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WEATHER ELEMENTS AND

MYOCARDIAL INFARCTION

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INTRODUCTION

Myocardial infarction, which the layman calls a "heart attack," refers to the death of part of the myocardium. Such death results from occlusion within the coronary arteries through coronary artery disease, embolus, thrombus or a combination of these. Infarction can also occur when the demands made upon diseased coronary arteries are too great.

A question of paramount importance in preventive medicine is: what causes coronary artery disease (CAD)? A dietary intake of large amounts of cholesterol and animal fat, personality type, inadequate physical activity, and cigarette smoking all appear to play a role. Another question of considerable interest is: once disease is present, what precipitates an acute attack of myocardial infarction? Extremes of weather have long been implicated as a triggering force.

In the plant kingdom, the geographic distribution of natural vegetation is determined largely by climatic parameters. As Strahler (64) has expressed it, "It is a law of bioclimatology that there is a critical level of climatic stress beyond which a plant species cannot survive; . . ."

It can readily be seen that this is not as true in the animal kingdom, particularly in regard to man. Man has learned to adapt to a wide range of climatic conditions. Nevertheless, there is abundant proof that weather does affect the human organism, especially when disease has altered it. It is the purpose of this study to explore the possibility of a "critical level of climatic stress" for the person suffering from CAD. The scope of inquiry was 261 cases of myocardial infarction at Akron City Hospital and 218 cases at Baptist Memorial and St. Anthony Hospitals in Oklahoma City during the year 1965.

No literature dealing specifically with the problem under investigation was found in the traditional geographic literature. Therefore, the library research took place at the Akron City Hospital library, and the Medical Library at Case Western Reserve University. Some material was obtained through the inter-library loan service at Kent State University.

CHAPTER I

THE EFFECT OF ATMOSPHERIC CONDITIONS ON THE CARDIOVASCULAR SYSTEM

Man increasingly has been able, particularly in the developed countries of the world, not only to protect himself from his physical environment, but indeed to create an artificial environment. In the United States almost all homes are artificially heated in the colder months, and an ever growing number are cooled during hot weather as well. Furthermore, a large number of people also work in artificial environments. Moreover, private automobiles and public carriers alike have heating systems, and many air conditioning as well. In addition, year round artificial regulation of temperature frequently extends to the local supermarket and clothing store, the library, the sports arena and concert hall. It would appear that almost all Americans escape the natural environment at least part of the time.

But what exactly have they escaped? Obviously extremes of temperature for the most part, particularly protection from the cold. Changes in relative humidity go hand in hand with changes in air temperature; the lowering of temperature decreases the ability of the air to hold moisture, therefore, relative humidity increases, and with warming the opposite is true. Such changes are also seen in nature in that the daily march of temperature is accompanied by lower relative humidity during the warmer part of the day and higher relative humidity during the cooler part of the day (assuming no change in the absolute humidity). All too often relative humidity levels drop to uncomfortably low levels (10%) in artificially heated buildings. Relatively few homes or public buildings have facilities to maintain relative humidity at a comfortable level.

Our penchant for spending most of our lives indoors has also considerably reduced our exposure to the wind, although a rather common experience is the feeling of a cold draft in one's home, particularly when near outside walls, on cold, windy days. Also, many people feel that the "healthy" thing to do is to sleep with a window open.

Buildings and clothing also protect us from the direct effects of sunlight and precipitation. This is not the case with atmospheric pressure and air ionization, both of which are present with man indoors.

All of the elements of weather just mentioned, with the possible exception of precipitation per se, have been implicated by physiologists and others as playing a role in man's state of health. Meteorotropic effects are evident in such diseases as asthma, cancer of the skin and acute glaucoma. The high incidence of CAD in the United

States and parts of Europe, insures much research into its causes and those factors which may trigger coronary occlusion and infarction. Burch (10) has stated that it is a, ". . . well-known physiologic fact that the cardiovascular system of homothermic animals, like man, participates significantly in adjustments to variations in environment."

Like all bodies, the human body loses heat through conduction, radiation, convection, and evaporation. The latter two are aided by air movement. Changes in the temperature of the external environment call the body's thermal regulatory processes into play. With increases in the external temperature the surface blood vessels become dilated as the blood carries heat from the site of production to these vessels for transference to the atmosphere. At skin temperatures above 96.8°F., cutaneous blood flow increases precipitously. The work load of the heart is closely related to the degree of cutaneous vasodilation (11). When convection, conduction and radiation rates are insufficient for the task of cooling, sweating takes place. According to Burch (10), "When sweating becomes necessary to maintain thermal equilibrium, not only is the cardiovascular system more actively at work, but an additional physiologic stress also concerned with water and electrolyte metabolism occurs." It can readily be seen that under atmospheric conditions of high relative humidity[1] and little air movement, evaporation will be retarded.

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[1]More precisely, the evaporation of sweat depends not

The body's reaction to cooling consists, initially, of peripheral vasoconstriction. This response reduces the amount and rate of blood flow through the tissues, thereby, reducing heat loss. The resulting increase in arterial pressure has a tendency to increase oxygen consumption by the heart. In the presence of CAD this could result in a serious discrepancy between the oxygen demand and delivery to those areas of the myocardium served by vessels narrowed by disease.

Should the external temperature continue to fall, physical thermoregulation must augment vasoconstriction. The first evidence of this is an increase in muscle tone which causes a remarkable increase in the production of body heat. Continued cooling ultimately results in shivering, once again causing a vast increase in heat production. In order to maintain the concomitant high metabolic rate, great demands are made upon the heart to increase output. Unusually strenuous physical activity, in such circumstances, might prove to be the coup de grace to the diseased heart. Raab, et al. (54) have suggested that coronary occlusions that result under such circumstances may, in fact, result from excessive amounts of sympathetic amines being released at such a rate that the system is unable to detoxify them fast enough to prevent cardiovascular damage.

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on the relative humidity, but rather on the physiological saturation deficit, i.e., the difference between the partial pressure of water vapor in the atmosphere and the saturation pressure of the water vapor at the surface of the skin.

It might be well at this point to mention that the foregoing statements about the response of the human organism to external thermal stress are generalized. The absence of response, or the degree to which it takes place follows Wilder's law of the initial value. The following from Grayson (28) will serve as an illustration.

> The sensitivity of this reflex (vasoconstriction), however, has been shown to be highly dependent on "core" temperature. It can be abolished by artificially raising the rectal temperature. Thus in a subject whose rectal temperature has been raised (by immersing the legs in hot water and covering with blankets) such reflexes can be totally abolished and it is possible to place the legs of such an artificially hyperthermic subject in ice cold water without invoking even a trace of cutaneous vasoconstriction. Conversely, cooling the subject has been shown to increase the sensitivity of such cutaneous vascular responses.

The body is insulated by a thin layer of air in which heat conduction is at a minimum. There is an inverse relationship between the thickness of this layer and wind speed. In fact, in wind gusts, this layer is completely removed. Dr. Frank Field (21) says, "On the other hand, if one were investigating an upsurge in cases of myocardial infarction on a windy day, reference to the average quantities just mentioned would probably be inadequate, since they would not reveal the strong gusts that might have provided the 'trigger' for susceptible persons who went outdoors even briefly." Therefore, whereas air movement is considered beneficial in thermoregulation when air temperatures are high, the opposite is true at low air temperatures. The adverse affect of cold wind is further enhanced when vapor pressure is high since the water vapor increases the rate at which the body loses heat through conduction. Czekin (13) found that at wind speeds of 5-10 meters per second, the incidence of infarctions was much higher than at speeds either below or above that range. The general conclusion that one gets from the literature is summed up by Tromp (66), "Patients suffering from arterio-sclerotic and related heart diseases . . . should protect themselves in winter by staying at home on very windy days. . . ."

The autonomic nervous system, comprised of the orthosympathetic, the parasympathetic, and the peripheral nervous systems, reacts to external stimuli (66). It is the peripheral nervous system that makes it possible for one to perceive a difference between the temperature of the body and its environment. The role of the other two parts of the system is to make it possible for man to adapt to sudden environmental change and then to reestablish physiological equilibrium. Some of the organs of the body are supplied by only one of the sympathetic nervous systems,[1] while others, such as the heart, are innervated by both. In the latter instance, the nervous systems are somewhat antagonistic to one another. The parasympathetic, when stimulated, secretes

[1]For example, the glands of the pancreas are innervated by the parasympathetic system, and the uterus by the orthosympathetic.

acetylocholine[1] which slows the heartbeat, causes the contraction of some of the peripheral blood vessels, and the blood pressure to drop. While the secretion of adrenaline by the orthosympathetic makes the heart beat faster and with greater force, dilates the blood vessels in the skeletal muscle and the heart, while constricting arterioles and capillaries in the skin, and increases blood pressure. Furthermore, through the indirect excitation of the liver, adrenaline reduces the coagulation time of the blood.[2] Clearly, a condition condusive to the formation of blood clots.

Research has shown that both sympathetic systems are affected by the passage of both warm and cold fronts, somewhat more quickly in the case of the latter (66). In addition, it appears that the reaction of the parasympathetic often takes place a few hours ahead of frontal passage, with the reaction of the orthosympathetic (adrenaline) following by about eight hours, and after frontal passage. However, this sequence may be reversed in those persons with an unstable sympathetic nervous system. Brezowsky (8) reported an increase in the coagulative ability of the blood ahead of weather fronts. In this connection, he stated that it is not a coincidence that there is an increase in the incidence of embolism and thrombo-phlebitis on the warm side of the biosphere.

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[1]Acetylcholine is also liberated by the pre-ganglionic fibres in the orthosympathetic system.

[2]Included in the several blood proteins manufactured by the liver are certain clotting substances.

Frontal activity is often accompanied by rapid, sometimes great, changes in barometric pressure. Attempts by several researchers to correlate changes in blood pressure with changes in barometric pressure have been successful, but the results of one study vs. another, contradictory, thus seemingly putting barometric pressure into the role of indicator. Further complicating the picture, it has been shown that people with normal blood pressure and hypertonics (without vascular disease) react differently from hypertonics with arteriosclerosis when subjected to rapidly falling pressure in a climatic chamber. In the latter group, both systolic and diastolic pressure increased. The former group exhibited a slight decrease in systolic blood pressure after about 10 minutes (66).

Barometric pressure also falls with an increase in elevation. Some of the physiological changes effected by altitude are an increase in heart rate and blood pressure, and an increase in the fibrinogen content of the blood. Yet infarcts and coronary thrombosis seem to be rare in Peru, but the same can not be said about Switzerland.

The ever increasing body of knowledge about the effect of the natural environment on man has resulted in a large number of studies dealing with myocardial infarctions and weather. In the following chapter, some of these studies are reviewed.

CHAPTER II

A REVIEW OF SOME OF THE PAST LITERATURE

The purpose of this chapter is to present a review of selected research that has been done in regard to heart attacks and weather. Such a presentation not only provides background material but also manifests the relationship between the present study and others that have been done.

"A.O.W." (2) in the Letters, Notes, Etc. section of the British Heart Journal of December 5, 1925, wrote the following words: "About forty years ago I began to notice that three elderly women with heart trouble used to send for me to visit them on almost the same day; they had no knowledge of each other." He goes on to say that these requests coincided with a high barometer. At this point, his interest thoroughly piqued, he began to follow, during the month of November, the announcements of "sudden deaths" that appeared in the London Times. He would then take note of the barometric pressure reading on the day of these deaths. He reports that he found a relationship between the sudden deaths and high pressure readings. One of the readers who took note of the above was Dr. Percy Stocks, (63) medical officer to the Department of Applied Statistics and

Eugenics in the University of London. He took figures on deaths due to diseases of the heart and circulatory system during the month of November, 1925, eliminated the effect of temperature, and reported that he found a small but significant relationship between heart deaths and high pressure. In the more than 40 years since this study, many researchers have been intrigued enough by the possibility of a connection between certain heart problems and the weather to do studies of their own.

After reviewing the notes taken from the studies shown in the bibliography, it appeared possible to group the approaches of the several researchers into three general categories, even though there is some overlapping.

These three categories are:

1. those who devote their attention primarily to the monthly and seasonal incidence of myocardial infarctions,
2. those who investigate the relationship between individual weather elements and the incidence of infarcts, and
3. those who look at weather elements in combination in relation to the onset of infarcts.

Category 1

In the first group one finds such researchers as Hoxie, (35) in Los Angeles reporting that acute attacks of coronary occlusion were most frequent in winter and spring.

Billings et al. (5) reported that in Nashville, Tennessee, the greatest number occurred in winter, followed closely by fall. During the 1930's and 1940's several researchers in the United States reported that in northern cities, for example, Philadelphia and Boston, the highest incidence of infarcts and coronary occlusions occurred in the wintertime. Then in 1953 Heyer, Teng, and Barris (32) reported from Dallas that there was an increased frequency of myocardial infarction during summer months in that city. It would appear at this point that the idea of seasonality of incidence was still moot.

However to say this would be to miss the point, which is, what is "winter," what is "summer?" Is "winter" in Los Angeles the same as "winter" in Boston? Certainly not if one were to use the same criteria of temperature to describe the season. In other words, one should note that here, as in other types of literature, terminology concerning seasons is used much too loosely. In many studies, a season is one fourth of a year, that is, "winter" is December, January and February, and so on. In the popular use of the term season, the year is divided on the basis of noticeable changes in temperature, (or precipitation in a tropical wet and dry climate). Yet in many parts of the world such changes either do not exist or are barely perceptible.

In addition to the above problem, one also notes from time to time an unfortunate lack of precision in some of the literature with relation to weather and climate. For example, Bradlow and Zion (6) while discussing the reports of Heyer, Teng, and Barris, (32) and that of Teng and Heyer, (65) write, "One group found a higher summer incidence but later reported an increased frequency during periods of sudden inflow of both polar air and tropical air masses." This quote implies that in the second and more recent report there was a change of mind in regard to the high summer incidence, because the researchers were now reporting increased frequency of infarcts during inflows of polar air. As if to say that polar air does not invade the Dallas area in summertime, when of course one does find the passage of cold fronts in the American southwest during the hottest time of the year. Moreover one finds in Schrire (59) the following sentence: "Even in the temperate areas which correspond to our local conditions in Cape Town, such as Los Angeles and Nashville, Tennessee, a higher incidence in the winter months has been recorded." Climatologists would have a great deal of difficulty equating the climate of Cape Town and Los Angeles with that of Nashville, Tennessee.[1]

A new dimension was added to the seasonal approach by Kishon and Kariv. (39) After finding nothing statistically

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[1]Cape Town and Los Angeles both have a Mediterranean climate characterized by warm, dry summers and mild, wet winters. Nashville has a continental climate with hot summers, cool winters, and no season of drought. These two areas also differ in regard to air masses, total amount and form of precipitation, winds and so on.

significant when comparing the incidence of infarcts and "standard" seasons, they re-divided the year into seasons that took cognizance of the temperature curve; that is, the "extreme cold season included that part of the year in which the minimum temperature[1] fell below 10°C. and until it rose again to 9°C.[2] The extreme hot season was considered as beginning as soon as the maximum temperature exceeded 27.5°C. and continuing until it dropped to 28.5°C." In this way they found that in three out of five years, after a lag of ten days after the start of a hot season and a cold season, there was a significantly increased incidence of infarcts in these extreme seasons as compared with the autumn and spring.

In their study individual weather elements were also examined.[3] These were shown graphically in the following manner. Mean temperature change and wind velocity as compared with that of the previous day was shown in relation to a base line. The daily mean relative humidity was also used

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[1]The authors do not explain whether this refers to the daily minimum. If so, if the temperature fell below 10°C. on one day, but was above this point for the next 8 days before dropping below 10°C. again, on which day would the "cold season" begin? The same problem obtains for the criteria for the "hot season." On the other hand, perhaps weekly or monthly maximum and minimum averages were used.

[2]The one degree difference was arbitrarily chosen in order to reflect physiological adaptation to temperature conditions.

[3]The five years under consideration were first divided into standard 10-day units of time.

and the rate of the value was expressed by the vertical distance of a symbol for each day from the base line, but in this case the values were not related to that of the previous day. Then in the center of the graph there are four lines representing (1) the mean barometric pressure curve representing the change that was observed in mean barometric pressure from one standard ten day period to the next. The other three lines indicating, respectively, (2) the mean values of daily maximum, (3) minimum and (4) average temperatures, also calculated for standard ten day periods. In their discussion the authors state that no clear correlation is evident between any of these elements and the incidence of cardiac infarction. They do go on to comment that since they found, on their graph, the barometric curve varying inversely with that of temperature that perhaps the increased incidence of infarcts that they found with extremes of temperature could also be related to extreme values of atmospheric pressure. The fallacy in this is that even though it is true that this inverse relationship appears to be evident when temperature and pressure are presented, as they were here, a closer examination of changes in temperature and barometric pressure do not show this perfect inverse relationship. One can frequently find falling temperature and falling barometer, and conversely rising temperature and rising barometer. At other times, even when it is clear that there is a connection between a change in pressure and a change in temperature, for example, in Akron when the winds are going to shift around to the northwest behind a cold front bringing an influx of colder, drier air, accompanied by a rising barometer, the two events are often separated by many hours. In addition to this, there is also the much less pronounced diurnal cyclic change in barometric pressure which is generally obscured by the larger changes in the atmosphere.

Fogel and Righthand (32), although concerning themselves mainly with the seasonality of infarcts, found no significant correlation between the incidence of heart attacks and weather. However, one must regard their use of the weather data with serious reservations. In this study, monthly values were used, that is, monthly values of total precipitation and maximum and minimum and average temperatures, and maximum and minimum and average pressure. The former values were obtained from Stamford, Connecticut (where the study was being done) and the latter values (pressure) were obtained from LaGuardia Field, New York City.

Category 2

Among those researchers who feel that there is a relationship between some weather element or elements and myocardial infarctions, are Maschas _et al_. (46) and Adesola (1). In the former study it was reported that the intensity of sudden variations of temperature significantly influenced the onset of infarcts. In the latter study the author says, "Generally as the minimum temperature falls, the admission rate increases. This inverse relationship remains significant both on examination of the crude data and on further analysis where the barometric pressure is constant." In support of this statement he offers the following figures.

Minimum Temperature	Observed Cases	Expected Cases
under 35°F.	44	34.5
35-39	37	38.4
40-44	70	48.7
45-49	35	45.3
50+	51	70.1

On the last page of this article the author makes an interesting comment: "Halse and Quennet found that hyperprothrombinaemia and thrombo-embolism occurred with a drop in barometric pressure. It is possible that this relationship is only an indirect one, and that temperature plays an important part here. If this proves to be the case, then we have here a possible explanation of the findings in the present study." It is not clear what is meant by this comment. It appears that Adesola feels that a drop in barometric pressure has falling temperatures as a concomitant.

Yet this is less true than the idea of a perfect inverse relationship between pressure and temperature previously discussed.

Hankiewicz and Machalski (31) approached the problem somewhat differently, analyzing various atmospheric conditions in the following manner. They recorded mean daily air temperatures and divided them into intervals. They then noted the number of days, expressed as a percent of the total number of days on which the air temperature was in any given interval, and compared these figures to the associated number of infarcts, expressed as a percent of the total number of infarcts. These two figures, that is the percent of infarcts and the percent of days, were then subtracted. Daily range of temperatures, atmospheric pressure, wind speed, and relative humidity were examined in the same manner. Similarly the maximum daily amplitude of air temperatures and the degree of nebulosity of the sky, on a scale of 1 to 10, were also compared to infarcts as a percent of the total. They concluded that no clear correlation exists between infarcts and atmospheric pressure, wind velocity, mean daily air temperature, and daily amplitude of air temperatures. On the other hand, they state that the peak occurrence of infarcts coincided with high relative air humidity and the maximum degree of cloud cover.[1] Inasmuch as relative humidity is a function of temperature and

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[1]No statistical tests for correlation, or comparing observed and expected frequency were done.

vapor pressure, it would be interesting to see this data re-examined from that point of view, that is, the occurrence of infarctions and varying values of temperature and vapor pressure. Along the same line of thought, the term nebulousness must also be dissected. One feels compelled to ask the question, "Why clouds?" To say that infarcts are associated with cloud cover is to say, in fact, that they are associated with a process or processes under which condensation takes place; it is these processes that one should examine. It is not difficult to conceive of a circumstance in which one would find both high relative humidity and heavy cloud cover, that is, an advection of warm moist air into an area of colder air would cause the relative humidity to rise and condensation in the form of clouds take place as the warm air rises above the cool.

Category 3

A much more exhaustive study than any of the preceding is that done by Hofer and Mueller (33). In their study not only do they explore individual weather elements such as barometric pressure, temperature, relative humidity, vapor pressure, wind velocity, degree of cloud cover, precipitation, thunderstorms, lightning, and also the earth's magnetism and sun spot activity in relation to the occurrence of infarcts, but also weather in combination; for example, they explore the possibility of the influence of warm fronts, cold fronts, and occluded fronts; and furthermore, they not only look at individual air mass types, but also air masses under broader headings and, lastly, the gross weather types. For their examination of most of these phenomena they use the chi-square statistic; for example, they compared the number of observed infarcts with the number expected during the days in which there was cold front activity. However, for the individual weather elements they used the Newman-Keuls test. In all statistical tests run, a competence coefficient of 99 percent was considered significant, while values between 95 and 99 percent were considered questionably significant. Using these criteria, the only statistically significant result that could be reported was that of the drop in the daily mean temperature and daily mean vapor pressure two days after a day on which one or more infarcts occurred. The connection is not readily apparent; the authors agree. They suggest three possible explanations: (1) that there is indeed a direct connection between the decline in temperature and vapor pressure and infarcts, (2) behind this coincidence of myocardial infarctions and decline in these two elements, there is a common cause, which is unknown, affecting both, or (3) this coincidence is merely an accident, that is a mistake of probability. In support of the latter they inform us that in a parallel study in the same period in Berlin the same results were not obtained, even though these two areas, Berlin and Greifswald, are not that different meteorologically speaking. It is possible that the two cities have a somewhat different microclimate, however, such data is not available.

Elsewhere in Germany, researchers have reported a connection between the onset of infarcts and weather fronts. In this category are Fieber (20) and Brezowsky and Diesfeld (8). Fieber, in Liepzig, investigated 583 cases of sudden heart disease. By use of the n-method,[1] a marked relationship with the passage of warm fronts and occlusions was shown. In addition, he also reports that there was a maximum of bright chromosphere eruptions two to three days prior to the onset of illness.

Brezowsky and Diesfeld base their analysis of 217 heart infarcts that occurred at the City Hospital of Ansbach during the years 1957 to 1962, on a scheme of weather periods that was developed by Ungeheuer at the medical meteorological advice station of the German Meteorological Service at Bad Tolz. This weather scheme divides the conditions of the atmosphere in this part of West Germany into phases such as one would find in an area that experiences the passage of mid-latitude cyclones.

Phase 1 - cool to mild and dry

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[1]Also known as the synchronous method, n days are the day of the event (in this case, infarcts). The days before and after are designated as n-1, n+1 and so forth. The occurrence of some meteorological condition, e.g., frontal activity is noted, in relation to the event, under the appropriate n column. In this way clustering can be seen.

Phase 2 - mild to warm and dry

Phase 3 - mild to warm and extremely dry[1]

Phase 4 - mild to warm and increasingly moist

Phase 5 - cool to cold and moist

Phase 6_z - cold and moist (delayed weather stabilization - "durch active Kaltluft verzögerte Wetterberuhigung")

Phase 6 - cold to cool and dry

Of these weather phases, 1, 2 and 6 are considered biologically favorable, weather 3 is considered somewhat biologically favorable, and weather phases 4, 5 and 6_z are considered biologically unfavorable. The number of infarcts that occurred during each one of these weather phases can be seen in the following table.

Wetterphase	1	2	3_A	4	5	6_z	6	Summe
Wetterphase	157	153	235	785	91	374	396	2191
Infarkte je Wetterphase	8	7	41	99	13	43	6	217

It can be seen from this table, and as also reported in the article, that there is strong relationship between the time of infarcts and weather phase 4 with some implication for weather phases 3_A and 6_z. In support of these observations,

[1]Weather phase 3 is subdivided into 3_A and 3_F. The former indicates air mass subsidence, the latter, foehn conditions.

the author quotes other studies, for example, one done by Brezowsky (7) earlier involving 70 cases in North Bavaria and the foreland of the Alps, and another done by Beleke (4) in Hamburg involving 69 cases of which 85 percent were found to occur on the front side of cyclones. On the other hand Brezowsky notes that Kutschera (41) reporting on 134 cases in Vienna found no connection with the passage of cyclones. Brezowsky posits that this may be due to the lesser number and intensity of fronts found in Vienna compared to Middle Europe. When one considers the continental position of Vienna, it would appear that this supposition is correct. Austria lies south of the main cyclonic track that crosses the Baltic Sea area of Europe, and north of the Mediterranean cyclonic track. Furthermore, during the months of October to March, the air becomes colder and more dense from west to east in Europe. This dense air precludes the incursion into eastern Austria of many of the depressions that affect northwest Europe at that time.

In their study of 113 patients in Sophia, Bulgaria, Nikolaev and Marinov (52) also employed the terminology favorable and unfavorable in regard to the weather. They included frontal activity, foehn conditions and unstable air masses in the unfavorable category and for all other types of weather, the term favorable is used. In the statistical analysis of their data these authors employed Pierson's criterion chi-square in the so-called method "with yes and with no" and also the biotropic index.[1] The tests were in agreement that for three of the four years there was a statistically significant relationship between the occurrence of infarcts and unfavorable weather. In the remaining year in which this could not be shown the authors state that there were a very small number of cases that year which could account for this lack of agreement.

In one of the more recent studies in the United States, Teng and Heyer (65) further analyzed the data that was the basis of the 1953 study reported by Heyer, Teng and Barris (32). They concluded that there is a statistically significant increase in infarcts during sudden changes in temperature, particularly in relation to influxes of polar air, and also during continued hot weather. This analysis began with a division of all types of weather in Dallas into five categories: (1) stable, (2) precipitation without a marked change in temperature, (3) sudden onset of warm weather, (4) sudden onset of cold weather, and (5) continued hot weather. They then found the number of days that each type occurred, the number of MI patients admitted during that time, and then the number of patients per day. Next they computed the probability of the differences in incidence occurring by chance. In regard to the weather data, they figured the average daily fluctuation of temperature during the study period. This figure was then compared to drops in temperature over a fifteen to forty-eight

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[1]This index is described in Chapter III, page 42.

hour period for polar inflows, and over a period of up to sixty hours for tropical inflows. They go on to say that they found an increase in infarcts during the 12 to 48 hour period after the onset of polar inflow. Such a statement presents two difficulties: (1) their weather data were plotted at six-hour intervals, and (2) they would have to know within 12 hours when the infarct occurred. To put it another way, an inflow of polar air could begin at 3 a.m., but it would not show up in their data until say 6 a.m., at which time they would start counting their 12 hour period which would then take them to 6 p.m. the same day. They merely state that they recorded the time of onset of infarction, but do not elaborate. They feel that the role of barometric pressure per se is impossible to know because pressure and temperature vary inversely together. Thus, while acknowledging that people are often in a place of controlled temperature, they tend to feel that temperature change is a significant factor in the onset of myocardial infarction.

Certainly diversity is a feature of the research reviewed above. Diversity as to number of cases studied, methods of analysis, and explanation of what the analysis shows. But more than this, one cannot help but be struck by the almost complete lack of dissidence in regard to the basic premise that there is a connection between weather and the onset of myocardial infarction. It is this all but complete unanimity that makes the study of Lewitus and Neumann (44) stand out in bold relief. These researchers began their study of 396 cases by examining the data to see if the cases occurred at random. Their calculations did indeed show that the infarct cases under consideration follow a Poisson distribution. They further tested seasonal means using the hypothesis that the data for the cold and warm seasons were taken from some population and that the mean for this population is μ and that the sampling error of the mean is $\sigma/\sqrt{N}$, in which N is the number of members in the sample. The results showed that the seasonal means differ by less than one standard error from the hypothetical population mean. Therefore, there is no reason to reject the hypothesis. Consequently they concluded that not only were the infarcts distributed in a random fashion, but also that the attacks occur independently from each other.

Against this background of research findings, the following study was undertaken. The null hypothesis tested is that there is no relationship between weather and the onset of acute myocardial infarction. A significance level of 5% was used throughout.

CHAPTER III

MYOCARDIAL INFARCTIONS AND WEATHER: A STUDY OF 261 CASES FROM AKRON, OHIO, AND 218 CASES FROM OKLAHOMA CITY, OKLAHOMA, FOR THE YEAR 1965

Material

This study differs from those read in that two cities - Akron, Ohio, and Oklahoma City, Oklahoma - located in different climatic areas were selected. Since it was desirable to obtain the largest number of cases possible in the time allowed for the reading of records, Akron City Hospital, (582 beds), and St. Anthony Hospital (500 beds) and Baptist Hospital (389 beds) in Oklahoma City were selected. Even though the number of beds in the two Oklahoma Hospitals was much greater, the number of infarct patients was relatively smaller than that at Akron; consequently, in order to have a comparable number of cases in each city it was necessary to read the records at both Oklahoma City hospitals. The records for the year 1965 were read for this study.[1]

The choice of which cases to include in the study presented some problems. From reading the literature and from

[1] At the time of the selection the writer was living near Akron, just previously having resided near Oklahoma City. The records for the four years, 1964-67, were considered and all were complete. The year 1965 was selected at random.

conversations with physicians, it became apparent that the diagnosis of myocardial infarction is made after the consideration of a number of criteria. Such criteria are (1) a clinical history suggestive of infarctions, (2) electrocardiographic evidence, and (3) laboratory evidence. In the case of death, sometimes confirmation is obtained through autopsy. Since geographers have little knowledge of the foregoing, reliance was placed upon the diagnosis of the attending physician to a large extent. As a result, all cases in which the primary final diagnosis was that of myocardial infarction were accepted without question. However, a problem arose in those cases in which the physician had entered as the primary final diagnosis something such as "possible myocardial infarction," or "coronary occlusion." In regard to such records, the case was accepted into the study if autopsy confirmation of recent infarct was available, or careful reading of the entire record presented a picture, in regard to the criteria listed above, compatible with that of infarction. In those cases where it was obviously not clear, detailed notes were taken of all pertinent data and shown to Dr. Andrew Kerr, Jr., cardiologist and Chairman of the Department of Medicine, at Akron City Hospital. His judgment as to whether or not the patient had had an infarct was final. The next step was to ascertain in each case the day on which the infarction occurred. In the majority of cases, the onset of illness was so traumatic that the patient, or family of the patient, was able to give the time of onset as to day, and sometimes within an hour. On the other hand, there were a large number of cases in which patients with severe coronary artery disease experienced symptoms such as increasing shortness of breath and intermittent pain for days, weeks, or even months prior to their admission to the hospital. In such cases careful note was made of the time indicated, as shown in the patient's history, when such symptoms became much more pronounced, to the extent that the patient sought medical attention. In such cases one finds such wording as "Last night the pain was much worse (searing, crushing)." Or, in the case of shortness of breath the patient would use different wording to indicate that the condition had become so severe that it could not be ignored and indeed apprehension had set in. Noting this problem, Billings, et al. (5) made the following observation:

> In reviewing the records it was frequently difficult to determine exactly when the acute attack began. Often there was a description of repeated, severe episodes of substernal pain followed within a few hours or days by a more severe but otherwise similar episode accompanied by the symptoms, signs and lab findings of myocardial infarction. We believe that these premonitory symptoms were those of coronary artery failure without permanent damage to cardiac muscle, and that when the blood supply to the myocardium became so deficient as to result in death of muscle the typical picture of myocardial infarction developed.

The meteorological data were obtained on punch cards from the National Weather Records Center at Asheville, North Carolina. These cards provided data from Akron-Canton Airport and Will Rogers World Airport, Oklahoma

City, for observations at three-hour intervals of the amount and kind of precipitation, the barometric pressure, dew point, wind speed and direction, dry bulb and wet bulb readings and relative humidity. Since the vapor pressure was not available on punch cards, and yet was needed in the study, the computer center at Kent State University was asked to write a program that would provide these values by utilizing the dry bulb and relative humidity values indicated on the cards, in conjunction with the Saturation Vapor Pressure over Water Table in the Smithsonian Meteorological Tables.

Method

In order to transform the large amount of meteorological data available into a more easily manageable and meaningful form, use was made of the computer facilities at Kent State University. Subsequently, and in addition, graphic and statistical techniques were utilized in the analysis of the data.

From the information obtained at the hospitals, the number of cases per day was plotted for both cities (Figures 1 and 2). Such data represent the occurrence of isolated events in a continuum of time. The question arises as to whether the observed distribution of the events is by chance. In order to resolve this question, the Poisson Distribution was used. For Akron the following figures were obtained:

FIGURE 1: The Daily Incidence of Myocardial Infarction at Akron City Hospital During 1965.

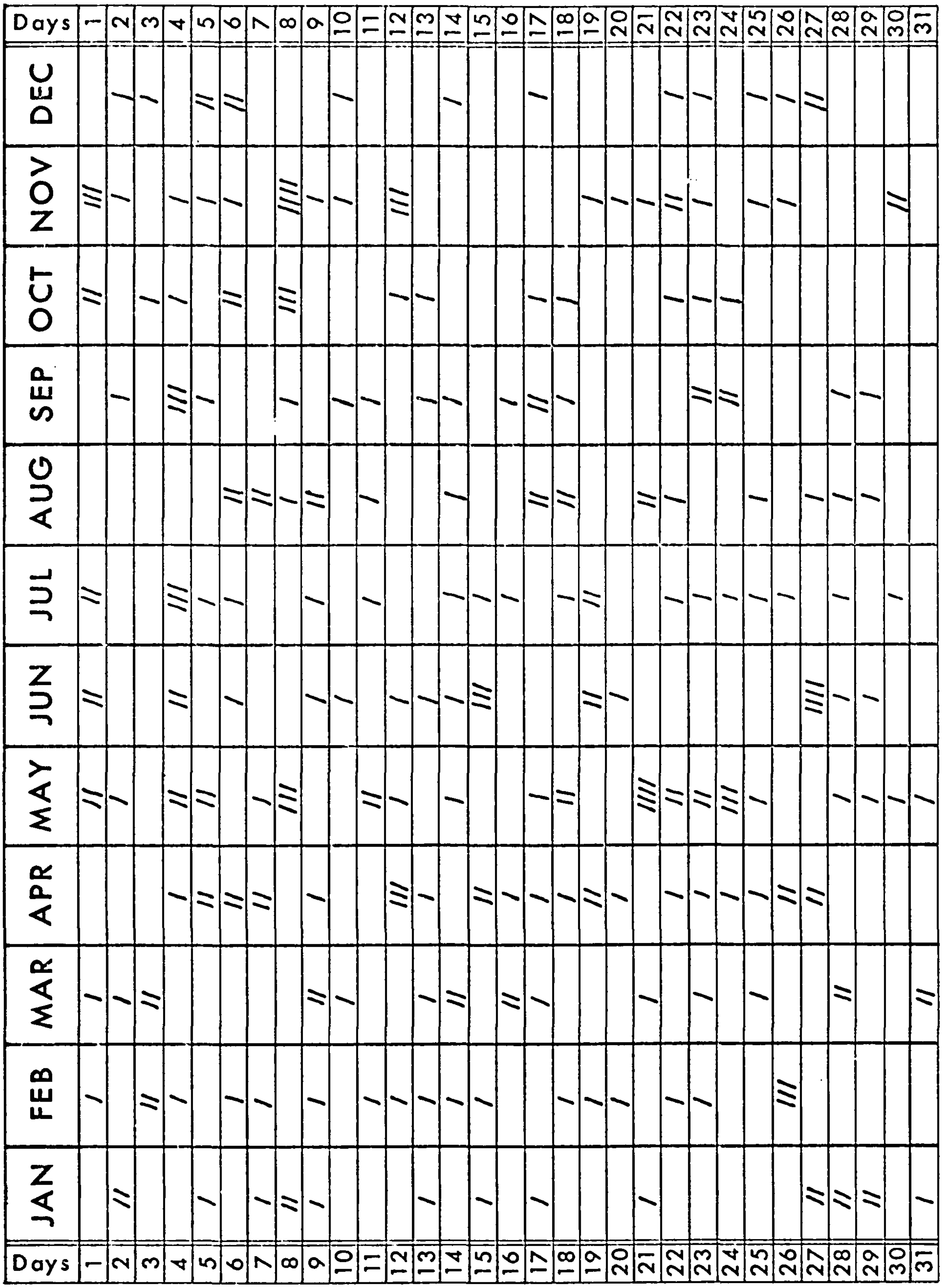

FIGURE 2 : The Daily Incidence of Myocardial Infarction at Baptist Memorial and St. Anthony Hospitals During 1965.

Days	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31
DEC	//		/		///	/	/	/	/	/		/	/	//			/	//		/				/			/				
NOV							/	/			/			/					/			/	//	//	///	/					
OCT	//	//								//			/		//	/			/		/		/		//	/	/				/
SEP			/		//		/	/		/		//	/					//			//	/		/	/			/	/	/	
AUG		/			//		/		//		//	//	/				/			//	/		//	/	/		/	/			
JUL		/		/		/		//				////	/	/				/	/	/	/	/	/	/						/	////
JUN		/	/	/		/		/					//		/		/			/	//	//		/		//	/			/	
MAY	/							//		//				//		/		/	/	/			/		/	/			/		//
APR			/			/		/	/	/					/					/	//	/		/		/	/				
MAR	///			//		/	/		/	/		/	//	/			//				/	/		/	//			/			
FEB			/	//					//				/		/		/		/	//					/						
JAN		/				//		/		/	/	//		/				///		/	/	//			/	/	/	/	/		
Days	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31

$N = 365$ days $\chi^2 = 1.857$

$\sum X = 261$ infarcts df = 2

$X^2 = 451$ $0.50 > P > 0.30$

$M_X = .715$

$S_X{}^2 = .734$

For Oklahoma City:

$N = 365$ days $\chi^2 = 2.742$

$\sum X = 218$ infarcts df = 2

$X^2 = 347$ $0.30 > P > 0.20$

$M_X = .597$

$S_X{}^2 = .594$

Therefore, the difference between the observed distribution and the Poisson Distribution for both cities can be regarded as a likely chance occurrence. However, the fact that the Poisson fit the data does not prove that the infarcts were distributed throughout the year by chance, but rather that the facts do not refute the hypothesis of chance distribution. Also, it had been noticed when making the graphs showing the number of attacks per day (Figures 1 and 2) that the infarctions appeared to occur in runs. Therefore a run test was performed on the Akron data to test whether this was just an impression or not.[1] It was found

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[1]After plotting the daily incidence of any disease (in this case, the incidence of infarcts) one frequently finds clusters of high and low figures, called runs, which make a strong visual impression, but which may be entirely due to chance. The test used to analyze the distribution is known as the run test, which consists of the following:

that the T value equalled 13.3. Therefore, less than a 0.1% probability exists that the run distribution would be due to chance only. The same test was not performed on the Oklahoma City data because they were insufficient. Due to the ambiguity created by the contradictory findings of the Poisson and the run test, and because the overwhelming impression that one derives from reading the literature is that the possibility does exist of a relationship between extreme weather and the incidence of infarction, it was

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The number of times that each value is observed, i.e., total daily infarcts, is noted. In this example, values of 0-4 only were observed, with the following frequencies 0(180X), 1(125X), 2(47X), 3(10X), and 4(3X). A line is then drawn through this series of values so that approximately the same number of occurrences can be found below and above this line; in this case, the line lies between 0 and 1. Looking at the figures observed during the first nine days of January, 0 2 00 1 0 121, it can be seen that there are three positive (m_1) and three negative (m_2) runs.

The following formula is used to determine whether the runs differ significantly from a chance distribution:

$$T = \frac{R_o = R_e}{\sigma}$$

if R_o = observed number of runs, R_e = expected number of runs, σ = standard deviation.

R_e is calculated with the formula:

$$R_e = \frac{2m_1m_2}{n} + 1$$

in which $n = m_1 + m_2$.

is determined by means of the formula:

$$\sigma = \sqrt{\frac{2\, m_1m_2(2m_1m_2 - n)}{n^2(n - 1)}}$$

If $T \geqq 2$, there is a 50% probability that the run distribution is due to chance only. If $T \geqq 2.6$, there is a 1% probability and if $T \geqq 3.2$, there is only 0.1% probability (66).

decided to proceed with an extensive analysis of the meteorological data.

Utilizing the printouts of the weather data obtained from the computer center, graphs were made for each month for each city, showing daily high and low barometric pressure and dry bulb temperature readings (Figure 3). In this way not only the daily range for these two elements, but also the direction and magnitude of day-to-day changes could be seen. The passage of fronts was also noted on these graphs. Information in regard to frontal activity was obtained from the daily weather maps printed by the United States Department of Commerce, Weather Bureau[1] which show surface weather conditions at 1 a.m. and 1 p.m. Infarcts were also shown graphically. Using the data thus depicted, relationships were sought statistically between the incidence of infarcts and frontal passage, temperature, and/or barometric pressure.

The wind-chill index was obtained from ESSA (Figure 4). This index was then compared with the printed weather data after the wind speeds of the latter (given in knots per hour) were converted to miles per hour. Each day in the colder half of the year, that is October to April, was classified as to whether it was very cold (VC), extremely cold (EC), bitterly cold (BC), (or none of these). These data were then compared to the incidence of infarctions.

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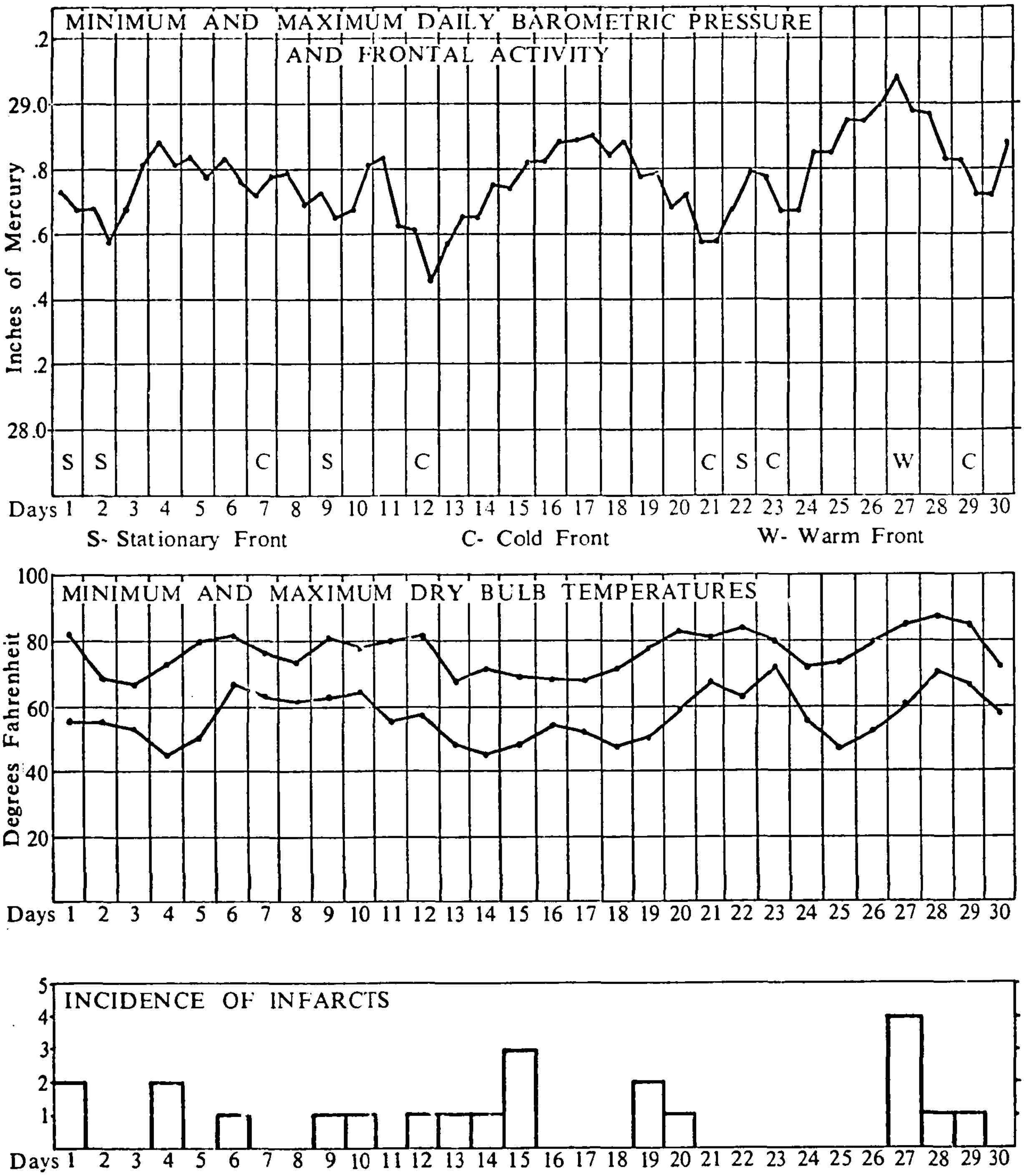
[1]Now known as the Environmental Science Services Administration (ESSA).

FIGURE 3: The Maximum and Minimum Daily Barometric Pressure and Dry Bulb Temperatures, Frontal Activity, and Incidence of Infarcts for June, in Akron.



°F Dry-bulb Temperature

WIND CHILL INDEX (EQUIVALENT TEMPERATURE)
Equivalent in cooling power on exposed flesh under calm conditions

MPH	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45
Calm	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45
5	33	27	21	16	12	7	1	-6	-11	-15	-20	-26	-31	-35	-41	-47	-54
10	21	16	9	2	-2	-9	-15	-22	-27	-31	-38	-45	-52	-58	-64	-70	-77
15	16	11	1	-6	-11	-18	-25	-33	-40	-45	-51	-60	-65	-70	-78	-85	-90
20	12	3	-4	-9	-17	-24	-32	-40	-46	-52	-60	-68	-76	-81	-88	-96	-103
25	7	0	-7	-15	-22	-29	-37	-45	-52	-58	-67	-75	-83	-89	-96	-104	-112
30	5	-2	-11	-18	-26	-33	-41	-49	-56	-63	-70	-78	-87	-94	-101	-109	-117
35	3	-4	-13	-20	-27	-35	-43	-52	-60	-67	-72	-83	-90	-98	-105	-113	-123
40	1	-4	-15	-22	-29	-36	-45	-54	-62	-69	-76	-87	-94	-101	-107	-116	-128
45	1	-6	-17	-24	-31	-38	-46	-54	-63	-70	-78	-87	-98	-101	-108	-118	-128
50	0	-7	-17	-24	-31	-38	-47	-56	-63	-70	-79	-88	-96	-103	-110	-120	-128

VERY COLD

BITTERLY COLD

EXTREME COLD

Wind speeds greater than 40 mph have little additional chilling effect.

FIGURE 4: The Wind Chill Table Used by ESSA.

The possible effect of another extreme environmental condition, that of high temperature and relative humidity, was also explored by using the temperature humidity index (THI). The following formula for obtaining the THI was given to the Kent State University computer center:

$$THI = T_{db} - (0.55 - 0.55\ RH)\ (T_{db}\ 58),$$

where T_{db} is the dry bulb temperature, and RH is the relative humidity. The center, in turn, furnished printouts showing THI's for every three hours and also the daily average THI. The latter were graphed for the months of May to September for Akron. Also shown on these graphs were the daily maximum and minimum wind speeds, since high wind speeds somewhat mitigate the discomfort associated with high THI values (Figure 5). Once again infarcts were shown at the bottom of the graph. Inasmuch as after this was done, no apparent relationship could be seen, (and the use of daily means tends to obscure somewhat the actual picture) the Oklahoma City data was not similarly graphed.

Daily extremes of temperature were also investigated by themselves, that is, not in conjunction with any other element such as wind speed or relative humidity. For each city the higher and lower temperatures experienced were divided into categories of 5^{o} each.[1] The number of days on which the highest or lowest temperature fell into one of

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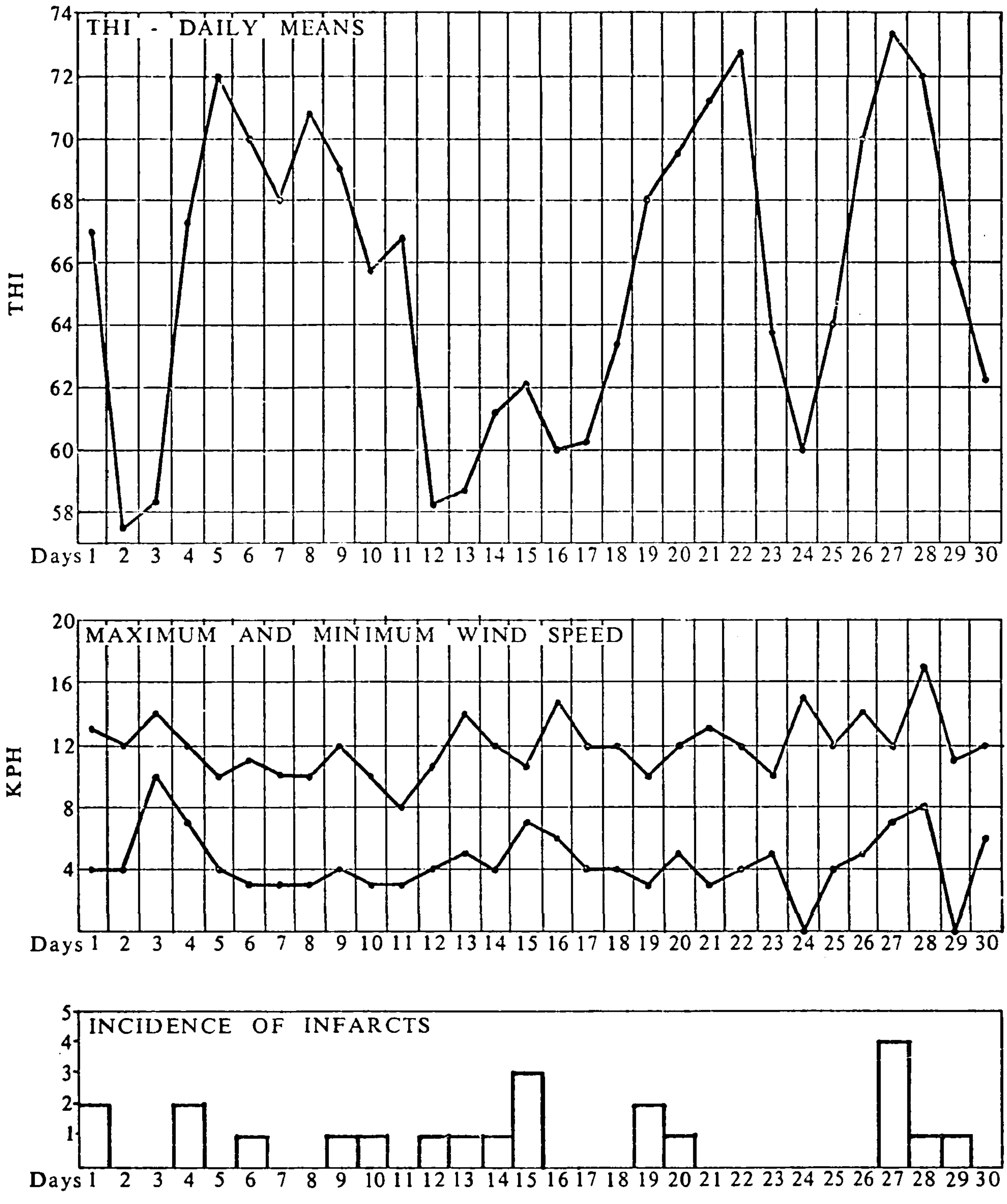
[1]Throughout this study temperatures will be given in ^{o}F., barometric and vapor pressure in inches Hg.

FIGURE 5: Daily Average THI, Maximum and Minimum Wind Speeds, and the Incidence of Infarcts in June, 1965, in Akron.



these categories was counted as was the number of infarctions that occurred on those days. The observed frequency of infarctions was then put into a table with the expected frequency[1] and a chi-square test performed (Tables 3 and 4, pages 47 and 48).

The computer center provided a printout showing the daily, monthly, and yearly mean values for wind direction and speed, barometric pressure, dry bulb temperature, relative humidity and vapor pressure. In addition, those days on which the temperature, vapor pressure or barometric pressure was 2 or more standard deviations away from the annual mean were shown. Also provided were the mean daily range of temperature, vapor pressure, and barometric pressure plus the standard deviations, and those days on which the range was 2 or more standard deviations away from the mean.

Finally, a frequency table of infarcts was constructed for each month for each city and the mean and standard deviation computed. All weather data were studied closely, for all days on which the incidence of infarctions was 2 or more standard deviations away from the mean. No meteorological patterns were discerned.

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[1]Throughout this study expected frequencies were calculated by dividing the total number of infarcts by 365 on the hypothesis that, if there were no variation with the weather, then infarcts would be expected to occur with equal frequency, i.e., on any one day 1/365 of the total infarctions.

Findings

Wind and Infarctions

As was pointed out in Chapter I, wind may be considered either desirable or undesirable depending on the air temperature. Utilizing the wind chill index obtained from ESSA, wind speed in relation to low temperatures was first compared to the incidence of infarcts in Akron using De Rudder's Index of Meteorotropism (M) (66).

$$M = \frac{N \times K_n}{n \times K_N} = M = \frac{365 \times 55}{86 \times 261} = \frac{20.075}{22,446} = .894$$

in which

N = the total number of days observed,

n = the number of days with a specific meteorological disturbance (in this case a combination of strong winds and low temperatures),

K_N = the total number of observed meteorotropic events (here, infarctions) and

K_n = the number of the same meteorotropic events observed during the number (n) of meteorologically disturbed days.

When there is pronounced meteorotropism, according to De Rudder, the ratio of M is approximately 2. In those cases, as here, when M is smaller than 1, a negative biotropic action exists; that is, the particular disease, in this case infarction, rarely occurs during the weather condition under consideration. For Oklahoma City an M value of .908 was obtained.

The conclusions drawn from the above analysis were confirmed as follows (Tables 1 and 2):

TABLE 1

STATISTICAL COMPARISON OF THE OCCURRENCE OF CASES OF ACUTE MYOCARDIAL INFARCTION IN THE THREE WIND CHILL CATEGORIES AKRON, 1965

	Number of Days	Cases Observed	Cases Expected
VC	57	13	40.8
BC	19	11	13.6
EC	10	11	7.0

$\chi^2 = 3.35$
Degrees of freedom = 2
$0.20 > P > 0.10$

TABLE 2

STATISTICAL COMPARISON OF THE OCCURRENCE OF CASES OF ACUTE MYOCARDIAL INFARCTION IN THE THREE WIND CHILL CATEGORIES OKLAHOMA CITY, 1965

	Number of Days	Cases Observed	Cases Expected
VC	37	22	22.00
BC	20	10	11.90
EC	2	0	1.19

$\chi^2 = 1.49$
Degrees of freedom = 2
$0.40 > P > 0.30$

Relative Humidity and Myocardial Infarction

Since relative humidity is a function of temperature and vapor pressure, that is, the amount of moisture in the air compared to how much moisture a parcel of air at a given temperature can hold, expressed as a percentage, scant attention was paid to relative humidity alone in this study. In conjunction with high ambient temperatures, high relative humidity values produce warm humid conditions which are considered unpleasant by most people. To measure the degree of this type of discomfort ESSA uses the THI with the degree of discomfort broken down into the following three categories: when the THI is 70 some people will be uncomfortable, at 75 more than half will, and at 79 everybody will feel discomfort. It was hypothesized that perhaps a high THI would be sufficient to push a person across the morbidity limit.[1] In 1965, in Akron, there were 32 days on which the THI was greater than 70; the highest value was reached on July 24 when it equalled 74.38 with a dry bulb reading of 92° and 58 percent relative humidity. During these 32 days there were 30 infarctions; consequently, using the Index of Meteorotropism, one finds that in this instance

$$M = \frac{365 \times 30}{32 \times 261} = \frac{10,950}{8,352} = 1.32.$$

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[1]The concept of morbidity limit was introduced by Tromp (66) to indicate, in his words "the specific physiological condition of a healthy person, which leads to disease if this particular labile condition is surpassed." In other words a person with a pre-morbid condition may reach the diseased state as a result of external meteorological stimuli; that is, a meteorological event or events may act as a triggering force.

It was found in this study that THI values greater than 70 were much more prevalent in Oklahoma City than in Akron in 1965. Therefore, whereas 70 was used as the cutoff point for Akron, 75 was used in Oklahoma City. There were 36 days in Oklahoma City in 1965 on which the THI value was 75 or more. During those 36 days 27 infarctions occurred. The highest THI reading occurred on July 23 with a dry bulb reading of 101° and a relative humidity value of 27 percent. Comparing this with Akron one can readily see that the relative humidity reading is lower, but the elevated temperature compensates for this so that the same degree of discomfort is reached. For Oklahoma City then, the figures are as follows:

$$M = \frac{365 \times 27}{36 \times 218} = \frac{9,855}{7,484} = 1.13.$$

Therefore, it would appear that pronounced meteorotropism does not exist in these two cities in regard to infarctions during hot, humid weather.

Vapor Pressure and Myocardial Infarctions

In Akron there were 8 days with a daily mean vapor pressure reading 2 or more standard deviations away from the annual mean of 0.300. During those 8 days there were only 4 infarcts, less than one would expect by chance. On 18 days the daily range was 2 or more standard deviations away from the annual mean range of 0.1115. In every case, the range was greater than the mean. The 21 infarcts that took place was above the expected number of 12.9, yet the resulting M value is only 1.63.

In Oklahoma City, there was no day on which the daily mean was 2 or more standard deviations away from the annual mean of 0.395. On the other hand there were 13 days, with a total of 10 infarctions, on which the daily range was 2 or more standard deviations away from the annual mean range of 0.123. The expected number of infarcts, 7.76, is not notably less than the number observed.

Temperature and Myocardial Infarctions

The temperature data for both cities were analyzed in several ways in order to check for associations between various temperature conditions and the onset of acute myocardial infarction. As previously mentioned, the higher and lower temperatures that occurred in 1965 in the two cities under consideration were divided into several categories. The number of infarcts that took place when the maximum or minimum daily temperature fell into one of the several categories[1] was counted. The number of observed cases was then compared to the number expected if the total number were evenly distributed throughout the year. In this way the figures in Table 3 were derived for Akron.

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[1]For example, on January 15, in Akron the maximum temperature was 18° and the minimum 6°. This day was placed in the 15° or less category.

TABLE 3

FREQUENCY DISTRIBUTION OF CASES OF ACUTE MYOCARDIAL INFARCTION AT DIFFERENT RANGES OF HIGH AND LOW TEMPERATURES IN AKRON

Temp. °F.	Number of Days	Cases Observed	Cases Expected
76-80	38	31	27.1
81-85	36	38	25.7
86 and above	15	18	10.6
26-30	42	27	30.0
21-25	19	3	10.8
16-20	15	6	10.7
15 or less	28	22	20.0

χ^2 (for the higher temperature) = 11.43
Degrees of freedom = 2
$P < 0.005$

χ^2 (for the lower temperatures) = 8.19
Degrees of freedom = 3
$0.05 > P > 0.02$

Hence, it can be seen that there is a relationship in Akron between extremes of temperature and incidence of infarctions. This is particularly true in the ranges of 81-85°, 86° and above, 16-20°, and 21-25°. It should be borne in mind, however, that such a relationship does not necessarily mean cause. If extremes of temperature per se act as a triggering force in regard to infarctions, one would expect to find this consistently true; that this is not the case can be seen by the figures for Oklahoma City given in Table 4.

TABLE 4

FREQUENCY DISTRIBUTION OF CASES OF ACUTE MYOCARDIAL INFARCTION AT DIFFERENT RANGES OF HIGH AND LOW TEMPERATURES IN OKLAHOMA CITY

Temp. °F.	Number of Days	Cases Observed	Cases Expected
85-89	35	20	20.8
90-94	33	26	19.7
95 and above	34	22	20.2
36-40	20	16	11.9
31-35	18	11	10.7
26-30	24	15	14.3
25 or less	18	9	10.7

χ^2 (for the higher temperatures) = 2.20
Degrees of freedom = 2
$0.30 > P > 0.20$

χ^2 (for the lower temperatures) = 1.61
Degrees of freedom = 3
$0.50 > P > 0.40$

Another way to view the temperature data in a search for relationships is to look at day-to-day temperature changes. Accordingly, all days were counted on which the maximum temperature was 10° more or less than the previous day. Then the number of infarctions was counted that occurred on the days involved. This method furnished the data in Table 5. A glance at this table readily shows that the incidence of infarctions does not increase greatly compared to the number expected, with changes in temperature of the magnitude shown. In fact, in Oklahoma City there appears to be an inverse relationship between temperature increase and rate of infarction. This trend continues to be seen when the increase in temperature is upped to 15°. Conversely, in Akron there is a slight tendency for infarcts to increase with drops in temperature. This trend also continues with a 15° temperature drop.

TABLE 5

EFFECT OF TEMPERATURE CHANGE ON INCIDENCE OF MYOCARDIAL INFARCTION

Max. Temp. 10° Higher Than Previous Day			Max. Temp. 10° Lower Than Previous Day		
No. of Days	MI Observed	MI Expected	No. of Days	MI Observed	MI Expected
Akron					
72	52	51.5	92	70	65.8
Oklahoma City					
72	30	43	78	45	46.6

It should be mentioned at this point that there is an inherent source of fallacy in the type of analysis just discussed; that is, the exact time of infarction is usually not known. Therefore, a 15° increase in temperature may take place, for example, from 6 p.m. one day to 10 a.m. the next day with three infarcts on day one. Yet, unknown to the researcher, all three occurred before 6 p.m.! Although not discussed in their article, this appears to throw some doubt on the results reported by Teng and Heyer (65).

The computer center also provided a printout of those days on which the daily mean temperature was 2 or more standard deviations away from the annual mean. A glance at these data showed that no more infarctions occurred on such days than one would expect by chance.

Yet another way of looking at temperature data is to consider the daily range of temperature, that is, the number of degrees between the highest and lowest temperatures from midnight to midnight. In the case of Akron the mean daily range was 16° with a standard deviation of 7.16°. There were 5 days during which the range was 2 or more standard deviations away from the mean, and the number of infarcts, 4, which took place on those days is not much greater than the expected number of 3.6.

In Oklahoma the mean daily range was 19° with a standard deviation of 7.02°. All days on which the temperature range was 2 or more standard deviations away from the mean were recorded. There were 15 such days and only 5 infarctions, compared to the expected number of 8.96 during any 15 day period. The computed M value is .558. This figure suggests that infarctions rarely occur on days with unusual temperature ranges.

Barometric Pressure and Infarctions

The possible role that barometric pressure might play in triggering infarctions was explored in some of the same ways as temperature. As in the case of temperature, it appeared evident that no more infarctions than one might expect by chance happened on those days on which the mean daily barometric pressure was 2 or more standard deviations away from the annual mean.

In regard to the daily range of barometric pressure, there were 23 days with a total of 19 infarctions, in Akron, on which the range was 2 or more standard deviations away from the mean daily range of 0.172. For Oklahoma City the figures were 20 days, with 10 infarcts, which were 2 or more standard deviations away from the mean daily range of 0.159. Therefore, for Akron the M value is 1.16, while for Oklahoma City negative meteorotropism might exist, for in 1965, M = .837.

When considering barometric pressure, two important differences between the occurrence of extremes of pressure and of temperature (in the cities under discussion) should be noted. First of all, pressure lacks the seasonality of temperature in that one does not find low pressure at a certain time of the year and high pressure at another as one does with temperature,[1] but rather the highest and lowest daily values

[1]Although in 1965 (and probably other years as well) in Oklahoma the months of April to September inclusive had

of pressure are found at the same time of the year.[1] Thus, if one desired to use the term season in regard to barometric pressure, it would have to be on the basis of the amplitude of the diurnal and interdiurnal values of pressure. These ranges are noticeably less in Akron from May through September, and in Oklahoma City from April through October. This feature of barometric pressure is particularly noticeable during July and August in Oklahoma City. Also, unlike temperature, the maximum and minimum values of barometric pressure for any given day may both fall into the high or both fall into the low categories shown in Table 6.[2] Therefore, in obtaining the following data only one reading, the most extreme, was taken per day in order to prevent double counting. As can be seen, in both cities, the difference between the observed number of infarcts and the number expected is not large enough to warrant statistical testing.

Weather Fronts and Myocardial Infarctions

In Table 7 is shown the total number of all types of

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monthly means less than the annual mean of 28.63. In Akron, the months of January to April inclusive, and also August, October, and December had monthly means less than the annual mean of 28.72 in 1965.

[1]In Oklahoma City in 1965, the highest pressure reading was 29.28 on January 16, and the lowest 27.98 on February 28.

[2]For example, on January 16 in Oklahoma City, the lowest pressure reading was 29.08, and the highest, 29.18.

TABLE 6

FREQUENCY DISTRIBUTION OF CASES OF ACUTE MYOCARDIAL INFARCTION AT DIFFERENT RANGES OF HIGH AND LOW BAROMETRIC PRESSURE

Inches Hg.	Number of Days	Cases Observed		Cases Expected
		Akron		
29.20-29.29	2	1	6	9.2
29.10-29.19	11	5		
29.00-29.09	24	17		17.1
28.31-28.40	11	11		7.8
28.21-28.30	5	3		
28.11-28.20	8	8	12	13.5
28.10 or less	6	1		
		Oklahoma City		
29.10 or more	3	0	10	9.5
29.00-29.09	13	10		
28.30-28.21	15	11		8.9
28.20 or less	12	10		7.1

fronts, warm, cold, occluded and stationary, and the number of infarcts that occurred during each month. It can be seen from these figures that there is no apparent relationship between the total number of fronts per month and the total number of infarcts; for example, the largest number of infarctions happened in May in Akron, and yet the corresponding number of fronts was not exceptionally large. Similarly, in Oklahoma City in June only one front passed the city and yet during that month there were 19 infarctions. A breakdown of the total number of fronts shows that for Akron there were 32 (28)[1] warm fronts, 57 (35) cold fronts, 21 (17) stationary fronts, and 18 (14) occluded fronts. For Oklahoma City the figures were 7 (8) warm, 57 (39) cold, 24 (15) stationary, and 7 (2) occluded.

TABLE 7

TOTAL NUMBER OF FRONTS AND INFARCTIONS PER MONTH IN AKRON AND OKLAHOMA CITY IN 1965

	Akron		Oklahoma City	
	Fronts	Infarctions	Fronts	Infarctions
Jan.	14	18	11	21
Feb.	14	20	5	14
March	7	20	9	18
April	11	28	9	13
May	11	34	6	17
June	10	22	1	19
July	9	22	14	23
Aug.	12	20	8	21
Sept.	11	20	7	19
Oct.	8	16	10	18
Nov.	12	26	8	14
Dec.	9	15	6	21
Total	128	261	94	218

Employing De Rudder's Index once again, the figures in Table 8 were obtained. The conclusion which may be drawn from this analysis is that frontal activity does not

[1]The figures in parenthesis denote associated infarctions.

significantly influence the incidence of infarctions since there is no index greater than 2. Yet, when the number of infarcts that occurred on the calendar day following frontal passage were counted, a different picture emerged. In Akron there was an increased frequency of infarcts following cold fronts (M = 2.20), while in Oklahoma City an increase was noted following warm fronts (M = 2.15).

TABLE 8

FRONTAL ACTIVITY AND INDICES OF METEOROTROPISM

All Fronts	Warm	Cold
Akron		
1.04	1.22	.859
Oklahoma City		
1.15	1.91	1.14

Discussion

There appears to be no way of reconciling the apparent contradiction indicated by the results of the Poisson and the run test. On one hand, the former leads one to believe that the events, i.e., infarctions, occur independently of each other while the latter strongly indicates statistically significant clustering of the events. Perhaps it is within the area of doubt left by the Poisson findings that the reported data fall. If this is true, one should be able to find confirmation in a detailed analysis of the data.

Wind

Such confirmation was not found in relation to wind speed, and most assuredly not with the wind-chill data. Indeed, in the latter there appears to be a negative relationship.

Water Vapor

The amount of water vapor in the air also does not appear to be a factor in the onset of infarctions. This was shown to be true by an examination of both the daily mean and the daily range of vapor pressure values. Relative humidity readings combined with high dry bulb temperatures, as measured by the THI, also bore no relation to the occurrence of infarctions. Thus it would seem that the discomfort experienced by most people when the THI is 75 or above, affects the potential infarct victim no differently.

Barometric Pressure

As was the case with vapor pressure, neither extremes of barometric pressure, nor deviations from the mean range produced an increase in infarctions. In fact, in both Akron and Oklahoma City the month in which the greatest number of infarcts occurred, May and July respectively, falls in that time of year when both daily and day-to-day ranges are slight.

Temperature

Some relationship between temperature and infarctions appears more probable on the basis of these data, than any of the weather conditions discussed above. Although no relationship was seen in regard to significant deviations from the mean daily range or in day-to-day change in maximum temperatures, extremes of temperature were related to an increased incidence in Akron.[1] That this could not also be demonstrated in Oklahoma City leads one to believe that, not extremes of temperature per se, but the difference in the activities of the people in the two cities during extremes of temperature may be the important factor.

It is commonly believed by many physicians and laymen that some infarctions occur while the victim is shoveling snow. While this is undoubtedly true, inspection of the precipitation data for Akron in 1965 showed no relationship between snow cover and the number of infarcts. In regard to winter sports, a check of ice skating and ski areas in the Akron area showed that the number of participants in the 40 and over age bracket is relatively small. On the other hand, the pace of everyday activity, shopping, meetings and

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[1]A contradiction does not necessarily exist between this fact and the THI analysis presented previously. A check of the temperature and THI data showed that, whereas all THI's of 70 or more were in conjunction with temperatures of 80° or more, the reverse is not true. Also, it probably would be correct to say that "unusual" physical activity is less common on hot days which are also humid.

so forth, slows little if at all. In Oklahoma City not only are the number of cold days few, but also there is a definite tendency for people in that city to avoid going out in cold weather.[1]

A similar explanation about the antithetical results of the chi-square tests in regard to higher temperatures seems credible. In Akron, by the time that the days grow longer and temperatures of 80° or more can be expected (May), there exists a pent-up urge to get outside. So that the lives of many people in summer are characterized by a flurry of activity, yardwork, sports, picnics, visiting, house repairs and painting and so forth. When temperatures go above 85°, however, enthusiasm wanes a bit; home and car air conditioning are not as common as in Oklahoma City.

In Oklahoma City, due to the fact that the colder half of the year is much milder than in Akron, outdoor activity is spread out over a longer period of time. Furthermore, at temperatures above 95° little yardwork is done. This is due to a combination of factors. The large amount of solar radiation received results in both elevated temperatures and evaporation rates, and a concomitant decrease in the effectiveness of precipitation. As a result, lawns turn brown and plants grow slowly unless watered, and water

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[1]The author has personally experienced nearly empty shopping centers and cancelled meetings during cold weather, particularly when snow was involved.

is expensive enough so that only the more affluent apply water to their yards. Therefore, yard work falls to a minimum during the hottest time of the year. On a hot summer day in Akron, beaches and parks are crowded, in Oklahoma City, almost deserted, (although crowds, of mostly younger people, throng municipal pools). The general impression one receives living in Oklahoma City is that at temperatures of 95°+, it is largely only young people who are active outdoors, the remainder of the population preferring to stay in their air-conditioned homes. As the high temperatures in Table 4 show, only in the 90-94° bracket is there a difference of more than 3 cases between the number of infarcts observed and expected, while this is true of all three brackets in Akron.

Frontal Activity

Brezowsky's (8) description of an undeniable connection between the warm, wet milieu of the biosphere and infarcts could not be corroborated. This seems to be most assuredly true in Akron, and probably also in Oklahoma City. The few warm fronts that did pass Oklahoma City in 1965 appear to have a high number of infarcts associated with them, but these same fronts were not associated with unusually high vapor pressure readings.

These data do not show, for Oklahoma City, the positive relationship between infarcts and the passage of cold fronts that Heyer and Teng (32) found in Dallas. The difference in number of infarcts relative to cold fronts between Akron and Oklahoma City is interesting. In Oklahoma City, no relationship can be seen, while in Akron the incidence of infarcts jumps the day following the passage of a cold front. This phenomenon does not seem to be related to changes in barometric pressure, vapor pressure, or temperature. In regard to the latter, it should be pointed that, while a drop in temperature is usually associated with the passage of cold fronts, this is not invariably the case. It would be even more erroneous to equate cold fronts with the colder part of the year, hence, to cold temperatures. In Akron in 1965, as many cold fronts (6) passed the city in July as in January.

Therefore, there appears to be two possible explanations for the apparent relationship between infarctions and the aftermath of cold fronts in Akron: (1) the coincidence is a mistake of probability; (2) behind the coincidence is a common, but unknown, cause.

The present study has shown no definite relationship between weather and the onset of myocardial infarctions. Therefore, the null hypothesis is accepted.

CHAPTER IV

CONCLUSIONS

It is clear that the body reacts to external stimuli such as changes in atmospheric conditions. The reaction of the body to changes in external temperature is well-known compared to reaction to other weather elements. Even in those areas where knowledge is more complete, the picture is obscured by the fact that each person is unique; hence bodily response is not precisely predictable. However, whatever the response, the cardiovascular system is involved.

This fact has led many researchers to investigate the possibility of a connection between weather and the onset of acute myocardial infarction. The several studies were grouped into three broad categories. The results of these studies have run the gamut of possibilities. As a result, no clear picture has emerged as to whether or not weather plays a role in precipitating infarctions, and if it does, which element or elements are involved and in what way.

Due to the doubt thus engendered, the fact that the concept of critical level of climatic stress is so intriguing, and the extent of CAD in this country, it was felt that yet another study would be worthwhile.

An analysis of the relationship between weather elements, singly and in combination, and the onset of acute myocardial infarction was made using 261 cases from Akron and 218 cases from Oklahoma City. The primary basis for the choice of these two cities was their location in different climatic regions.

No increase in the frequency of infarctions was noted in relation to wind, vapor pressure, barometric pressure, or the THI. An increased frequency was noted in relation to extremes of temperature and to the passage of cold fronts, but only in Akron. Since these results could not be duplicated in Oklahoma City, serious doubt exists as to the role of these factors in precipitating infarctions.

If a critical level of climatic stress exists for the potential infarct victim, it was not discovered in this study. The null hypothesis was accepted.

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