Acta Derm Venereol (Stockh) 1989; Suppl 144: 149–152

Responses of Skin Temperature to Different Thermic Stimuli in Atopic Eczema

O. P. HORNSTEIN and G. HEYER

Department of Dermatology, University of Erlangen-Nürnberg, Erlangen, FRG

Patients suffering from atopic eczema (AE) often exhibit general disturbances of vasovegetative skin functions. Thus, in 21 patients with AE we studied the response of the skin of one forearm to standardized 15-min exposure of the other forearm to a moderate cold bath (17–18°C) and then, after a resting time, to a hot bath (40–41°C). The results were compared with those in 23 age- and sex-matched healthy controls under the same experimental conditions. In most patients, the unilateral skin exposure to warmth left the temperature of the contralateral forearm nearly unchanged or even slightly decreased, whereas the exposure to cold induced either a slight rise in contralateral skin temperature or only a minute decrease. In contrast to the normal consensual temperature reaction of the non-exposed forearm to warmth exposure of the contralateral arm in most controls, the results in atopic patients indicate a "rigid" or even inverse ("paradoxical") response to the thermic stimuli applied. This abnormal pattern of thermoregulation may reflect an intrinsic disturbance of the peripheral and hypothalamic autonomous neural system involved in the pathogenetic conditions of AE. *Key words: Thermoregulation; Abnormal reactivity, Hypothalamic dysfunction.*

O. P. Hornstein, Department of Dermatology, University of Erlangen-Nürnberg, Hartmannstrasse 14, D-8520 Erlangen, FRG.

Many atopics are known for an abnormal cutaneous vasoactivity to temperature with vasoconstriction on warming and vasodilatation on cooling (2). Korting (5) noticed in the 1960s some signs of a disturbed cutaneous thermoregulation, and later Abrams and Farber (1) demonstrated, by means of plethysmographic investigations of the finger microcirculation, a weak vasomotoric response in the skin of atopic patients to variations in the room temperature. Kocsard et al. (4) studied the thermoregulatory behaviour of atopic subjects by immersing one hand in hot water of 41°C for 10 min and measuring the skin temperature on the contralateral side simultaneously. They found during the heat exposure not the normal consensual rise of the temperature but an inverse reaction on the other arm.

This "paradoxical" thermoregulatory inversion seems to indicate not only an alteration of the peripheral microcirculation but also a reflectory imbalance on the level of hypothalamic vasomotoric centres where afferent neuronal impulses are transmitted into efferent ones leading to a rapid consensual thermoreaction of the corresponding body site.

In recent neuro-endocrinological studies we found signs of an altered rhythm of the nocturnal secretion of melatonin and cortisol in a number of patients with atopic eczema (3, 6). We therefore felt that a dysregulation of subcortical or hypothalamic, resp., centres of the brain might be involved in the pathogenesis of atopic eczema (AE).

PATIENTS AND METHODS

We studied the thermoreactive behaviour of 21 patients with AE (9 males, 12 females, range of age 16–47 years, mean 23 years) on the 5th or 6th day of their hospitalization and 23 healthy control subjects (15 males, 8 females, ranging in age from 18–52 years, mean 28 years).

Each subject was seated in a climatized room with constant room temperature at 22–23°C for half an hour. None of the individuals were under corticoid treatment during or 3 months prior to the study, and none was allowed to smoke before the examination. The time of examination was between 1.00 p.m. and 3.00 p.m. All the tests were performed between January and May.

First, the right forearm was submerged in a bucket filled with cold water, tp. 17–18°C, for 15 min, and at the same time the skin temperature in the left elbow flexure was measured (Fig. 1) using a fine-calibrated contact thermometer (7), which records caloric differences of less than 0.1°C. Then, after an ample resting period of 90 min, the procedure was repeated using hot water, tp. 40°C, for the right forearm and measuring the skin temperature in the left arm flexure simultaneously.

RESULTS

Cold water exposure. 11 out of the 22 patients showed a fall of their consensual temperature between 0.3 and 1.5°C. In 3

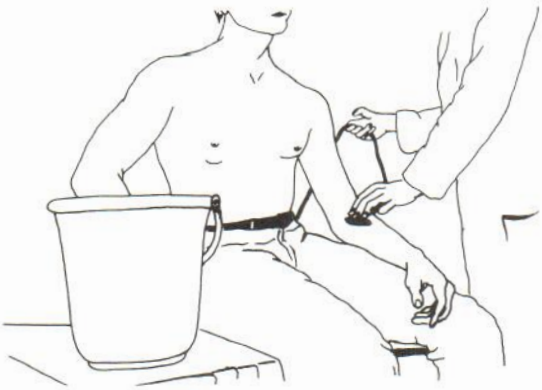

Fig. 1. Setting of consensual thermoregulatory experiments. Submersion of the right forearm in (cold or hot) water for 15 min, simultaneous contact thermometry at the opposite arm flexure.

patients the temperature increased, after an initial decrease, over that measured at the starting time (amplitudes from −1.4 to +0.9°C). In 5 patients after a slight initial rise, after 8 minutes a minor decrease of the temperature was recorded. All the changes in temperature, except in 2 patients, oscillated between −1°C and +1°C Fig. 2).

In contrast, 10 of the 23 control persons showed a more of less continuous rise of the temperature, and in the remaining ones a rather steady fall of the skin temperature was measured, in a few cases with a terminal reverse of the lowered values (Fig. 3).

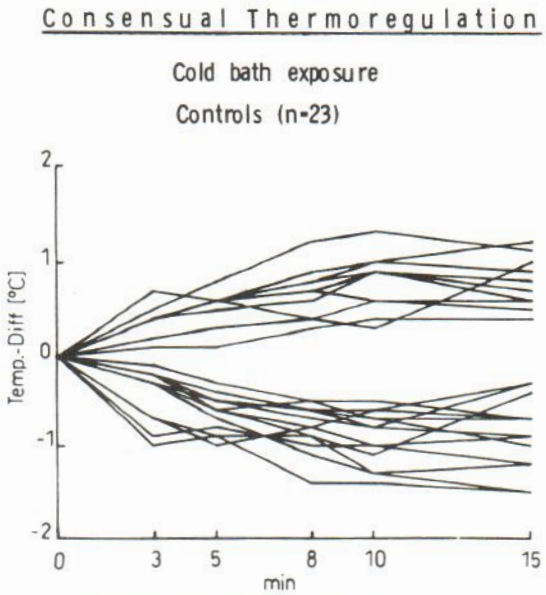

Fig. 3. Divergent skin temperature response to cold exposure in the control group.

Hence we found, in comparison to the controls, in the patient group the temperature profile to vary only slightly upon cold exposure, showing a "undecided" pattern which was apparent by the zigzag line of the course of the temperature curve.

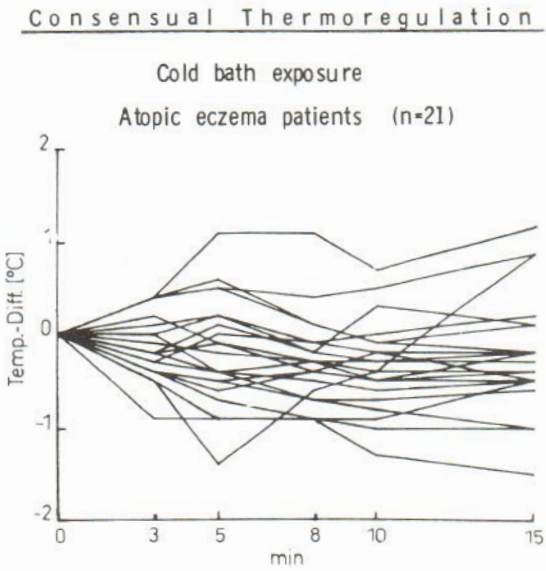

Fig. 2. Hyporeactive or inverse skin temperature response to cold exposure in the majority of AE patients. The ordinate indicates the initial values of all profiles as zero and the temperature differences related to this starting point.

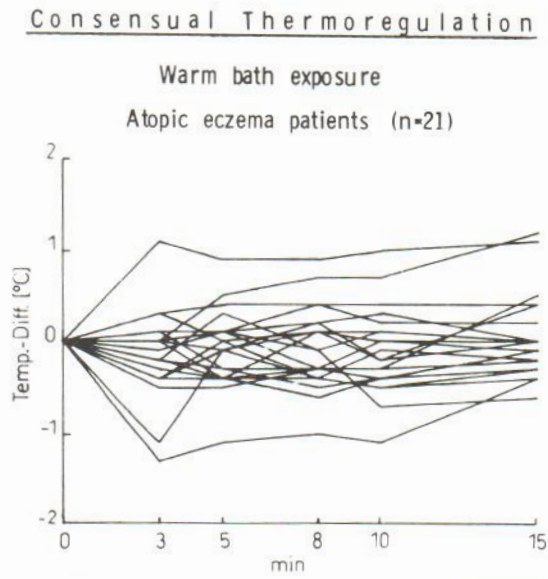

Fig. 4. Hyporeactive ("lazy") skin temperature response to heat exposure in most AE patients except a few showing either consensual or inverse temperature profiles.

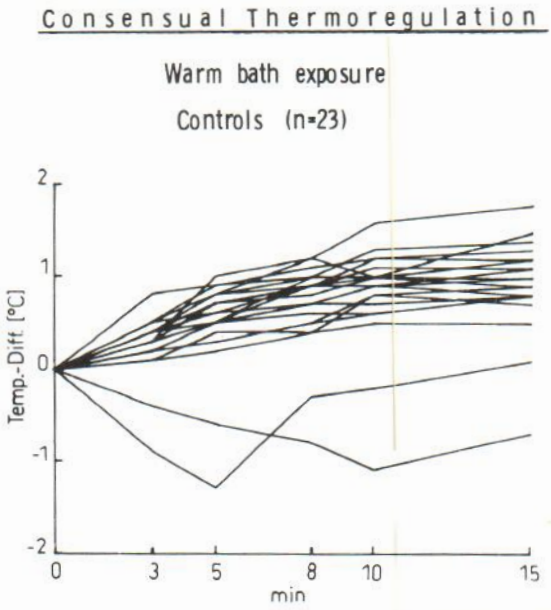

Fig. 5. Consensual skin temperature response to heat exposure in most controls except two subjects showing initial fall of temperature.

Hot water exposure. 4 out of the 22 patients exhibited an elevation of the temperature up to 1.2°C, yet in 8 the temperature fell till −1.3°C. Another 6 patients varied only slightly in their consensual skin temperature around the zero line (i.e., the continuation of the initial value). The remaining 3 patients showed more pronounced amplitudes of their temperatures, yet the values oscillated also in the vicinity of the zero line (Fig. 4).

Out of the 23 control subjects, 21 showed a continuous rise of their consensual temperature leading to values above 0.5°C after 10 min forearm exposure to hot water. In 2 subjects there was a remarkable fall of their temperature by −1.3°C after 5 min or −1.1°C after 10 min, resp., followed by a slight increase of the values (Fig. 5).

DISCUSSION

This study shows, as a common feature in the group of the patients, an altered regulation of the consensual skin temperature to cold and heat exposure. The thermographic pattern behaved inversely to cooling or heating of the contralateral forearm, or showed a rather "lazy" reaction to the thermic stimuli applied. Most patients reacted promptly to the initial caloric stimulus, but after 3–5 min exposure their skin temperature changed into a rather rigid or even "contrasensual" pattern.

How can these abnormal patterns of cutaneous thermoreactivity in patients with atopic eczema be explained?

The hypothalamic center for thermoregulation is maintaining the homeostatic "core" temperature of the body by inducing quick adaptations of the involved cutaneous mechanisms such as local blood flow, sweating, or muscle shivering, to exo- or endogenous caloric stimuli. Injuries to distinct nervous control centres, for example brain stem contusion, are known to alter also their thermoregulatory function. Minor deviations from the normal consensual response of the skin temperature to regional heat or cold exposure may also reflect an imbalance of the central thermoregulation, whatever the primary reason may be.

Thermoregulatory anomalies in patients with AE were first reported by Korting (5) and then by Kocsard et al. (4). In this study we have more extensely examined the feature by exposing the skin to different temperature stimuli. Interestingly, a few control subjects also showed an abnormal consensual response to temperature. They neither presented signs nor a history of atopy. An abnormal thermoreactivity is not specific for patients with AE, but it may indicate an intrinsic disturbance of both the peripheral and central autonomous nervous system involved in the pathogenetic conditions of AE.

In conclusion, in addition to local disturbances of the cutaneous microcirculation in patients with atopic eczema, there are also anomalies of the central thermoregulation in these patients. The abnormal thermoregulation to external cold and heat exposure is reflected by an either "lazy" or inverse ("paradoxical") consensual response to contralateral thermic stimuli. The anomalies of the thermoregulation in atopics with eczema may indicate a disturbance of the central vasomotoric control system possibly contributing to the pathogenetic conditions of atopic eczema.

REFERENCES

1. Abrams G, Farber EM. Peripheral vascular responses in atopic dermatitis. Arch Dermatol Res 1963; 88: 554.
2. Borelli S, Kopecka B. Über das reaktive Verhalten der Hautdurchblutung auf Kälte- und Wärmereize bei Kranken mit atopischer konstitutioneller Neurodermitis. Dermatol Wochenschr 1966; 152: 1365.
3. Heubeck B, Schönberger A, Hornstein OP. Sind Verschiebungen des zirkadianen Cortisolrhythmus ein endokrines Symptom des atopischen Ekzems? Hautarzt 1988; 39: 12.
4. Kocsard E, Ofner F, Broe GA. Paradox temperature response in atopic dermatitis. Dermatologica 1973; 146: 8.

5. Korting GW. Einige Wesenszüge des endogenen Ekzematikers. Dtsch Med Wochenschr 1960; 85: 417.
6. Schwarz W, Birau N, Hornstein OP, Heubeck B, Schönberger A, Meyer C, Gottschalk J. Alterations of Melatonin Secretion in Atopic Eczema. Acta Derm Venereol (Stockh) 1988; 68: 224.
7. Stüttgen G, Flesch U. Dermatologische Thermographie. Berlin, Edition Medizin, 1984.