 0.06) -0.20 (0.68)	0.04 ± 0.79 (-0.32 - 0.40) -0.10 (0.95)	-0.04 ± 1.20 (-.57 - 0.49) -0.05 (1.28)	-0.05 ± 0.70 (-0.53 - 0.63) -0.05 (1.03)	-0.09 ± 1.24 (-0.98 - 0.80) -0.7 (2.15)
3rd right	-0.36 ± 1.44 (-0.80 - 0.09) -0.40 (1.23)	0.15 ± 0.85 (-0.14 - 0.44) -0.05 (0.58)	0.02 ± 0.77 (-0.37 - 0.33) -0.20 (0.85)	-0.21 ± 0.91 (-0.61 - 0.20) -0.10 (1.00)	0.35 ± 0.49 (-0.06 0.76) 0.40 (0.73)	-0.18 ± 1.77 (-1.45 - 1.09) 0.10 (2.85)
4th right	-0.11 ± 1.33 (-0.52 - 0.30) -0.10 (1.13)	0.68 ± 1.94 (0.02 - 1.33) 0.25 (0.83)	0.25 ± 0.48 (0.03 -0.47) 0.2 (0.45)	0.11 ± 0.68 (-0.19 - 0.41) 0.10 (0.75)	0.28 ± 0.42 (-0.08 - 0.63) 0.30 (0.63)	0.53 ± 1.51 (-0.55 - 1.61) 0.45 (1.68)
5th right	0.00 ± 1.00 (-0.31 - 0.31) -0.05 (1.03)	0.78 ± 0.197 (0.13 - 1.46) 0.35 (0.80)	0.30 ± 0.63 (0.02 - 0.59) 0.20 (0.55)	0.13 ± 0.58 (-0.13 - 0.38) 0.10 (0.83)	0.41 ± 0.57 (-0.07 - 0.89) 0.50 (0.75)	0.59 ± 1.42 (-0.42 - 1.60) 0.45 (1,7)
1st left	-0.04 ± 0.80 (-0.29 - 0.21) -0.15 (0.60)	0.01 ± 0.86 (-0.30 - 0.29) -0.10 (0.93)	-0.07 ± 0.74 (-0.41 - 0.26) -0.10 (0.75)	0.10 ± 0.95 (-0.33 - 0.52) -0.05 (0.58)	-0.14 ± 0.57 (-061 - 0.34) -0.20 (1.03)	-0.17 ± 1.33 (-0.78 - 1.12) -0.10 (1.43)
2nd left	-0.42 ± 1.38 (-0.85 - 0.01) -0.65 (1.33)	-0.05 ± 0.71 (-0.29 - 0.19) -0.15 (0.75)	0.14 ± 1.29 (-0.44 - 0.73) -0.10 (0.95)	-0.23 ± 0.86 (-0.62 - 0.15) -0.30 (1.05)	-0.21 ± 1.0 (-1.05 - 0.62) -0.05 (1.48)	-0.40 ± 2.10 (- 1.90 - 1.10) -0.20 (2.55)
3rd left	-0.26 ± 1.43 (-0.71 - 0.19) -0.40 (1.18)	00.5 ± 0.88 (-0.25 - 0.35) -0.15 (1.1)	0.20 ± 0.99 (-0.25 - 0.65) 0.00 (1.10)	-0.23 ± 0.84 (-0.69 - 0.15) -0.25 (1.15)	0.11 ± 0.65 (-0.43 - 0.66) 0.20 (1.10)	-0.31 ± 1.64 (-1.48 - 0.86) -0.10 (1.70)
4th left	-0.02 ± 1.20 (-0.40 - 0.35) -0.1 (0.93)	0.20 ± 1.09 (-0.17 - 0.57) 0.20 (0.85)	0.53 ± 0.77 (0.18 - 0.89) 0.40 (0.95)	-0.11 ± 0.77 (-0.45 -0.23) -0.15 (0.83)	0.19 ± 0.54 (-0.27 - 0.64) 0.30 (0.60)	0.08 ± 1.60 (-1.07 - 1.23) 0.35 (1.30)
5th left	-0.04 ± 1.15 (-0.39 - 0.32) -0.15 (1.28)	0.18 ± 1.95 (-0.49 - 0.84) 0.30 (1.08)	0.52 ± 0.79 (0.16 - 0.88) 0.60 (1.00)	0.05 ±0.81 (-0.32 - 0.41) 0.05 (0.85)	0.06 ± 0.58 (-0.42 ± 0.55) 0.25 ((0.88)	0.00 ± 1.72 (-1.23 - 1.23) 0.05 (0.83)

tected. Figure 1 shows mean values ± standard deviation of all MCP, PIP and DIP- joints of healthy subjects. The respective temperature readings of MCP-joints appear in table 2. Values of PIP- joints are shown in table 3 and of DIP joints in table 4. Finally table 5 gives the values of the longitudinal temperature gradient of each finger. There is a highly symmetric temperature distribution on the fingers. The side-to side differences for individual joints varies within a narrow range (MCP: 0.06 to 0.24 °K, PIP: 0.16 to 0.25 °K, DIP: 0.01 to 0.2 °K, Longitudinal Gradients: 0.04 to 0.15° K). Highest readings were found at the interphalangeal joint of the thumb. The difference between the mean temperature of the index (calculated by the sum of the temperatures of the 3 joints divided by 3) and the little finger was 0.0 °K for the right hand and 0.14°K for the left hand.

Osteoarthritis

All joint temperatures measured followed a normal distribution, but the longitudinal gra-

Table 6 Non tender joints,
mean± standard deviation, 95% confidence interval of mean, median, interquartile interval

MCP 1 right (n=123)	MCP 2 right (n=121)	MCP 3 right (n=119)	MCP 4 right (n=128)	MCP 5 right (n=129)	MCP 1 left (n=127)	MCP 2 left (n=122)	MCP 3 left (n=125)	MCP 4 left (n=128)	MCP 5 left (n=130)
33.0 ± 1.6 32.8 - 33.3 33.3 (2.1)	32.4 ± 1.9 32.1 - 32.8 32.7 (2.5)	32.4 ± 1.8 32.1-32.7 32.5 (2.7)	32.7 ± 1.8 32.4 -33.0 32.8 (2.4)	32.6 ±1.8 32.3 -32.9 32.8 (2.4)	33.0 ± 1.5 32.8 -33.3 33.3 (1.9)	32.3 ± 1.8 32.0 - 32.6 32.5 (2.3)	32.4 ± 1.7 32.1-32.7 32.6 (2.2)	32.6 ± 1.8 32.3-32.9 32.7 (2.3)	32.4 ± 1.7 32.1-32.7 32.6 (2.6)
IP 1 right (n=119)	PIP 2 right (n=113)	PIP 3 right (n=114)	PIP 4 right (n=117)	PIP 5 right (n=120)	IP 1 left (n=122)	PIP 2 left (n=116)	PIP 3 left (n=120)	PIP 4 left (n=120)	PIP 5 left (n=123)
33.1 ± 187 32.8-33.5 33.6 (2.1)	32.5 ±2.1 32.1 -32.9 32.8 (2.4)	32.5 ± 2.1 32.1-32.9 33.0 (2.5)	32.5 ± 2.0 32.2-32.9 32.9 (2.6)	32.4 ± 2.0 32.0-32.7 32.9 (2.4)	33.0 ± 1.8 32.7-33.4 33.4 (2.3)	32.5 ± 2.1 32.1 -32.9 33.0 (2.8)	32.4 ± 2.1 32.1 -32.9 32.9 (2.8)	32.5 ± 2.0 32.1-32.8 33.0 (2.8)	32.3 ± 2.0 31.9-32.6 32.8 (2.9)
	DIP 2 right (n=114)	DIP 3 right (n=118)	DIP 4 right (n=121)	DIP 5right (n=117)		DIP 2 lef (n=120)	DIP 3 left (n=123)	DIP 4 left (n=124)	DIP 5 left (n=120)
	32.7 ± 2.0 32.4-33.1 33.0 (2.3)	32.6 ± 2.1 32.3-33.0 33.0 (2.3)	32.6 ± 2.0 32.2 - 32.9 33.1 (2.3)	32.3 ± 2.0 31.9-32.7 32.6 (2.1)		32.5 ± 2.2 32.1-32.9 33.1 (2.8)	32.5 ± 2.1 32.2-32.9 33.0 (2.6)	32.6 ± 2.0 32.2-32.9 33.0 (2.6)	32.3 ± 2.0 31.9-32.6 32.8 (2.2)
G1 right (n=114)	G2 right (n=106)	G3 right (n=107)	G4 right (n=114)	G5 right (n=112)	G1 left (n=117)	G2 left (n=109)	G3 left (n=113)	G4 left (n=117)	G5 left (n=114)
-0.02 ± 0.8 -0.16-0.13 -0.0 (0.8)	-0.26 ±1.2 -0.49-0.04 -0.3 (1.13)	-0.16 ±1.2 -0.40-0.07 -0.1 (1.0)	0.20 ± 1.5 -0.07-0.47 0.1 (0.83)	0.30 ± 1.4 0.05-0.56 0.20 (0.9)	0.00 ±0.8 -0.16-0.15 -0.1 (0.6)	-0.24± 1.2 -0.48-0.01 -0.3 (1.1)	-0,12 ±1,2 -0.34-0.10 -0.1 (1.1)	0.07 ±1.1 -0.13-0.27 0.0 (0.9)	0.16 ± 1.0 -0.03-0.36 0.1 (1.1)

Table 7 Tender joints,
mean± standard deviation, 95% confidence interval of mean, median, interquartile interval

MCP 1 right (n=16)	MCP 2 right (n=19)	MCP 3 right (n=21)	MCP 4 right (n=12)	MCP 5 right (n=11)	MCP 1 left (n=13)	MCP 2 left (n=17)	MCP 3 left (n=12)	MCP 4 left (n=12)	MCP 5 left (n=10)
33.9 ± 0.9 33.4 -34.4 34.0 (0.93)	33.1 ± 1.9 32.2-34.0 33.9 (2.7)	33.1 ± 1,7 32.3 -33.8 33.5 (1.))	33.3 ± 1.1 32.6-34.0 22.4 (1.7)	33.2 ±1.1 32.5-33.9 33.1 (1.7)	33,5 ± 0.9 33.0-34.0 33.7 (1.7)	33,2 ± 1.3 32.5-33.9 33.0 (2.1)	33.2 ±1.0 32.5-33.8 22.2 (2.0)	33.7 ± 1.1 33.0-34.4 33.8 (1.7)	33.4 ± 1.5 32.3-34.4 33.2(1.6)
IP right (n=21)	PIP 2 right (n=27)	PIP 3 right (n=25)	PIP 4 right (n=23)	PIP 5 right (n=20)	IP left (n=17)	PIP 2 left (n=21)	PIP 3 left (n=19)	PIP 4 left (n=20)	PIP 5 left (n=17)
33.1 ± 1.8 32.3 - 34.0 33.3 (2.9)	33.1± 1.8 32.4-33.8 33.8 (2.1)	32.9 ± 1.8 32.2-33.7 33.6 (2.3)	32.3 ± 1.8 31.6 -33.1 32.4 (2.4)	31.9 ± 1.8 31.0-32.7 32.2 (3.3)	33,4 ± 1.5 32.6-34.2 33.7 (1.5)	33.3 ± 1.5 32.6-34.0 33.7 (1.9)	33.0 ± 1.5 32.3-33.2 33.4 (1.6)	32.5 ± 1.7 31.7-33.3 33.0 (2.3)	32.3 ± 1.6 31.5-33.1 32.7 (1.7)
	DIP 2 right (n=26)	DIP 3 right (n=22)	DIP 4 right (n=19)	DIP 5 right (n=23)		DIP 2 left (n=19)	DIP 3 left (n=17)	DIP 4 left (n=16)	DIP 5 left (n=20)
	32.5 ± 2.1 31.7-33.4 33.2 (2.7)	32.4 ± 2.1 31.5-33.3 32.9 (3.2)	32.4 ± 1.9 31.5-33.3 32.6 (3.5)	32.4 ±2.1 31.5-33.3 32.8 (3.4)		33.3 ± 1.3 32.6-33.9 33.2 (2.4)	32.6 ± 1.7 31.8-33.5 33.0 (2.2)	32.4 ± 1.7 31.5-33.3 32.4 (1.9)	32.8 ±2.1 31.8-33.8 32.8 (2.3)
G1 right (n=12)	G2 right (n=19)	G3 right (n=10)	G4 right(n=4)	G5 right (n=6)	G1 left (n=8)	G2 left (n=7)	G3 left (n=4)	G4 left (n=6)	G5 left (n=3)
0.23 ±0.6 -0.18 ± 0.6 0.05 (0.8)	0.06 ± 0.8 -0.35-0.47 0.0 (0.9)	0.1 ± 0.7 -0.40 - 0.6 -0.3 (0.9)	0.48 ± 1.0 -0.75-1.71 0.1 (1.5)	0.57 ± 0.9 -0.4-1.5 0.25 (0.9)	-0.36 ±0.7 -0.92-0.20 -0.25 (0.5)	-.14 ± 0.3 -0.45-0.16 0 (0.5)	0.03 ± 0.8 -1.26-1.31 0.1 (1.5)	0.53 ± 0.9 -0.43-1.50 0.25 (1.4)	0.7 ± 0.5 -0.54-1.94 0.7 (0)

dients of 3rd and 4th finger on the right hand side and the 4th and 5th finger on the left hand side were significantly different from a normal distribution of values. The temperatures of joints of osteo-arthritic patients (Figure 2) were similar to the values of healthy subjects. At the MCP joint of the right thumb a significantly higher temperature (2-tailed p: 0.04) was found in osteoarthritrs patients than in healthy subjects. The longitudinal temperature gradients at the right thumb, ring and little finger and also at the left index and little finger were also significantly different.

Inflammatory arthritis

Most joint temperature followed normal distribution, but the temperature readings of MCP 3 and PIP 3 of the right hand and of DIP2 at the left hand were outside the normal distribution. The mean temperatures of most joints of patients with inflammatory arthritis were above the temperature readings of healthy subjects. Only at the following locations the difference did not reached the 2-tailed significance level of 0.05: PIP joint and DIP joint of the right ring finger, DIP joint of the right little finger; on the left hand the pip joints 3-5, and DIP joints 2-5. The longitudinal gradients of both ring fingers were significantly different in both groups.

Carpal Tunnel Syndrome

All joint temperatures appeared normally distributed. No significant difference was detected at any joint in both groups, but patients with carpal tunnel syndrome showed slightly warmer joints than healthy subjects.

Thoracic outlet syndrome

All joint temperatures followed normal distribution. The joint temperatures of patients with thoracic outlet syndrome were found to be higher than those of healthy subjects. This temperature difference was significant at the MCP-joints 1& 3, right hand side and the interphalangeal joint of the left thumb.

Raynaud´s phenomenon

All joint temperatures appeared to be normally distributed. Although none of the Raynaud patients presented with Raynaud´s phenomenon of all fingers, all the finger joints of patients with this condition showed lower temperatures than healthy subjects. At 13 joint locations the temperature difference crossed the significance level for 2-tailed $p<0.05$. The mean value of the longitudinal gradient did not differ significantly between healthy subjects and patients with Raynaud´s phenomenon in individual fingers, but showed a higher standard deviation and in-

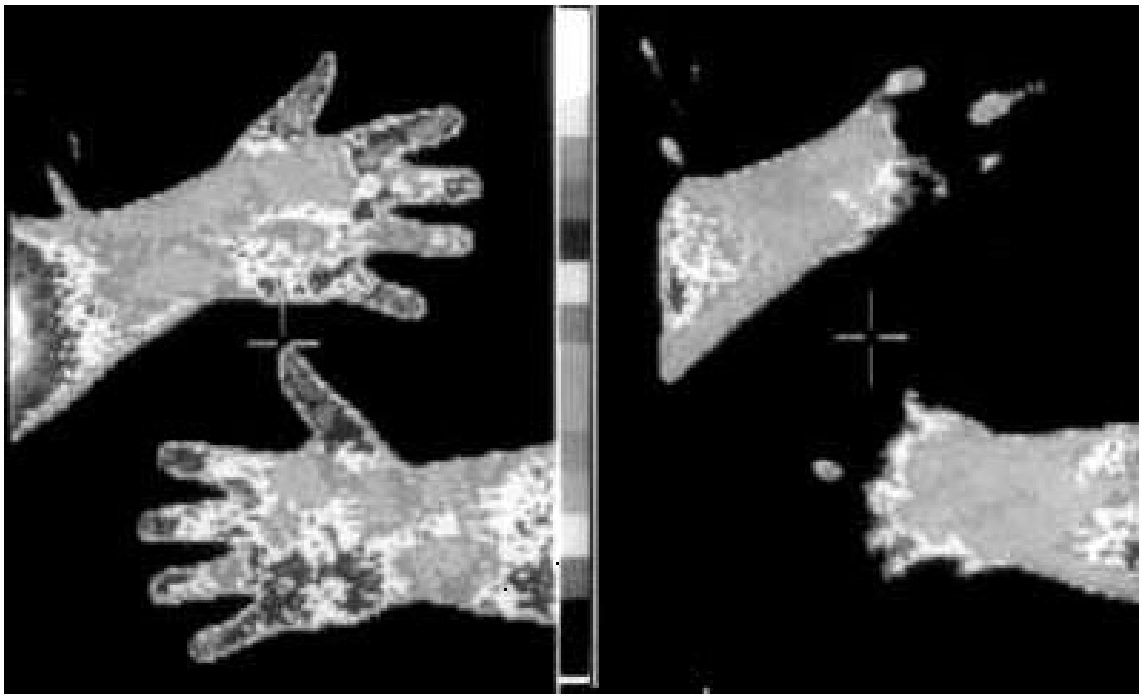


Figure 4
Premature temperature recovery after mild cold challenge. Within 30 seconds after immersion of both hands, coverd by plastic gloves in a water bath og 20 °C for 6o seconds, the MCP joints 1 and 5, the PIP-joints 3 and 5 of the right hand and the left PIP 3 showed already up in this very early phase of temperature recovery.

terquartile interval in patients with vasospastic symptoms than in healthy subjects.

Non tender joints

Most joints presented without tenderness. Slightly higher temperature readings were obtained over the non-tender joints in patients than those in healthy subjects.

Tender joints

Tender joints presented with higher temperatures than non-tender joints. The readings of the MCP-joints 1 to 3, and the PIP joint 2 on the right hand side and of the MCP of the left index were significantly different at the 2-tailed p-level <0.05. However, the tender interphalangeal-joints of the ring-finger and the little finger presented with slightly lower temperatures than non-tender joints.

Temperature recovery after mild cold challenge

The temperature of all swollen joints and some of the tender joints recovered faster than the other joints after a cold challenge. Figure 4 shows a typical case of premature temperature recovery of tender MCP and PIP-joints.

Discussion

This study confirms higher joint temperatures of the thumb than all other fingers. Combining joint temperatures of index and little finger for defining the temperature of the entire finger, resulted in nearly identical values of both fingers. This finding is in contrast to other authors (2,5) and own previous studies (8), where a temperature difference of 0,5°K between index and little finger was found in healthy subjects. However, in all other studies the temperature of the entire finger was derived from regions of interest encompassing the finger from the tip to the MCP-joint, whereas in this study only the area over the joints was used for the determination of the temperature of the entire finger.

The longitudinal gradient of fingers was between -0.4 and 0.8 °K, with some significant differences in individual fingers between groups. A comparison with values reported by other authors is not possible due to different methods of defining the longitudinal gradient and location of measuring sites. Ring (10) compared the gradient from two regions of interest: one at the dorsal hand and the other enclosing the fingers 2-5. Tauchmannova et al. reported in several studies (11,12, 13) temperature gradients of individual fingers from the dorsal hand to the fingertips. A group at the Rehabilitation Clinic of the University Hospital Vienna (14) calculated gradients from the radial wrist to the fingertips of 2nd to 4th finger. In a previous study, gradients were determined from the MCP- joints to the fingertips (15). All these studies reported different cut off points of normal gradients. Positioning of regions of interest in areas of joints might result in false negative results of longitudinal temperature gradients, as heat from inflamed joints may mask an established vaso-s pastic disease. The wide confidence interval of the mean of gradients of individual fingers with tender joints can be interpreted in the aforementioned way.

When the data distribution is normal, or nearly so, it is possible to calculate that 68.3% of the data lies ± 1 SD from the mean. The lower and upper values of the interquartile interval represent the 25th and 75th percentile, and the median the 50th (16). The "normal range" corresponds to the interval that includes 95% of the data. This is equal to 1.96 SD on either side of the mean but is often approximated to mean ±2SD. Figures 1-3 show clearly that an overlap of temperature values exists already visible at the level of 68% of all data between the diagnoses groups. This overlap is still there when using the interquartile interval, which shows a magnitude between 1.5 and 3.2°K. Defining lower and the upper cut-of- point by 95% of data, would result in temperature values at the upper limit which are only available in case of fever or very hot environment. For example, in healthy subjects the right interphalangeal joint of the thumb has a mean value 33.05 C °, plus two fold SD (1.67 X 2= 3.34) results in an upper limit of 36.39°C. Defining a value for a lower limit of normality is questionable as low temperatures are not necessarily the expression of vasospastic disease. The positive predictive value of cold fingertips for a maintaining a negative longitudinal temperature gradient after cold challenge was only 58,5 % in a previous study (17).

The wide overlap of joint temperatures from healthy subjects and diseased patients might be partially due to the fact that hand symptoms may be present for several possible reasons. Not all conditions will produce changes of the skin surface temperature over finger joints. Inflammatory arthritis will present with clear hyperthermia, swelling and tenderness, and osteo-

arthritis will produce tenderness combined with only slight temperature changes. The MCP-j oints 1-3 are known as typical sites of inflammation in rheumatoid arthritis patients. Significantly different temperature levels in tender and non-tender MCP-joints 1-3 might be caused by the symptoms of patients with rheumatoid arthritis within the diagnosis group named "Inflammatory Arthritis". However, pain and tenderness and even swelling can occur without symptoms of disturbed temperature as shown in patients with knee pain (18).

Another source of the large variation of finger joint temperatures may be the lack of standards for performing thermal images of the hands. Electronic images are matrices of a limited number of pixels. Therefore when positioning an object for measurement using an electronic imaging method, care must be taken to ensure that the maximum information can be obtained by using as large an area of the matrix as possible before image capture. This is not always followed, so non-standardised positions together with the lack of standards for regions of interest may contribute to the variation of temperature values obtained. One of the authors has recently defined standard views for various body regions (19). This protocol describes anatomical landmarks and specific distances on the object that must be included within the image. These views when tested by different investigators have been shown to be easily reproducible with high accuracy. Regions of interest on thermal images recorded in the aforementioned way have been defined by shape and the relationship of the shape to anatomical landmarks. The evaluation of the reproducibility of temperature measurements on the anterior arm was obtained for 5 newly trained investigators, and resulted in deviations from the mean temperature of the region of interest between 0.001 and 0.10 °K at the elbow, between 0.06 and 0.27 at the upper arm and between 0.02- and 0.1 at the upper arm. The building of this database, which will include 30 regions of interest on all fingers, is still under progress. The study will be open for contributions from other centres if thermal imaging has been performed according to the protocol (20).

The application of a cold challenge and observation of the pattern of temperature recovery in the hand could be a more useful technique. It would overcome the problems of wide range of normal static temperature readings obtained from finger joints. Premature temperature recovery of swollen joints occurs regularly, but can also be found in most of tender joints. Premature temperature recovery is defined by achieving baseline temperatures of a distinct joint before the finger distal from that joint had shown re-warming.

This study based on the thermal images of the hands of 140 subjects, has shown that defining the "normal range "failed" to be of value for individual temperature readings measured from these thermal images. Although an overlap in temperatures of tender and non-tender joints exists, hyperthermic changes can be detected by either the disturbance of symmetric temperature distribution from side-to side or by changes of the temperature gradients along individual fingers. Additional information can be derived from observation of temperature recovery after a mild cold challenge. Evaluation of finger temperatures from the reference database of normal thermograms of the human body might ultimately solve the problem of being able to establish a normal range for finger joint temperatures in the near future.

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News in Thermology

15th Symposium of the Austrian Society of Thermology

In the end of April the 15th Thermological Symposium of the Austrian Society of Thermology and the Ludwig Boltzmann Research Institute for Physical Diagnostics will be held in Vienna. The main theme of the meeting is "Standards in Thermology" and speakers from Great Britain, Italy, Poland, Slovakia and Austria have already expressed their agreement to attend and to present their work. In contrast to the main theme, the symposium will change its standard venue, which served very well for 13 of the 14 previous meetings. This year the conference will move from the SAS Hotel to the Business Center of one of the famous Austrian cellars for producing sparkling wine, located in the 19th district of Vienna. This venue is easily to access by public transport. A tour through the wine cellar will close this year´s meeting.

3rd Instructional Course on Thermal Imaging in Medicine

After two successful courses on Thermal Imaging in Medicine in July and November 2001, requests were made recently for another course within a period of 8 months. The 3rd Short Course February will be held on February 13-15, 2002 at the School of Computing of the University of Glamorgan in Pontypridd, Wales, UK. Dr K Ammer, Prof F Ring and Dr P Plassmann will lecture on the theoretical and historical basis of thermal imaging in medicine, clinical applications and future developments of thermal imaging in medicine. A supervised practical session is included which focuses on the capture and analysis of images. Registration Fee is £300. Cheques should be made payable to The University of Glamorgan. The Fee includes lunch and refreshment breaks, the hardback book -The Thermal image In Medicine and Biology, and a CD of Archived IR Imaging in Medicine publications, The course is recognized by The University and certificates will be issued to all who complete the short course.

Further information can be obtained from
Prof Francis Ring
(01443 483717 , e-mail efring@glam.ac.uk) or

Dr Peter Plassmann
(01443 483486, e-mail pplassma@glam.ac.uk)

School of Computing, University of Glamorgan, Pontypridd, CF37 1DL

Meeting of the Medical Thermology Group in the UKTA

On behalf of the Thermology Group in the UKTA, Mr. Kevin Howell will organize two one day meetings on May 17 and 18, 2002 at the Sheila Sherlock Education Centre, Royal Free Hospital, London, UK.

The Scientific committee, Prof. C.M. Black, Dr. C.P. Denton, Prof. E.F.J. Ring, Dr. F. Khan, Mr. K. Howell, has chosen "Physiological measurement of Raynaud's phenomenon and peripheral microvascular disorders: from research into clinical practice" as main theme of the meeting on Friday 17th May 2002. Raynaud's phenomenon (RP) is problematic both for the clinician and for the clinical scientist charged with evaluating the condition in the laboratory. No universally accepted standard exists for the clinical definition of RP, making the evaluation of physiological measurement techniques in this field especially difficult. The meeting aims to bring together specialists in the physiological measurement of RP and associated peripheral microvascular disorders, along with clinicians experienced in the field, to discuss recent advances in the assessment of the microcirculation. The provisional programme contains the following topics:

- The clinical diagnosis and classification of RP

- Erythromelalgia
- Hand-arm vibration syndrome and work-related upper limb disorders
- Capillaroscopy
- Thermography and skin temperature measurement
- Laser-doppler flowmetry and perfusion imaging
- Photoplethysmography
- Measurements of tissue oxygenation
- Physiological measurement of RP in clinical practice
- Physiological measurement of RP in epidemiological studies
- Physiological measurement of RP in research and clinical trials

On Saturday 18th May 2002, "Medical Infrared Thermography" is the theme of the other meeting. From its inception in the 1960s the medical use of infrared thermography has grown steadily in the UK and Europe. In recent years greatly improved instruments at competitive prices have opened up new opportunities in medical infrared imaging. This meeting is the keynote British infrared medical conference in 2002, and delegates from across the UK and Europe have expressed an interest in attending to present their work. In addition to an extensive medical programme, wider issues of interest to all delegates will be discussed, including instrument calibration and the future development of thermography worldwide.

The Scientific committee, Prof. E.F.J. Ring, Dr. P. Campbell, Mr. K. Howell, Prof. B. Jones announce the topics of the provisional programme:

- History of infrared thermography
- Recent advances in infrared imaging instrumentation
- Calibration and standardisation of medical thermography
- Medical infrared image processing and computing
- Thermal physiology
- Surgical applications of thermography
- Thermal imaging of the breast
- Thermography in dermatology
- Thermography in neurology
- Thermography in rheumatology
- Veterinary thermography
- Developing infrared thermography in the UK and Europe

For an abstract form, visit the conference website from 1st February: **www.scleroderma.net/infrared**, or contact the meeting organiser at the e-mail address below. Abstracts should be limited to 250 words and submitted in Word format via e-mail or on diskette.

Instructions for submitting abstracts

The deadline for receipt of abstracts is **31st March 2002**

Please submit all completed forms in electronic format on diskette to the address at the foot of the page or preferably via e-mail to **infrared_thermography@hotmail.com**

Please indicate for which day your abstract is being submitted by inserting an **X** in the box next to the relevant session on the form

Complete the personal details/address section by placing the insertion point within each box and typing

Abstracts should be typed within the box provided and should begin with the abstract title and authors. Please underline the presenting author. Abstracts must fit within the limits of the box, and are restricted to 250 words, exclusive of title and authors. We recommend Times New Roman, font size 11 for the abstract.

The mailing address for abstracts submitted on diskette, and for all registration forms, fees and queries is:

Mr. Kevin Howell,
Department of Rheumatology,
Royal Free Hospital, Pond Street,
London NW3 2QG, UK

Tel +44 (0)20 7472 6550 Fax +44 (0)870 1331058
e-mail **infrared_thermography@hotmail.com**

Registration fee (before 31st March 2002): £ 37 per delegate for one day, £ 70 per delegate for both days.Registration fee (after 31st March 2002) £ 40 per delegate for one day, £ 75 per delegate for both days. Registration is limited to 75 delegates for each meeting.

Complete the registration form (as seen on Page 39) and send it with payment by cheque

(payable to "**Special Trustees for RFH Grant 97**") or bank draft in pounds sterling to the conference organiser Mr.Kevin Howell (address as aforementioned)

Accommodation will be the delegate's own responsibility, but we are able to supply an extensive list of local hotels and guesthouses on request.

Conferences on Thermology in Poland

TThe Polish Society of Thermology announce their 5th Congress of Thermology to be held in **Zakopane September 28 –29, 2002.**

Prof.Dr.Anna Jung is also preparing the organisation of the 9th European Congress of Thermology, which will take place in **Krakow 6-8 June 2003**. New facilities in the famous historical City of Krakow will provide an excellent venue for the Euopean Congress of Thermology. The conference will also support the work of the Polish Thermology Society in its aim to increase the clinical use and application of temperature related techniques for diagnosis and treatment.

Thermo-Budapest 2003

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.

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.

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Generalversammlung der Österreichischen Gesellschaft für Thermologie am 18.12.2001

T.Schartelmüller

Sekretär der Österreichischen Gesellschaft für Thermologie

Die Eröffnung durch den Präsidenten erfolgt um 19:00 Uhr. Da die Zahl der anwesenden Mitglieder nicht zur Beschlussfähigkeit ausreicht, wird der Beginn auf 19:30 Uhr verschoben.

Bericht des Präsidenten

Die Österreichischen Gesellschaft für Thermologie hat derzeit 31 ordentliche und 1 Ehrenmitglied. Leider hat die Fa.NBN Elektronic ihren Status als unterstützendes Mitglied zurückgelegt. In einer ausführlichen Diskussion mit Herrn Ing. Eipeldauer als Vertreter der Fa NBN drückt der Vorstand seine Verwunderung und seine Bedauern aus, dass jene Firma, die praktisch alle Infrarotkamerasysteme in Österreich vertritt, die Aktivitäten der Infrarotdiagnostik in der Medizin nicht weiter unterstützungswürdig hält.

Die Zeitschrift Thermology international bleibt die derzeit einzige regelmäßig erscheinende Fachzeitschrift auf dem Gebiet der medizinischen Thermologie, wobei in den letzten beiden Jahren versucht wurde, durch die Schaffung einer Sektion Technische/Industrielle Thermo- graphie das Interesse von Autoren zu wecken, die sich außerhalb der Medizin und Biologie mit der Infrarotdiagnostik beschäftigen.

Der 5. Internationale Thermographiekongress, der von der Österreichischen Gesellschaft mitveranstaltet wurde, hat zwar Teilnehmer aus 5 Erdteilen angelockt, jedoch war die Zahl insgesamt zu klein, dass die Veranstaltung einen finanziellen Gewinn erzielt hätte.

Bericht des Kassiers

In den Jahren 1999 und 2000 konnten dank der Mitgliedsbeiträge und Spenden leichte Zuwächse des Vereinsvermögens erzielt werden.

Die Überprüfung der Buchhaltungsunterlagen ergab keine Unregelmäßigkeiten, daher konnte nach Antrag des Rechnungsprüfers der Kassier und somit auch der Vorstand entlastet werden.

Mitgliedsbeitrag 2001/2002

Der Antrag des Präsidenten auf Ausweisung des Mitgliedsbeitrages in Euro endet in einer Festsetzung des Beitrages in Höhe von 40 Euro. Diese geringe Erhöhung von auf ATS 500,- auf ATS 550, 41 wird einstimmig angenommen.

Neuwahl des Vorstandes

Präsident: DDr.Kurt Ammer

1.Vizepräsident: Prim Prof.Dr.Otto Rathkolb

2.Vizepräsident: Dr.Thomas Maca

Kassier: Prim.Dr.Johann Mayr

Sekretär: Dr.Thomas Schartelmüller

Rechnungsprüfe: Dr.Peter Melnizky,
Dr.Brigitte Engelbert

Die Wahl des neuen Vorstandes erfolgt ohne Gegenstimmen.

Allfälliges

Dr. Ammer berichtet, dass in Großbritannien das Interesse an der Infrarotthermographie wieder im Wachsen ist. Dies kann am Zulauf zu den von der Universität Glamorgan veranstalteten Ausbildungskursen in medizinischer Thermographie und an der Finanzierung von Forschungsprojekten durch stattliche Stellen abgelesen werden.

Schluss der Generalversammlung: 20:37 Uhr.

Veranstaltungen (MEETINGS)

13.-15.February 2002

3rd Course on Thermal Imaging In Medicine at the University of Glamorgan

Speakers: Dr.K.Ammer, Prof F.Ring, Dr.P.Plassmann,

13.02.2002 Theoretical and Historical Basis of Thermal Imaging in Medicine

14.02.2002 Clinical applications bof Thermal Imaging
Workshop on Thermal Imaging in Vascular Disorders

15.02.200 Practical session; capturing and analysing Images
Future Developments of Thermal Imaging in Medicine

March 1 - 2, 2002

The 8th International Conference on Infrared Thermal Imaging

TAMPA, FLORIDA U.S.A.

Sponsored by ASI Inc.

Conference Times:

Friday, March 1, 2002 - 9:00 a.m. - 5:00 p.m.
Saturday, March 2, 2002 - 9:00 a.m. -1:00 p.m.

Location: Tampa, Florida

Hotels: Hyatt Regency, Marriott Airport Hotel, Radisson, Wyndham Westshore, Sheraton Suites, Hampton Inn Westshore.

Airport: Tampa International Airport

REGISTRATION INFORMATION

Get registration form at
http://www.thermology.com/conference8.htm

Print the registration form and send via Regular Mail with Conference Fee

Fee: $195 per person - if registered by January 1, 2002 - space is limited

Payable to: Ashwin Systems International Inc. P.O. Box 1014, Dunedin, FL 34697 U.S.A. - 727/785-5844

April 1-5, 2002-Orlando

Thermosense XXIV, Opryland Hotel in Orlando, FL, USA

Conference Chairs:
Xavier Maldague,Université Lava
Andres Esteban Rozlosnik lSI Termografia Infrarroja

Featured Speakers
Wally Born: Use of Infrared Thermography for Fire Detection in Alberta
Wolfgang Bauer: Thermal techniques and Modeling in Manufacturing and Processing Industries for High Temperature Furnaces
Carlos Di Bella: Thermal Applications to Agriculture from Remote Sensing Products. A Review
Alexander Dillenz: Burst phase angle thermography with elastic waves
Jean-Claude Krapez: Thermoelastic coupling and use of thermography in fatigue testing
Andreas Mandelis: Review of Progress in Theoretical, Experimental and Computational Investigations in Turbid Tissue Phantoms and Human Teeth using Laser Infrared Photothermal Radiometry (PTR)
Svetlana Morozova: High-precision blackbody sources and facilities developed at VNIIOFI (Russia)
Minh Phong Luong: Thermodetection of irreversible phenomena in materials and structures
Further information
chttp://www.thermosense.org

27.April 2002, Wien

15th Thermological Symposium of the Ludwig Boltzmann Research Institute for Physical Diagnostics and the Austrian Society of Thermology

Theme: Standards in Thermology

Speakers: Prof.Francis Ring

Prof.Tom Eliott

DDr. Kurt Ammer

Prof.B.Wiecek

Venue: Seminarrooms of the Company Schlumberger, Heiligenstäderstraße 39, A-1190 Vienna, Austria

Information:

DDr. Kurt Ammer

Ludwig Boltzmann Research Institute for Physikal Diagnostics, Hanuschkrankenhaus, Heinrich Collinstr 30,
A-1140 Vienna; Austria,

Tel: 43 1 914 97 01 Fax: 43 1914 92 64

Email: Kammer1950@aol.com

May 17-18, 2002

Meeting of the Medical Thermology Group in the UKTA

Venue : Sheila Sherlock Education Centre, Royal Free Hospital, London, UK.

Friday 17th May 2002

Physiological measurement of Raynaud's phenomenon and peripheral microvascular disorders: from research into clinical practice

Saturday 18th May 2002

Medical Infrared Thermography

Information: Mr. Kevin Howell, Department of Rheumatology, Royal Free Hospital, Pond Street, London NW3 2QG, UK

Tel +44 (0)20 7472 6550 Fax +44 (0)870 1331058

e-mail: infrared_thermography@hotmail.com

Web site: www.scleroderma.net/infrared

Registration form for the Meeting of the Medical Thermology Group in the UKTA

I wish to register for the following meeting days, and enclose the appropriate registration fee:

Friday 17th May 2002 only □ £37 before 31/3 □ £40 after 31/3
Saturday 18th May 2002 only □ £37 before 31/3 □ £40 after 31/3
Both days □ £70 before 31/3 □ £75 after 31/3
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Please send me a list of local accommodation □

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

Abstracts covering a maximum of two pages (A4 format) including figures must be mailed to the Seminar Secretariat before January 15th, 2002, indicating clearly the title, The names of authors, afilliation and addres with phone, fax and email, if possible. The organisers sttrongly recommend Internet communication. Authors will be informed about final acceptance of their contribution till March 15th 2002.

QIRT´2002 Deadlines

Jan. 15, 2002 Deadline for abstracts

March 15, 2002 Information about the acceptance of the paper-final instructions for authors

April 15, 2002 mailing of final announcement, detailed proghramme and registration form

Sept 1, 2002 camera-ready paper

Information:

Please reply by email on the site

http://www.fsb.ht/Qirt2002

Or return a letter to ther conference secretary

QIRT´2002

Igor Sindov, Faculty of Mechanical Engineering and Naval Architecture

O-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: Zakopane

Information: Prof Dr.Anna Jung
Department of Peditarics and Nephrology
Central Clinical Hospital MMU,
Szerow str 128, 00-909 Warsaw, Poland

Phone/Fax +48 22 681 67 63
email: ajung@cskwam.mil.pl

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Payment should be sent (without any charges for the European Association of Thermology) to the following bank account:Bank Austria, Vienna, Austria, bank number:20151, account number: 965023054

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Österreichische Gesellschaft für Thermologie

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Österreichische Gesellschaft für Thermologie

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Diese Zeitschrift ist eine gemeinsame Publikation der Ludwig Boltzmann Forschungsstelle für Physikalische Diagnostik und der Österreichischen Gesellschaft für Thermologie.

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