
Rheumatology

The Interdisciplinary Concept

Vol. 16

Series Editors

M. Schattenkirchner, Munich

F.-W. Hagen, Munich

KARGER

Basel · München · Paris · London · New York · New Delhi · Bangkok · Singapore · Tokyo · Sydney

Rheumatic Diseases and Sport

Editor
H.-W. Baenkler, Erlangen

27 figures and 46 tables, 1992

KARGER

Basel · München · Paris · London · New York · New Delhi · Bangkok · Singapore · Tokyo · Sydney

Rheumatology

The Interdisciplinary Concept

Library of Congress Card No. 67-16407

Drug Dosage

The authors and the publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accord with current recommendations and practice at the time of publication. However, in view of ongoing research, changes in government regulations, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions. This is particularly important when the recommended agent is a new and/or infrequently employed drug.

All rights reserved.

No part of this publication may be translated into other languages, reproduced or utilized in any form or by any means, electronic or mechanical, including photocopying, recording, microcopying, or by any information storage and retrieval system, without permission in writing from the publisher.

© Copyright 1992 by S. Karger AG, P.O. Box, CH-4009 Basel (Switzerland)
Printed in Switzerland on acid-free paper by Thür AG Offsetdruck, Pratteln
ISBN 3-8055-5425-7

Bayerische
Staatsbibliothek
München

Contents

Foreword	VI
Preface	VII
Budsayavith, Y. (Erlangen): Rheumatology and Sports Medicine. Relations and Points of Contact	1
Baenkler, H.-W. (Erlangen): Exercise and the Immune System: The Impact on Diseases	5
Schuh, A.; Senn, E. (Munich): Climate and Rheumatic Diseases	22
Zimmermann, M. (Heidelberg): Physiology of Pain and Pain Therapy in the Musculo-Skeletal System	40
Seidl, O. (Munich): Psychosomatic Considerations in Physical Activity of Rheumatic Patients	59
Maisch, B. (Marburg): The Heart in Rheumatic Disease	81
Truckenbrodt, H.; Häfner, R.; von Altenbockum, C. (Garmisch-Partenkirchen): Sports in Juvenile Chronic Arthritis	118
Puhl, W. (Ulm); Maier, P. (Bad Waldsee); Günther, K.P. (Ulm): Effects of Physical Activity on Degenerative Joint Disease	129
Hirschfelder, H. (Erlangen): The Influence of Biomechanics on the Joints of Persons Participating in Sports Activities	142
Joisten, U.; Albrecht, H.J. (Oberammergau): Physical Activity and Spondylarthritis	153
Senn, E. (Munich): The Meaning of Sportive Elements for Physiotherapeutic Treatment of Rheumatic Diseases	160
von Wilmowsky, H. (Püttlingen): Treatment of the Physically Active Rheumatic Patient: A Pharmacotherapeutic Approach	170
Burmester, G.R.; Krause, A. (Erlangen): Immune Interventions and Treatment of Infections in Physically Active Arthritic Patients	187
Hagena, F.-W.; Zimmer, M. (Munich): The Rheumatic Patient with Joint Replacement and Sports	208
Krüger, K. (Munich): Rheumatoid Arthritis and Sports	219
Subject Index	228

Climate and Rheumatic Diseases

A. Schuh, E. Senn

Institute of Medical Balneology and Climatology,
Ludwig-Maximilians-Universität, Munich, FRG

Weather Sensitiveness of Rheumatics

In a group of average healthy persons, between 30 and 50% of the people claim to be sensitive to changes of the weather [Faust, 1973; Schaich, 1974]. However, the average course of health is about the same for people who claim to be sensitive and those who do not [Richner, 1976]; this supports the old statement [De Rudder, 1951] that all people react to the weather, but only weather-sensitive people relate it with the weather.

A person's statement to be 'sensitive to the weather' offers clear information about the structure of his/her personality. Already Curry [1951] and Lampert [1962] described weather caused types of human reaction. Recently the type of the weather sensitive was defined by the help of the 'Freiburger Persönlichkeitsinventar' [Geiger and Gensler, 1975; Faust, 1978]. Women call themselves more often 'weather-sensitive' than men, young people only to a small percentage; with increasing age, between 30 and 60, the rate rises up to 60% of the total population. These people name, referring to psychic symptoms, the following, by Faust [1976] in decreasing prevalence ordered, complaints: tiredness, ill-humor, unwillingness to work, lack of concentration, problems to fall asleep, nervousity, tendency to make mistakes, indisposition and fear.

During a study with 2,000 participants [Dirnagl, 1985], weather-sensitive and nonsensitive people were questioned about reasons and fre-

quency of their visits to doctors. Symptoms or suspected diagnosis, being the cause of the visit to a doctor, showed that weather-sensitive people named 'rheumatic complaints', besides the symptoms of the so-called 'vegetative dystonia', most often.

Among rheumatics, weather sensitivity is far more spread than among the rest of the population; that is stated in many publications [e.g. Tromp, 1980]. Thompson questioned already in 1951 112 patients suffering from chronic polyarthritis and found out that 83% looked upon their symptoms as weather-related. Levis-Faning [1950] came to the same result: 61 of 369 patients blamed certain states of the weather for their pains. The numerous statistical surveys can be summarized to 75–90% of all rheumatics being weather-sensitive; weather and climate are supposed to play a central role in releasing or intensifying these states of pain.

Possible Meteorological Parameters and Climatic Conditions

People with average weather sensitivity, as well as people with rheumatic diseases come up with complaints about a falling off in health preferably in time and local connection with atmospheric disturbances, i.e. changes in weather or strong variation of particular, meteorological parameters from the seasonal weather course (cf. 'Weather, Climate and Rheumatism'). Also certain climatic conditions shall have effect on the frequency of complaints. The terms 'weather' and 'climate' differ in the following way: 'weather' is understood as a short-term state. As 'climate' is defined the mean state of atmosphere over a specific place, as well as the for this place characteristic average course of weather. Weather and climate are always composed of a certain combination of the meteorological parameters.

Change of Weather, Low-Pressure Area, High-Pressure Area

Variations from the average course of weather are quite common in the middle latitudes; the zone of western winds is quite often characterized by unstable weather. The drifts here are a consequence of moving low-pressure areas (cyclone) and high-pressure areas (anticyclone) combined with rushes of subtropical air far into the northern region, as well as cold air from the polar regions into the south. A low-pressure area is formed when warm masses of air meet air coming from the polar regions: Normally the temperature decreases from equator to pole parallel to the lati-

tude; the isotherms lie in parallel order. This balance is disturbed by the rush of warm and cold masses of air: in the atmosphere a flat temperature wave is generated. Therefore, when different air masses meet each other a deformation of the former even drift, which is called 'front area' (warm front) is formed. To do this the warm air moves over the cold air (slip up); the warm air is at the same time cooled down and condensates. The warm front is characterized by an area of rain in front, with stratus clouds and drizzle resp. rain. At the other side of the low-pressure area cold air slips at the same time under the warm air and lifts it rapidly; the cold front is created. Due to the fast lift of air it comes to fast cooling and strong condensation: Along the cold front cumulus clouds are formed with showers and hail. According to the earth's rotation, a rotation of both fronts around the center of the wave disturbance is formed; the air pressure decreases towards the center. Thus, the general drift of the cyclones is formed. Within 24 h the cold front catches up with the warm front and the fronts close (occlusion). At this point of time the 'aging process' of the low-pressure area takes its course. The aged low-pressure area slows down its moving speed. The whirl grows weary, following air masses fill up the funnel, the air pressure increases and the cloud cover, and therefore the low-pressure area, breaks up.

The high-pressure areas (anticyclones) are described as 'hill of cold, heavy air' [Möller, 1973], on front- and backside the air moves down (slip down) and warms up. A high pressure area can reach the size of a continent (e.g. a Russian winter high) and remain stationary for weeks; normally small areas with high pressure follow the low-pressure areas of the general drift (intermediary high). The consequence is constantly changing weather.

Variation of Particular Meteorological Parameters

At each change of weather a large number of meteorological parameters change at the same time. To make a more uniform approach possible between this multifactorial process and its contingent effects on man, climatology summarizes the meteorological parameters into so-called 'effect complexes' (fig. 1).

The thermic effect complex is of special importance: It takes into consideration air temperature, air humidity, wind speed and infrared radiation. The generic term 'air humidity' has to be differentiated into relative air humidity and absolute air humidity, the so-called 'steam pressure': the relative air humidity (in percent) quotes the degree of the air's satiation

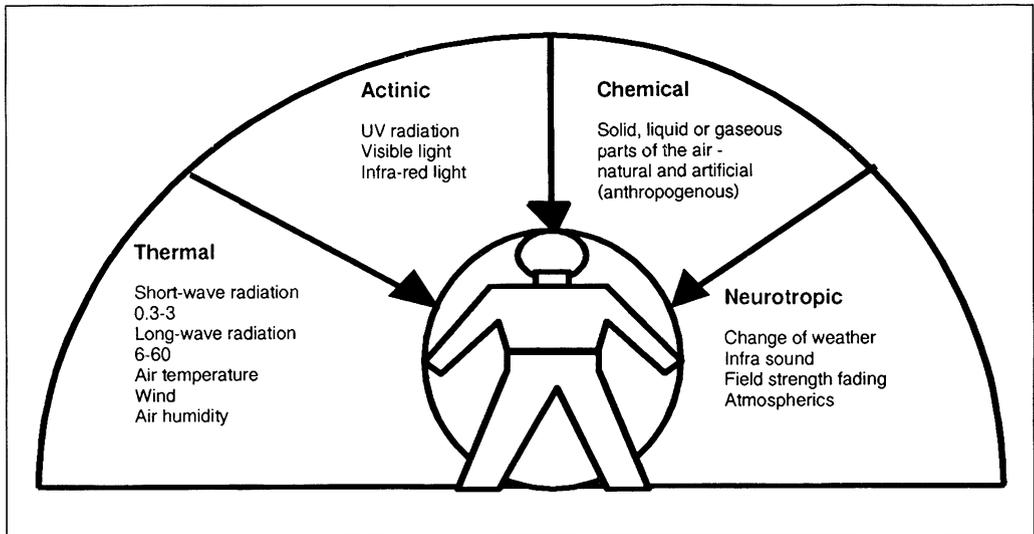


Fig. 1. Atmospheric effect complexes.

with water steam. It shows, under the actual temperature, the still possible absorption of water steam by the air, whilst the steam pressure (in hPa) equals the water steam actually present in the air. The particular factors of the thermic effect complex – besides the weather change as a whole – take up a major place in medical-climatological causal research.

Weather, Climate and Rheumatism

Experimental Bases

Several authors have dealt with the effects of weather on rheumatics (table 1). As indicator for the influence in most cases the objective feeling of pain was referred to; acute states of pain under certain meteorological conditions, as well as increase of pain within the last 24 h before the occurrence of this meteorological situation, was examined.

In few studies besides subjective statements also 'hard' connections were considered; Hollander and Yeostros [1963] constructed a climate chamber which permitted to simulate certain weather situations for objective research. The symptoms were evaluated following the 'clinical index';

Table 1. Experimental studies on connections between weather and rheumatism

Author	Rheumatic process	Connection between meteorological factors and weather situation	Methodics	Remarks
Weitbracht and Simon, 1989	lumbal prolapse of disc, increase of pain	1) temperature decrease 2) humidity increase	114 patients, recording of clinical admission	accumulation of admission during the winter month, flow of cold air responsible for increase of pain, cause probably direct lumbal hypothermia
Harlfinger et al., 1986	arthritis, arthrosis, increase of pain (last 14 h)	1) passage of front 2) following advection of cold air (high pressure area with cold air) 3) weather situation with dynamic cold air advection	state of health during Smile test (24 h before), patients with rheumatic pain, > 1,600 interviews	assumed connection between weather and rheumatism; strongest sensations of pain during passage of front, they remain the day after, at cold air advection; patients can feel the upcoming weather (Smile test)
Wiebe et al., 1985	arthritis, increase of pain	increase of humidity	100 patients in the north of Holland	connection in summer stronger than in winter
Patberg et al., 1985	arthritis, intensification of pain	not only process of change, but also actual situation 1) decrease of temperature 2) cold 3) decrease of steam pressure 4) increase of humidity	100 patients with arthritis, pain, questioned over 1 year, lived in sea climate (Holland)	symptoms of arthritis are influenced by weather, but not the disease itself, pain in summer stronger than in winter (in winter, people stay more indoors; the steam pressure is inside higher than outside and the relative humidity is lower)
Latmann and Levi, 1980	arthritis, blood parameters for occurrence of inflammation – sedimentation – C-reactive protein	no connections	patients with arthritis, not pain, but objective blood parameters at the day when inflammation starts	weather influences symptoms of arthritis but not the inflammatory process, weather influences the personal well-being but not the disease itself

Sönning et al., 1979	arthritis, acute rheumatic attack, increase of pain	1) less warm air the day before, increase of cold air advection the following day. at the begin of cold air flow in, i.e. within the front 2) cold sea air 3) thunderstorms (strong vertical exchange movements), connection with change of ground near humidity milieu	123 patients, arthritis: first occurrence of inflammatory symptom after longer time	
Hollander and Yeostros, 1963	arthritis, symptoms according to clinical index	falling air pressure with increasing humidity	research in climate chamber	hypothesis: normal tissue compen- sates falling pressure (intracellular fluid evacuates into the blood circula- tion, sick tissue retains liquid) → increased intracellular pressure → pain and swelling
Tromp, 1963	pain release resp. intensification	cold		hypothesis: reason for pain increase is increase of viscosity of synovial liq- uid
Barcal et al., 1961	arthritis, arthrosis, intensification of pain	1) front side of low pressure areas 2) change of weather 3) not: existing bad weather	> 100 patients, subjective complaints, objective state	announcing pains 15 h before change of weather
Pehl and Weskott, 1955	rheumatics: intensification of pain	1) change of weather 2) decrease of temperature 3) increase of humidity 4) wetness and cold 5) increase and decrease of air pressure	100 patients	significant weather sensitiveness
De Rudder, 1952	rheumatic disposition	1) humid cold 2) lack of sun		experience that patients come back from journeys to southern countries without rheumatic complaints

this index contains according to Hill [1966] the calculation of arthritis activity based on joint stiffness, the amount of ASA necessary to soothe the pain, firmness of the hand's clasp, walking time for a standardized distance and other not relevant measurements of another collection of parameters, the so-called 'articular index'.

Exceeding that, in the past years Latman and Levi [1980] measured the blood parameters BSG and CRP (blood sedimentation rate and C-reactive protein), to correlate the course of inflammation with the actual weather situation. Weitbrecht and Simon [1989] eventually correlated the clinical admission of people with lumbar prolapse of disk and the weather situation. Several authors also published bibliographies (table 2). Studies, based on subjective statements of pain, as well as studies based on measurable states of inflammation of arthritis were described.

Except for one study [Latman and Levi, 1980], each of the in table 1 listed experimentally based studies points out a correlation between a defi-

Table 2. Literature review on connection between weather and rheumatism

Author	Rheumatic process	Connection between meteorological factors and weather situation	Methodics	Remarks
Latman, 1987	arthritis: first signs of inflammation	different parameters	literature references	data material is not evident enough to draw serious conclusions, methodics are often questionable
Dirnagl, 1978	rheumatics: pain frequency and intensity	1) rapid change of weather combined with 2) decrease of temperature	literature references	weather sensitiveness of rheumatics cannot be traced back to standardized weather situation, individually different ways of reaction are assumed
Pilger, 1970	arthritis: intensification of pain	contradictory statements 1) humid cold 2) lack of sun 3) change of weather 4) falling pressure with increasing humidity at the same time	literature references	many studies uncontrollable

nite meteorological situation or a certain course of weather: The frequency of pain is increased when the course of weather changes: The *sudden change of weather* proceeds from high-pressure area to low-pressure area; in the beginning of cold air influx, i.e. within the cold front range, the most complaints about pain are found [Sønning et al., 1979]. During the passage of the front more cold air is brought up into the low-pressure area; in the middle latitudes it is normally humid cold sea air. The major reason is seen either in the cold air advection [Harlfinger et al., 1986; Sønning et al., 1979], or in increase of humidity [Pehl and Weskott, 1955; Wiehe et al., 1985]. This temperature decrease at the front side of a low-pressure area [Barcal et al., 1961], with increase of humidity at the same time, is mainly held responsible for the increasing frequency of pain. During the sudden change of weather with the increase in humidity, the air pressure drops. The connection of these two parameters shall also cause a deterioration of the symptoms – measured at clinical index [Hollander and Yeostros, 1963]: At changes in the climate chamber the clinical index only increased on the combination of falling barometric pressure and increasing air humidity. Sønning et al. [1979] found in addition to that an increased pain frequency at days with thunderstorms; they too see the connection in the change of temperature-humidity milieu.

Some studies do not take the complex process of changing weather – meaning the simultaneous change of several parameters – into consideration, but hold responsible *single meteorological parameters*. Tromp [1963] takes cold in general to be a pain-releasing or intensifying parameter. In the same way already De Rudder [1952], as well as Pilger [1970], believed humid cold and lack of sun to be the main release.

The above listed studies substantiate unanimously the increase of pain frequency at changes of weather, i.e. approaching low-pressure areas in connection with decrease in temperature and increase of relative humidity. Contradictory statements can however be found: Fuss [1981] gives a tendency to pain increase at rising temperature, based on her study; Pilger [1970] points out, following his literature investigation, that many authors particularly claim they did not find any influence of air humidity at all. On the other hand, other authors [Dirnagl, 1985] assume the content of absolute air humidity (steam pressure) to be the really relevant figure; Flach [1938] described singular examples that attacks of pain occurred particularly when the steam pressure in comparison to its normal level decreases. To refer to the steam pressure as indicator for pain stimulating processes has its justification: as rheumatic pain occurs outside buildings as well as

inside, one has to look for meteorological parameters which change inside parallel to outside. Entering a room, e.g. the air temperature, air movement and relative humidity change, not the absolute humidity. Unfortunately the influence of steam pressure on rheumatic processes was in none of the controlled studies pursued.

Interpreting the literature, utmost caution is indicated, not only in terms of meteorological process, but also looking at the development of disease: it cannot always be clarified upon which specific form of disease the research was done in the described study. The majority of studies deals with 'rheumatoid arthritis', some authors however only talk about 'rheumatism'; a differentiation between inflammatory and degenerative clinical picture is not made.

The question, whether not only the weather, but also the average state of atmosphere, i.e. the *climate*, can be correlated with the occurrence and deterioration of rheumatic diseases, has yet not been finally clarified: Lawrence [1963] describes – based on epidemiological surveys – that arthritis can be found most often between 50° and 60° northern latitude, and that it decreases outside this zone in higher or lower latitudes. That corresponds with the figures in table 3, according to which the numbers for England and Scandinavia are the highest (4–7%), decreasing to the north (Alaska 1%) and near the equator, Puerto Rico (0.92%). In the Federal Republic of Germany about 1 million people (1.6%) suffer from chronic polyarthritis; 0.7% of the total population, i.e. 419,016 people in 1985, claimed medical or professional rehabilitation measures because of rheumatic diseases [Mikrozensus, 1987]. The results of surveys by Mendez-Bryan et al. [1963] in Puerto Rico showed a significantly smaller incidence of arthritis compared with studies in temperate climate zones; there the lowest rate anyway was found. In two later studies, Lawrence [1966] and Lawrence et al. [1966] could not find differences between the population of Middle America (18° n. lat.) and Europe (54° n. lat.) and between the inhabitants of Jamaica and Southern England (table 4).

Studies dealing explicitly with climatic influences in degenerative changes more or less do not exist. One exception is described by Lawrence [1977] in his review, though without reference: According to that, among the population in Jamaica's warm climate (18° n. lat.) only 21% degenerative changes could be found, whilst the percentage of North England's inhabitants (52° n. lat.) is around 50%.

Already in 1966, Hill pointed out that the evidence of the so far existing studies is reduced by the fact that there were no standardized diagnosis

criteria and no standardized selection of the population examined; these studies therefore did not allow definite conclusions. The deficit in controlled, worldwide coordinated studies of connections between rheumatic diseases and climatic conditions could not be solved until the present day.

Lawrence [1969] and Fleming et al. [1976] report in their studies that the pain frequency with chronic polyarthritis is also influenced by the season, pointing out the summer and warmth as positive, coolness and winter as negative; Patberg et al. [1985] note in their study with 100 arthritis patients a stronger pain increase in summer than in winter (table 1). The authors explain this assuming that meteorological factors affect the body mainly in summer and less in winter, as in winter one is less outside and more often inside buildings, where the climate, compared to that outside, differs in almost all meteorological parameters: mainly there is less relative humidity inside.

Table 3. Arthritis frequency in different climates and different parallels of latitude

Population, latitude	Arthritis incidence rate	Author
50–60° n.L.	'most frequently'	Lawrence, 1963
Federal Republic of Germany	1.6%	Microcensus, 1987
Puerto Rico	0.92%	Mendez-Bryan et al., 1963
England, Scandinavia	4–7%	Mikkelsen, 1966
Africa, near equator	'minor incidence'	McKinley, 1967
North America	3.2%	Engel and Burch, 1967
Alaska	1.0%	Blumberg et al., 1961

Table 4. Comparisons of arthritis frequency between different populations

Population, latitude	Arthritis incidence rate	Author
America and Europe between 18 and 54° n.L.	no differences	Lawrence et al., 1966
Jamaica and Southern England	no differences	Lawrence et al., 1966
Red Indians in Montana (48° n.L.) and Arizona (33° n.L.)	pain incidence higher in the north (Montana)	Burch, 1966, quot. acc. to Pilger, 1970

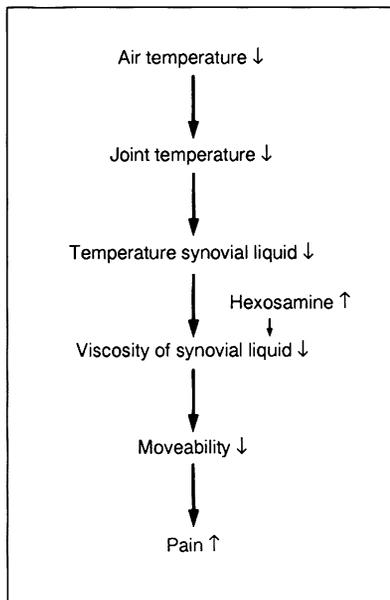


Fig. 2. Hypothesis concerning pain increase at chronic polyarthritis caused by decrease in air temperature, resp. coldness.

Despite these different statements, patients with rheumatic diseases are sent in regions with dry-warm climate only based on 'practical knowledge'. Hill [1966] supposed that the little changes in weather in these regions have a positive influence on the 'weather sensitivity' of rheumatics; convincing physiological reasons for the improvement of the complaints do not yet exist.

Hypotheses about Influencing the Rheumatic Process

Considering the connection weather-rheumatic process, analyzing the effectors, i.e. the meteorological parameters and their interrelationship, effect priorities result for the temperature-humidity milieu. Receptors for changes in temperature-humidity milieu are at first the body's periphery and the respiratory system: skin and upper respiratory tract react directly on changes in the environment's temperature and humidity.

Most convincing are those hypotheses about influence of weather and climate on rheumatic diseases, which establish the reference to the patient's sensitiveness to cold (fig. 2): Latman [1987] and Rothschild and Masi [1982] proceed on the assumption that a short stay in strong cold or a

longer stay in a cool environment causes a temperature decrease in the joint. As the joints are not covered by protective muscle or fatty tissue, the temperature of synovial liquid falls faster than rectal or muscle temperature. The synovial liquid becomes the more viscous the lower the temperature is [Hertel and Ingenpass, 1974]. This temperature decrease of synovial liquid causes a greater stiffness of the joints [Hunter et al., 1952]. Tromp [1963] also figures the increase in viscosity of synovial liquid to be a reason for the pain increase. It is known [Tromp, 1963] that cold environment condition lowers the hexosamine output of healthy people. In empiric studies, Tromp and Bonma [1966] found that the hexosamine content in rheumatic's urine was significantly lower than in healthy person's. The authors concluded that rheumatic pains are the consequence of low environment temperatures which lead to a concentration of hexosamine in the synovial liquid and therefore to an increase in viscosity. By that the joint is additionally limited in its movability: pain is the consequence.

Hollander and Yeostros [1963] point out in their hypothesis that pain is increased when the air pressure falls, and at the same time the relative humidity grows, as follows: Normal tissue is able to compensate falling air pressure by evacuating intracellular liquid into the blood circulation. Afflicted tissue, however, is not as permeable and retains the liquid. Therefore, increased pressure exists in afflicted tissue, compared with healthy tissue. This pressure gradient leads to increased pain and to the swelling of the afflicted tissue. All authors though leave open which meaning the increasing air humidity has in combination with falling temperature or falling air pressure.

Conclusion

Certain weather conditions lead with high probability to increase of frequency and intensity of pain, the weather influencing the symptoms of rheumatic diseases (pain, well-being), though not the disease itself: no connections were found with objective parameters (e.g. inflammatory factors). The weather sensitiveness of rheumatics cannot be drawn back to a homogeneous meteorological process; the reactions are individually different. Most often, increase in pain frequency and intensity correlates with a change of weather in the form of an approaching low-pressure area combined with temperature decrease and increasing relative humidity (fig. 3). Additionally, the releasing, resp. intensifying effect of coldness, particularly humid cold and lack of sun, on pain can be proven.

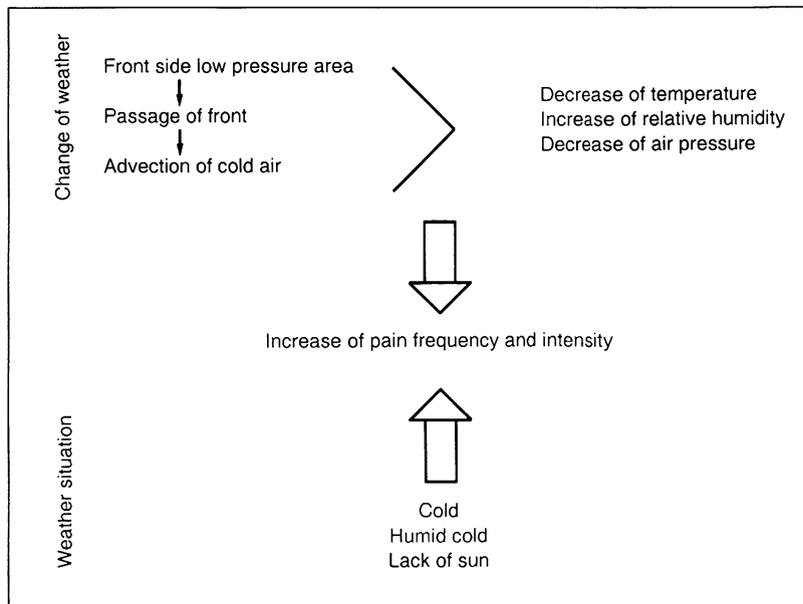


Fig. 3. Increase of pain frequency and intensity caused by change of weather, resp. certain weather states.

Climate Therapy with Rheumatic Diseases

The climate therapy of rheumatic diseases has to express as major goal the prophylaxis against weather sensitiveness. By goal-oriented endurance training, with cold adaptation at the same time – within the scope of a terrain cure under cool conditions – weather sensitiveness can be significantly reduced [Schuh, 1989]. Base has to be a cautious acclimatization to temperature influences and variations, which is achieved by exposition to outdoor conditions, and slight hardening against cold stimuli.

The reduction of weather sensitiveness by acclimatization to climatic stimuli is also an aspect of the thalassotherapy. In his literature review, Jordan [1978] describes the contemporary state of knowledge concerning thalassotherapy of chronic polyarthritis and concludes that climate therapy on cool ocean coasts can be considered as prophylactic and therapeutic ‘hardening measure’ in the sense of a ‘cold desensitization’, although there are not enough secured facts.

In climate therapy in middle and high mountain regions, besides the treatment of skin diseases, the treatment of rheumatic diseases is in the foreground: to treat diseases of the inflammatory-rheumatic form even in 1934 a clinic for rheumatics in Davos existed [Neergard, 1934]. Some of the earlier authors [Amelung and Evers, 1962; Böni, 1959] write about positive results treating PCP; experimental studies do until today hardly exist. An exception is Fellmann's study [1972], in which, in a study with 30 patients suffering from PCP and morbus Bechterew, he could prove a subjective success of the climate cure: the cure's success was rated higher by the patients than what could be objectively found. The comparison of the objectively measurable results with other, with these diseases not so usual forms of climate therapy, leads to the conclusion that the climate cure can positively influence the course of disease of chronic-inflammatory rheumatic diseases and of the progressive-chronic polyarthritis, but still does not reach the success of the other therapeutical measures. Unfortunately, in the study it is not pointed out which elements the described climate cure consisted of.

The climate-therapeutical exposition method which seems, according to the nowadays state of knowledge and besides the hardening, to be the actual agent of climate therapy, is the heliotherapy. The body is exposed to the sun. With heliotherapy, adaptations in a variety of levels are to be achieved; one of the most known goals is an increase in vitamin D production. In his review paper, Peter [1990] summarizes the actual state of knowledge on therapeutic effects of ultraviolet, resp. heliotherapy treating locomotor and support system. According to Peter [1986, 1989], inflammatory activity and pain intensity of chronic polyarthritis is improved by whole-body ultraviolet radiation in the same way as by moor-baths; also Grigoriowa et al. [1987, quot. acc. to Peter, 1990] report a reduction of inflammatory process and improvement of joint function after increasingly dosed whole-body radiation. Peter lists in his paper also a number of recently published studies [e.g. Bühring, 1988 and Lemke et al., 1988, both quot. acc. to Peter, 1990], which confirm that heliotherapy for a variety of diseases of bone, like osteomalacia and osteoporosis, still has its justification. The mode of action of ultraviolet radiation on rheumatic diseases is still relatively unknown. According to Peter [1989], an unspecific stimulation of the immune regulation can be supposed: amongst others the fall of increased immune complex values and of immunoglobulin G to normal values and the increase of the at first decreased number of T lymphocytes is described. Although herewith first results are presented, there is still research and the securing of results to be done, concerning therapeutical

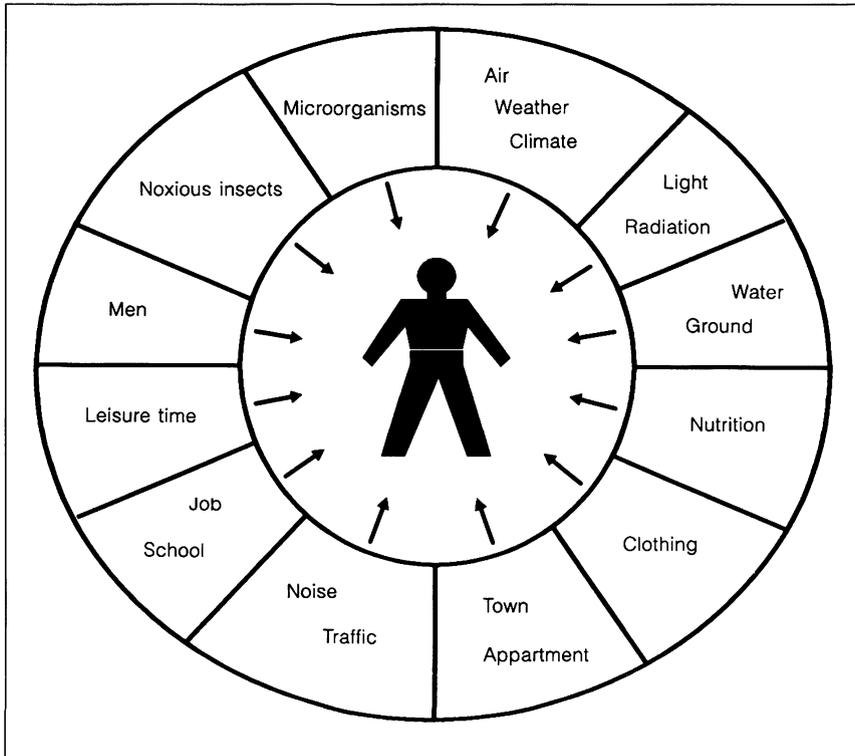


Fig. 4. Relevance of weather influences on rheumatic diseases.

success of heliotherapy and the physiological details it is based on, particularly in terms of arthritis.

In contrast to the variety of climate-therapeutical elements within a climate cure, in a therapeutical sense the long-term change into a different climate, according to today's state of knowledge, cannot be recommended: the knowledge so far still has to be worked upon.

Outlook

The influence of weather and climate on rheumatic diseases can, at least in terms of intensifying pain, be looked upon as secured; the deterioration of health state cannot be definitely correlated with certain meteorological

logical parameters or weather conditions. It is obvious that weather-caused pain additionally stresses the course of disease of rheumatics. However, the relevance of weather influences on rheumatic diseases together with other possible reasons have to be integrated into a common system of references (fig. 4). In the complete course of rheumatic diseases weather-caused influences only form a small part besides many other influences.

References

- Amelung W, Evers A: Handbuch der Bäder- und Klimaheilkunde. Stuttgart, Schattauer, 1962.
- Barcal R, Matousek J, Lavicka J: Zur Frage der Wetterempfindlichkeit bei Rheumatikern. Arch Physik Ther 1961;5:403.
- Böni A: Physikalische Medizin bei rheumatischen Erkrankungen. Dt Meteorol J 1959;10: 448.
- Bundesminister für Jugend, Familie, Frauen und Gesundheit (ed): Daten des Gesundheitswesens – Ausgabe 1987. Stuttgart, Kohlhammer, 1987.
- Curry M: Klimaunterschiede und wetterbedingte Reaktionstypen. Med Meteorol Hefte 1951;5:39.
- Dirnagl K: Wettereinflüsse auf Befinden und Krankheit; in Oepen I (ed): An den Grenzen der Schulmedizin. München, Deutscher Ärzteverlag, 1985.
- De Rudder B: Grundriss einer Meteorobiologie des Menschen, ed 3. Berlin, Springer, 1951.
- De Rudder B: Rheuma und Atmosphäre; in Hochrain M (ed): Rheumatische Erkrankungen. Stuttgart, Thieme, 1952.
- Engel A, Burch TA: Chronic arthritis in the United States. Arthritis Rheum 1967;10: 61.
- Faust V: Zur Symptomatik der Wetterfühligkeit. Münch Meteorol Wochenschr 1973;115: 441.
- Faust V: Was ist Wetterfühligkeit? Z Allg Med 1976;52:225.
- Faust V: Biometeorologie, ed 2. Stuttgart, Hippokrates, 1978.
- Fellmann N: Vergleich klimatherapeutischer Behandlungsergebnisse bei Erkrankungen des entzündlich-rheumatischen Formenkreises mit den Erfolgen heute üblicher Behandlungsmethoden. Z Angew Bäder- und Klimaheilk 1972;19:2.
- Flach E: Atmosphärisches Geschehen und witterungsbedingter Rheumatismus. Dresden, Steinkopff, 1938.
- Fleming A, Crown JM, Corbett M: Early rheumatoid disease. Ann Rheum Dis 1976;35: 357.
- Fuss J: Der Einfluss des Wetters auf das Schmerzempfinden der Rheumakranken; Diss Düsseldorf 1981.
- Geiger H, Gensler G: Korrelationen zwischen der Wetterfühligkeit und dem Ausfall verschiedener Funktionsprüfungen, sowie einer Befragung nach dem Freiburger Persönlichkeitsinventar. Z Phys Med 1975;4:201.
- Harlfinger O, Jacobi E, Richter O, Krüsskemper E, Krüsskemper HL: Untersuchung zur 'Wetterfühligkeit' Rheumakranker. NB Medici 1986;9:566.

- Hertel E, Ingenpass G: Die Viskosität der Gewebsflüssigkeit und ihre Bedeutung für rheumatische Arthropathien. *Z Rheumatol* 1974;33:379.
- Hill DF: Climate and arthritis; in Hollander JL (ed): *Arthritis in Allied Conditions*, ed 7. Philadelphia, Lea & Febiger, 1966.
- Hollander JL, Yeostros SJ: The effect of simultaneous variations of humidity and barometric pressure on arthritis. *Bull Am Soc* 1963;44:489.
- Hunter J, Kerr EH, Whillans MG: The relation between joint stiffness upon exposure to cold and the characteristics of synovial fluid. *Can J Med Sci* 1952;30:367.
- Jordan H: Zur Thalassotherapie der Rheumatoid-Arthritis. Ein Überblick. *Z Physiother* 1978;1:1.
- Lampert H: Die Reaktionstypenlehre und ihre Bedeutung für Balneologie und Klimatologie. *Arch Phys Ther* 1962;14:3.
- Latman N, Levi LN: Rheumatoid arthritis and climate. *N Engl J Med* 1980;13:1178.
- Latman NS: Influence of atmospheric factors on the rheumatic diseases. *Experientia* 1987;43:32.
- Lawrence JS: Epidemiology of rheumatoid arthritis. *Arthritis Rheum* 193;6:106.
- Lawrence JS: Climate and arthritis; in Licht S (ed): *Arthritis and Physical Medicine*. Baltimore, Waverly Press, 1969.
- Lawrence JS: Influence of Weather and Climate on Rheumatic Diseases. *Progress in Biometeorology*. Div A: 83. Amsterdam, Sweets & Zeitlinger, 1977.
- Lawrence JS, Behrend T, Bennett PH, Bremmer JM, Burch TA, Gofton J, Brian WO, Robinson H: Geographical studies on rheumatoid arthritis. *Am Rheum Dis* 1966; 25:425.
- Lawrence JS, Bremmer JM, Ball J, Burch TA: Rheumatoid arthritis in a subtropical population. *Am Rheum Dis* 1966;25:59.
- Levis-Faning E: Report on an inquiry into the etiological factors associated with rheumatoid arthritis. *Ann Rheum Dis* 1950;9:43.
- Mendez-Bryan R, Roger NL, Gonzales-Alcover R: Prevalence of rheumatoid arthritis in a tropical area. *Arthritis Rheum* 1963;6:765.
- Mikkelsen WM: Epidemiology of rheumatoid arthritis; in Hollander JL (ed): *Arthritis and Allied Conditions*. Philadelphia, Lea & Febiger, 1966.
- Möller F: Einführung in die Meteorologie. Mannheim, B.J.-Wissenschaftsverlag, 1973.
- Neergard K: Die Klimatherapie des Rheumatismus. *Balneologie* 1934;I:160.
- Patberg WR, Nienhuis RL, Veringa F: Relation between meteorological factors and pain in rheumatoid arthritis in a marine climate. *J Rheumatol* 1985;12:711.
- Pehl R, Weskott H: Wetter und Rheuma. *Münch Med Wochenschr* 1955;97:946.
- Peter A: Besonnung und Resistenz. *Ärzteforum Phys Ther* 1986;4:2.
- Peter A: Moorbäder und Ultraviolettstrahlen unter dem Aspekt der Immunregulation bei Rheumatoid-Arthritis. *Z Physiother* 1989;41:167.
- Peter A: UV-Exposition und Heliotherapie im Kurort als Adjuvanz einer Balneotherapie. *Z Phys Met Baln Klim* 1990;19:1.
- Pilger A: Chronische Polyarthritiden und Klima. *Med Clin* 1970;65:1363.
- Richner H: Zusammenhänge zwischen raschen zwischenatmosphärischen Druckschwankungen, Wetterlage und subjektivem Befinden. Zürich, LAPETH-8, 1974.
- Rothschild DM, Masi AT: Pathogenesis of Rheumatoid Arthritis: A Vascular Hypothesis. *Semin Arthritis Rheum* 1982;12:11.
- Schaich J: Wetterfühligkeit – Nichtwetterfühligkeit. Analyse einer Erhebung; diss München 1974.

- Schuh A: Das Krankheitsbild des Trainingsmangels und seine Behandlung durch Klimatherapie in Form einer Terrainkur unter kühlen Bedingungen; Habschr München 1989.
- Sönning W: Wettereinfluss bei rheumatischen Erkrankungen. *Ärztl Praxis* 1979;31:3138.
- Sönning W: Zur biosynoptischen Arbeitshypothese. *Z Phys Med* 1983;1:25.
- Sönning W, Jendritzky G, Storck H: Der Wettereinfluss im Verlauf der chronischen Polyarthrititis. *Wetter Leben* 1979;31:244.
- Thompson HE: Climate and rheumatic diseases. *Ariz Med* 1951;8:31.
- Tromp SW: The influence of weather and climate on urinary volume, pH, 17-ketosteroids, hexosamines, Cd, K, Na and urea. *J Biometeorol* 1963;7:59.
- Tromp SW: *Biometeorology*. London, Heyden, 1980.
- Tromp SW, Bouma J: Possible relationship between weather, hexosamine excretion and arthritic pains. *Int J Biometeorol* 1966;10:105.
- Weitbrecht W-U, Simon F: Einfluss von meteorologischen Parametern auf Akutaufnahmen von Patienten mit lumbalen Bandscheibenvorfällen. *Z Orthop* 1989;127:650.

Subject Index

- Absolute air humidity 24
ACE inhibitors 89
Acetabulum 146
Acetaminophen, anti-inflammatory treatment 181, 182, 185
Acetylsalicylic acid 43, 44
ACTH stimulation, adrenal cortex 176, 177
Acute fibrinoid exudation 95, 96
Acute heart failure 83
Adrenal cortex suppression 176
Adrenal hormones 15
Adrenaline 15
Adrenoceptors 90, 91
Aerobics 67, 70, 71, 162, 165, 220
Age and juvenile chronic arthritis 118, 119
Aggression and psychosomatics 61–71
Algesic substances 43
Allergy 16
Alpha adrenoceptors 90, 91
Amino acid metabolism 11, 12
Analgesics, treatment of inflammatory-rheumatic diseases 181, 182, 185, 186
Ankle joint, clinical examination 150
Antibiotics 202–205
Antibodies 6, 7, 13, 91–93, 97–99, 101, 103–105, 109
 monoclonal 195, 196
Antibody therapy 193–196
Antidromic vasodilatation 46
Antigen-presenting cells 189, 190
Antigens 8, 9, 98, 189–191
Anti-HLA class II antibodies, immunomodulation 195
Anti-idiotypic antibodies 194, 195
Anti-idiotypic network 9
Anti-M7 proteins 91
Antimalarials 179, 185
Anxiety 68, 70
Aortic regurgitation 107
Arachidonic acid 45
Arrhythmias 93
Arthritides 200, 204
Arthritis, *see* Rheumatoid arthritis
Arthroplasty 209
 hip 135–138
Articular index 28
Aschoff nodules 95, 96
Athlete therapy 153–158
Atrial natriuretic peptide 89, 90
Autonomic nervous system 89, 90
Autoreactivity hypothesis 87
Azathioprine 179, 180

B cells, immunoglobulins 191–195
Back pain 167, 168
Bacteria, rheumatic diseases 200
Badminton 133
Bang nodule 95, 96
Bechterew's disease, *see* Spondylitis ankylosans
Benzathine penicillin G 203, 204
Benzodiazepines 184
Beta adrenoceptors 90
Bicycle riding 123, 124, 135, 164
Biomechanics, joints 142–152
Bowling 133
Bradykinin 43, 48
Brain 50
'Brain-endorphin hypothesis' 67
Build-up, sports training 161

C fibers 45
Calcitonin gene related peptide 45
Calcium homeostasis 86, 87
Cardiac amyloidosis 101

- Cartilage 142, 143
 CD2 cells 190
 CD3 cells 190
 CD4 antigens, rheumatic arthritis 196
 CD4 cells 12, 13, 15
 CD8 cells 12, 13, 15, 191
 Ceftriaxone, Lyme borreliosis 204
 Cellular immunity 98
 Central nervous system 7, 11, 51–54
 Chlamydia bacteria 200
 Chloroquine 180, 185
 Chronic arthritides, inflammatory bowel diseases 121
 Chronic heart failure 83
 Chronic polyarthritis 35, 36 99–101
 Ciclosporin A 178, 179
 Circulatory failure 83
 Climate, effect on rheumatic diseases 22–37
 Climate therapy 34–36
 Clinical index 25, 29
 Clone 9, 10
 Cold 29
 Cold desensitization 34–36
 Cold front 24
 Collagen 87
 Collagen damage hypothesis 87
 Collagen diseases 101–106
 Colony-stimulating factor 100
 Compensatory lordoses, clinical examination 149
 Competence signal 88
 Compression neuropathia 47
 Congestive heart failure, *see* Heart failure
 Coordination and sports activity 156
 Coronaritis 102
 Coronary arteritis 105, 106
 Coronary heart disease 110
 Corticosteroids 106
 Cortisol 10, 14
 Cortisone administration 176
 CREST syndrome 108–110
 Crohn's disease 193, 194
 Cross-country skiing 124, 125, 133, 136, 157, 158, 164, 216
 'Cushing level' 176, 177
 Cyclic adenosine monophosphate 90
 Cyclooxygenase 43, 44
 Cyclophosphamide, alkalinizing substance 178, 179
 Cytokines, immunomodulation 191, 196–199
 Cytoskeleton 88, 89
 Dancing 124
 Deafferentation pain 40
 Defense mechanisms 6–9, 14–17
 Degenerative joint disease, effect of physical activity 129–138
 Delayed-type hypersensitivity 98
 Depression 67, 68, 70
 Diastolic dysfunction 86, 87
 Disease course hypothesis 64, 66
 Disease-modifying agents 178
 Disease onset hypothesis 64, 65
 Dorsal horn 52–54
 Downhill skiing 134
 Doxycycline, Lyme borreliosis 204
 Drug therapy, rheumatic disease 170–186
 Dysregulation pain 40

 Early-onset pauciartthritis 120, 121
 Ejection fraction 84
 Elbow joint, clinical examination 149
 EMG levels 48
 Endoprosthesis, sports activities 209, 217
 Endorphins 67
 Endothelial cells 100
 Endurance training 221
 Energy production 84–86
 Enthesopathies 175, 177
 drug therapy 172
 Examination, clinical, sports activities 148, 150
 Exercise
 immune diseases 16, 17
 immune system 11–18
 osteoarthritis 130–132
 psychosomatics 66–71
 Exhaustion 83
 Expiratory position 155
 Extracellular matrix 91

 Factor VIII 195
 Fibrin 99
 Fibroblasts 87, 88
 Fibromyalgia 167, 183
 Fibrosis 87, 88, 110
 Fitness center visit 165
 Foot, clinical examination 150

 Gait pattern, examination 148
 Gamma interferon 179
 Gi protein 90
 Gold 179–181
 Gold sodium thiomalate 46
 Golfing 136
 Gonarthrosis 133

- Gonococcal arthritis 202
 Granulocytes 14
 Growth factors 87, 88
 Gymnastics 135, 151, 165, 215
 effect on endoprosthesis 215

 Heart failure 82–92
 Heliotherapy 35, 36
 Heritability coefficients 63
 Hexosamine 33
 High-pressure weather area 23, 24
 Hiking 135
 Hip arthroplasty, physical activity
 135–138
 Hips, clinical examination 149, 150
 HLA-B27-associated chronic arthritis 121
 HLA class II antigens 199
 HLA-DR2 99
 HLA-DR4+ 99
 Hormones, immune system 10, 15
 Horseback riding 133
 Hostility, psychosomatics 61–64
 Humoral immunity 98, 99
 Humpback formation 154
 Hypertension 107, 108

 Ice skating 134
 Idiopathic thrombocytopenic purpura 194
 IgA 7
 IgE 8
 IgG 8, 105
 IgM 8
 IgM-rheumatoid factor positive – seropositive
 – adult polyarthritis 120
 Immune complexes 8, 9, 103, 104
 Immune response, regulation 9–11
 Immune system
 exercise 5–18
 immunomodulation 188–205
 Immunodeficiency 16, 17
 Immunoglobulins 8, 13, 191–195
 Immunopathy 8
 Immunosuppressive drugs 178, 185
 Impulse loading 150–152
 Infections 4
 arthritis 200–205
 Inflammatory rheumatism 1, 2
 Insulin-derived growth factor 88
 Insulin receptors 14, 15
 Interferons 199
 Interleukin 1, inflammatory rheumatic
 processes 197, 198
 Interleukin 2 190, 191, 193

 Interleukin 6, inflammatory rheumatic
 processes 198
 Isometric strengthening 221
 Isotonic strengthening 221

 Jogging 134, 136, 164
 Joint affection 167
 Joint cooling 166
 Joints 41, 42, 119–126, 129–138
 biomechanics 142–152
 Jones criteria 94
 Juvenile chronic arthritis 118–126
 Juvenile psoriatic arthritis 121
 Juvenile spondylarthritis 121

 Knee joint region, clinical examination 150
 Kyphoses 154
 clinical examination 149

 Latitudes 30
 Leukocytes 15, 16
 Leukocytosis 15
 Ligament-stabilized joints 122
 Light, immune system 11
 Locomotor system 118, 119
 diseases 170, 171
 Locus of control theory 69
 Low-pressure weather area 23, 24
 Lumbalgias, clinical examination 149
 Lumbar scoliosis, result of false postures 146,
 147
 Lyme borreliosis 201, 202
 treatment 204, 205
 Lymphocytes, exercise 12

 Major conflict group 63
 Major histocompatibility molecules 188, 189
 Mechanical overload hypothesis 87
 Mediator and growth factor hypothesis 87
 Memory 9
 Metabolism 16
 Metamizole, anti-inflammatory treatment 181
 Methotrexate 178–180
 Mitral stenosis 94, 95
 Mobility, physical therapy 155, 156
 Monoclonal antibodies, *see* Antibodies,
 monoclonal
 Morphine 53, 54, 182
 Motor control dysfunction 48–51
 Motoric apparatus 61
 Motoric basic features 155
 Motoric strength, improvement 155
 Mountain hiking, therapeutic sport 163, 164
 Mountain tours 134

- Mucosa-associated lymphatic tissue 7
 Muscle-aggressive granuloma 95, 96
 Muscle hypertonus 48–50
 Muscle massage 166
 Muscle stretching 166
 Muscle tonus hypothesis 61
 Muscular force 146
 Musculoskeletal system pain 40–54
 Myocarditis 91–95, 101–103
 Myocardium, diseased 87, 88
 Myolemma 91
 Myositis 180
 Myotendoperiostosis 167
 Myotonolytics 182, 183, 185
- Neighboring joints, effect of mechanical impairment 146–148
 Nerve axons 47
 Nerve sprouting 47, 48
 Nerve transection 47, 48
 'Neuro-endocrino-psycho-immunology' 11
 Neurogenic immobilization 50
 Neurogenic inflammation 45
 Neurohumoral factors 89, 90
 Neuroma 47, 48
 Neuropathic pain 40, 47, 48
 Neuropeptides 53
 Neurotransmitters 51–54
 'Neutril-nil method' of examination 148
 Neutrophils 15
 Nociceptor pain 40, 41–43, 51–54
 Nociceptors 41–43
 Nonconflict group 63
 Nongonococcal arthritis 202, 203
 Nonspecific defense mechanisms, immune system 187, 188
 Nonsteroidal anti-inflammatory drugs 171–174, 185
 Noradrenaline 90
 Nutrition, immune system 10, 11
- Opioids 46
 Oral gold 179–181
 Osteoarthritis 70, 129–132, 167
 Osteoporosis 175, 181
- Pain 51–54, 67, 68
 musculoskeletal system 40–54
 weather 25, 29, 31–33
 Parenteral gold 179, 180
 Passive physiotherapy 166
 Pauciartthritis 120, 121, 125, 126
 D-Penicillamine 179, 180
 Pericarditis 93, 102, 103, 107
- Perseverance training 155
 Photosensitization inducement 180
 Physical activity 3, 17, 82
 degenerative joint disease 129–138
 rheumatoid arthritis 59–71
 spondylarthritis 153–158
 Physical education 124, 125
 Physiotherapy 121
 integration of sportive elements 160–168
 Platelet-derived growth factor 88
 Platelets 15
 Polyarthritits 34, 35, 46, 120, 121, 125, 126
 Polyclonal human immunoglobulins 193–195
 Progressive systemic sclerosis 107–110
 Prostaglandins 43
 inhibition of synthesis 171, 172
 Prosthesis 135–137, 209–217
 Psoriasisoid arthritis 62
 Psychic well-being, effect of physical activity 156, 157
 Psychoimmunological hypothesis 65
 Psychopharmacological agents 183–185
 Psychosomatic pain 40
 Psychosomatics, inflammatory rheumatic diseases 60–70
- Reactive arthritis 200
 treatment 203
 Reactive pain 51
 Regulator cells 9
 Reiter syndrome 106, 107, 201, 203
 Relative air humidity 24, 29
 Renin-angiotensin-aldosterone system 89
 Renin-angiotensin system 108
 Respiration, restriction 155
 Retropatellar arthrosis 147
 Rheumatic carditis 95, 96
 Rheumatic diseases 1–4
 drug therapy 170–186
 effect of climate 22–37
 heart 81–110
 Rheumatic fever 94–99, 201
 treatment 203, 204
 Rheumatic heart disease 81–87
 Rheumatic inflammatory diseases 177
 Rheumatic spondylitis, physiotherapy 167
 Rheumatoid arthritis 60–71, 219–227
 frequency 31, 32
 joint replacement 208–217
 kinetotherapy 221
 physical activity 59–71
 psychosomatics 60–70
 range of motion 221
 sports activities 222

- Rheumatoid factor 102
 Rheumatoid heart disease 81–87
 Rheumatology, sports medicine 1–4
 Rowing 133, 135
 Run-in 162
 Running and osteoarthritis 130, 131
 Run-out 162
- Sacroiliitis 124
 Sailing 133, 134
 Sarcolemma 91
 Scleroderma heart disease 109, 110
 Sclerotic pelvis 146
 clinical examination 148
 Season 31, 32
 Semper-et-omnia syndrome 176
 Septic arthritis 200
 treatment 202, 203
 Seronegative polyarthritis 120
 Seronegative rheumatism 63
 Seropositive rheumatism 63
 Serotonin 43
 Sexual hormones, immune system 10
 Shoulder, clinical examination 149
 Single-sided overstrain 161
 Skiing, effect on endoprosthesis 216
 Soccer 134
 Specific hypothesis 64, 66
 Spinal column 148
 clinical examination 148, 149
 Spinal cord 51
 Spondylarthritis 106
 physical activity 153–158
 Spondylitis ankylosans 106, 107, 154–158
 sports participation 158
 Spondylolisthesis, result of false postures 147
 Sports
 effect on endoprosthesis 213–217
 joint replacement 208–217
 juvenile chronic arthritis 118–126
 rheumatics 69
 rheumatology 1–4
 risks 3, 4
 Sports medicine consultation 150–152
 Squash 134
Staphylococcus aureus 200
 Steam pressure 24, 25, 29, 30
 Steroid myopathy 176
 Steroids 14
 Still's disease 102
 Strain on endoprosthesis 211–217
 Streptococci 97
- Stress
 arthritis 68, 69
 immune system 11, 65
 Structural scolioses, clinical examination 149
 Substance P 44–47, 52, 53
 Sulfasalazine 149, 150
 Swimming 123, 132, 133, 135, 151, 157, 158, 164, 167, 215
 Sympathetic dysfunction 51
 Synaptic terminals 52
 Synovial fluid, viscosity 33
 Synoviocytes 46
 Systemic juvenile chronic arthritis 119, 120
 Systemic lupus erythematosus 102–106
 Systolic wall stress 84
- T cell receptors 189, 190
 T cells 6–10, 88
 antigen recognition 188–191
 Table tennis 124, 125, 133
 Talipes equinus, result of false postures 147
 Temperature 32, 33
 Tendon, effect of corticosteroid therapy 175, 177
 Tennis 133, 136, 165
 Thalassotherapy 34
 Therapeutic sport 153, 163–168
 Thermic effect complex 24, 25
 Thoracic vertebral column, immobility 154, 155
 Total hip arthroplasty 135–138
 Training, sport-type-specific state 161, 162
 Trendelenburg's test 150
 Tumor necrosis factor, inflammatory
 rheumatic processes 197, 198
- Ultraviolet radiation 35
- Valvular lesions 93
 Vascular tone 89, 90
 Vasculitis 99
 Vasoconstriction 89
 Vasodilatation 89
 Ventricular inflow 86
 Volleyball 157, 158
- Walking 133, 136
 Wall tension 83, 84
 Warm front 24
 Warmth 166
 Weather sensitivity 22–33

