CHAPTER 2

INDICATIONS OF STRESS FROM BONE AND TEETH

Alan H. Goodman[1]

Department of Anthropology
University of Massachusetts-Amherst

Debra L. Martin

School of Natural Science
Hampshire College

George J. Armelagos
George Clark

Department of Anthropology
University of Massachusetts-Amherst

INTRODUCTION: MODELING DISEASE IN PREHISTORIC POPULATION

The purpose of this chapter is to review current uses of skeletal and dental evidence[2] in reconstructing patterns of health in prehistoric human populations. This brief overview may be supplemented by reference to a variety of recent publications. For more detailed discussions of the diagnosis of disease in prehistoric populations, the reader should consult volumes by Ortner and Putschar (1981) and Steinbock (1976). Information on nutritional and physiological stress can be obtained from Buikstra and Cook (1980), Huss-Ashmore et al. (1982), Wing and Brown (1980), and Mielke and Gilbert (1984).

[1]*Present address: Department of Orthodontics, University of Connecticut Health Center, Farmington, Connecticut 06032.*

[2]*See Allison et al. (Chapter 20 this volume) for soft tissue analysis.*

ISBN 0-12-179080-0

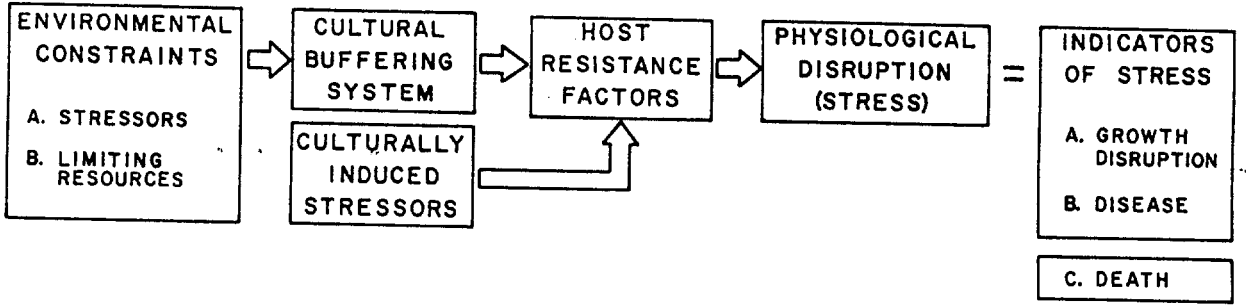

FIGURE 2.1 Model for interpretation of stress indicators in paleoepidemiological research.

This overview is organized around a model of the causes and results of physiological disruption or stress (see Figure 2.1). This perspective has emerged out of a desire to answer processual questions. The severity, duration, and periodicity of a disease may be as important to understanding biocultural process as the identity of the particular pathogenic agent. In theory, if two different diseases are equal in the severity and duration of physiological disruption that they cause, the impact on the individual and the population should be equal.

Stress is a product of three sets of factors which are represented schematically in Figure 2.1: environmental constraints, cultural systems, and host resistance. Environmental constraints include both limiting resources and stressors. These may vary over time and space. If uncorrected, these constraints will result in increased physiological disruption. Cultural systems may act to buffer the impact of environmental constraints. For example, our technological society has effectively buffered cold stress by the invention and use of central heating systems. However, cultural systems may also magnify existing stresses or produce novel ones. For example, while intensification of subsistence systems may allow for greater population density, subsistence intensification may result in a lower quality of diet for each person and amplify interpersonal strife as access to quality nutrients is limited.

If stress is not adequately buffered by extraindividual means, its effects may be buffered only by individual host resistance. Host resistance varies by age and sex. Genetic factors also play an important role in resistance to certain diseases; however, these are difficult to trace in the archaeological record.

When host resistance and environmental constraints are held constant, variation in stress levels may be related to cultural differences. The primary purpose of this volume is to determine changes in health associated with culture change where environmental and host resistance factors are presumed constant.

Physiological disruption cannot be directly measured in skeletal remains. However, stress does leave a series of indicators in bone and teeth. A primary goal of paleopathology is to 'read' these indicators of stress.

In this chapter indicators of stress are organized under three major headings: (1) indicators of general, cumulative stress; (2) indicators of general, episodic stress; and (3) indicators of stress associated with specific diseases. These groupings are somewhat arbitrary; it could be argued that there is a continuum of indicators from most to least general and most to least episodic. Other grouping schemes are also possible but may not be as useful in understanding underlying similarities in the causes of stress. General indicators are based on the organism's nonspecific response to noxious stimuli (Selye 1950, 1971). Cumulative indicators provide a summation of the amount of stress over long periods of time while episodic indicators are more precise and confined in the time at which stress occurred.

General, cumulative indicators include mortality measures and growth assessments. Under stressful conditions the growing organism will either slow growth or cease to grow since its available nutrients are better put to combating the stress (Acheson 1960). Since stress generally results in the release of catabolic hormones (those which release energy) the anabolic process of growth is inhibited (Cannon 1932; Tanner 1978). Therefore, indicators of growth may be indicative of stress of a general nature.

Periodic indicators of stress provide information on the age at which stress episodes occurred. Two common examples of general, periodic indicators are Harris lines (disruptions in linear bone growth) and enamel hypoplasias (disruption in tooth enamel matrix formation).

Finally, some diseases or disease classes may leave more specific indications of stress on bone and teeth. This is true for trauma and degenerative pathologies, and for some infectious diseases and nutritional deficiencies.

To summarize the model, stress is a result of environmental constraints, cultural filters, and host resistance factors. While stress is not directly measurable in skeletal remains, it may be inferred from a series of indicators. When host resistance and environmental constraints are held relatively constant, variation in stress may be attributed to cultural differences. Increasing levels of stress may also act as a cause of changes in cultural patterns. An ultimate goal of paleopathology is to understand the process by which cultures may both cause and respond to stress.

GENERAL AND CUMULATIVE STRESS INDICATORS

Mortality

Mortality data for skeletal populations derive from assessment of individual ages at death (see Ubelaker 1978 for an overview of methods of skeletal age determination). Traditional presentations of mortality data involve either the direct estimation of life expectancy at birth (based on the mean age at death) or the construction of life tables. In addition to estimating life expectancy at birth, life tables provide estimations of life expectancy, probability of dying, and survivorship for all age classes (see Swedlund and Armelagos 1976: Appendix A for method of computation).

Two types of criticisms have been voiced against measurements of mortality and specifically against the use of life tables in paleodemography. The first concerns the appropriate method of presentation and use of mortality data from archaeological populations. Angel (1969) argues that life tables are too sophisti-

cated a tool for limited available paleodemographic data. However, where population sizes are sufficiently large and appear to be well represented, life tables may provide a series of meaningful and valid statistics (Acsadi and Nemeskéri 1970; Moore et al. 1975; Swedlund and Armelagos 1969).

More fundamental criticisms of life tables concern the various assumptions which are inherent in the method. The construction of these tables assumes that a skeletal sample is truly representative of a real population; that no population growth is occurring; that there is little stochastic fluctuation in the population's size; and that individual ages-at-death are accurately determined. Moore et al. (1975) have determined that undernumeration of infants, the principal source of skewing in archaeological samples, has little effect on mortality estimates except in the youngest age classes. Stochastic fluctuation may introduce errors in very small populations. However, this error may be estimated. Similarly, growth or decline in population size introduces error. However, the error is small under conditions close to equilibrium. These same authors provide a reminder that mortality statistics are probability statements and should be regarded as having ranges of reliability (Moore et al. 1975). Under normal conditions the estimated errors in paleodemographic data are small. Finally, it has recently been proposed that paleodemographic analyses are seriously flawed due to inaccuracy in age determination (Bocquet-Appel and Masset 1982). However, Van Gerven and Armelagos (1983) have argued that serially aging the skeletal series reduces this error.

Part of Van Gerven and Armelagos's argument in favor of the use of life tables includes an assessment of the validity of life table data in specific contexts. Life table analysis has frequently led to interpretable and meaningful conclusions in paleodemographic analyses. For example, Green et al. (1974) show a decrease in life expectancy in Sudanese Nubians who are buried without superstructures and in those who died while their village was in decline.

We believe that the major limitations of the paleodemographic method are practical ones. These include the representativeness and size of the sample and the ability to provide accurate assessment of developmental age (Lovejoy et al. 1977). Age at death stands as perhaps the most important single indicator of stress. Age at death is of additional importance as other stress indicators are related to it. If other stress indicators are associated with decreased ages at death, then this supports their validity as indicators of stress.

Growth Assessment

Growth assessment is a common tool for analysis of the degree of environmental stress in prehistoric populations. Assessments may include the construction of growth curves based on measured

length and width of long bones for each age category of subadults; the measure of attained length and width from adult long bones; the estimation of stature derived from adult long bone lengths; and the determination of sexual dimorphism derived from male-female differences in selected anthropometric measures.

These indicators are based on the theoretical proposition that the slowing or cessation of growth is a logical response by an organism to increased stress (Cannon 1932). However, all growth comparisons must consider the importance of genetic factors affecting size and shape. When genetic variables are controlled, the experimental and clinical literature strongly suggests that delayed or decreased growth may be reflective of physiological disruption (Acheson and Fowler 1964; Dickerson and McCance 1961; Engfeldt and Hjertquist 1961; Himes 1978; Johnston 1976; McCance and Widdowson 1962; Platt and McCance 1964; Stewart 1975; Tanner 1977, 1978).

Subadult Long Bone Length and Width Curves

The analysis of growth in subadult long bones is based on the fact that dental development is less affected by stressful conditions than skeletal growth (Garn et al. 1959, 1965; Lewis and Garn 1960). Provided that a large population of subadults is available for study with intact long bones and dental ages, long bone growth may be plotted against dental age.

Such growth curves have been constructed by Sundick (1978) and Ubelaker (1978), among others, for archaeological populations. The growth curves of prehistoric populations generally differ from modern standards in two principal ways. The first is a reduced rate of growth between the ages of approximately 2 and 5 years and the second is a delay in the timing of the adolescent growth spurt. The decrease in rate of growth at ages 2-5 relative to modern standards (Maresh 1955) may be indicative of undernutrition or other stress acting on that segment of the population. For example, Cook (1976) interprets such a disease in growth rate in a prehistoric population from Illinois to be due to a poor weanling diet. The delay in the adolescent growth spurt may be the result of chronic undernutrition or other chronic stresses. Frisancho and Garn (1970) attribute such a growth delay in Quechua Indians living on the Peruvian altiplano to chronically low nutrient availability. The view that this delay is adaptive in the face of limited calories is supported by Thomas (1973), who demonstrates that the delay in growth significantly reduces the energy needs of the population.

While comparison of prehistoric growth curves with modern standards may be illustrative, Buikstra and Cook (1980) warn against overinterpretation. Prehistoric data are not strictly comparable to modern data; age estimates for prehistoric individuals are based on developmental criteria (tooth eruption) whereas modern individuals are usually aged by the calendar.

Moreover, prehistoric data are cross-sectional (different individuals dead in different age classes) rather than longitudinal (the same individuals progressing from age class to age class).

In light of these limitations, perhaps the most valid use of prehistoric growth curves involves comparisons of curves from genetically similar populations. An example of this approach is Lallo's (1973) study of long bone growth curves from Dickson Mounds populations (also see Goodman et al., Chapter 11 this volume). Lallo is able to demonstrate a statistically significant secular decrease in mean tibial length for individuals of ages 5-10 when comparing the Dickson Middle Mississippian to earlier Dickson populations.

Similarly to the above, long bone width, circumference, and cortical thickness may be plotted against dental age. Lallo (1973) has also demonstrated a secular decrease in tibial circumference relative to age which parallels the length decrease mentioned above (also see Goodman et al., Chapter 11 this volume).

Huss-Ashmore (1981) in an analysis of juveniles from prehistoric Sudanese Nubia has shown that long bone growth in length may be maintained at the expense of cortical thickness (also see Garn et al. 1964). Thin cortices in growing children are a clear indication of stress, but in a sense may also represent an adaptive response. Decreased bone mass in growing children permits continued growth of bones in length as well as the liberation of minerals and nutrients to aid in the maintenance of soft tissue systems in which nutrients are most required. Comparison of long bone widths and length curves may provide a hierarchy of growth responses to stress (Huss-Ashmore et al. 1982). Width is first affected. But if stress is severe and long lasting, then length increase may also slow or stop.

Adult Long Bone Length and Width

The measurement of size and shape of adult long bones is among the most standard of procedures in anthropometric and skeletal analysis. Many researchers have provided data on adult long bone width and length in prehistoric samples and have derived stature estimates from these data. (See Ubelaker 1978, for stature formulas.) Comparison across populations again introduces problems in interpretation as it is difficult to estimate the degree of genetic involvement in size and shape differences. Size and shape differences in adult skeletons may be more easily related to environmental conditions and physiological disruption if there is relative genetic homogeneity in samples. The relative genetic continuity of successive samples provides the basis for the interpretation of recent secular increases in height within a country or defined geographic area (Acheson and Fowler 1964; Craig 1963). These secular increases have most frequently been interpreted as being due to a decrease in infant-childhood disease and elimination of nutritional stresses (Dobos 1965:77-79; Huber 1967). Similar

interpretations may be applied to similar patterns in prehistory if genetic continuity can be postulated or proved. See, for example, Larsen (1982 and Chapter 14 this volume), who demonstrates a decrease in adult long-bone length in successive populations on the Georgia coast.

While long bone length yields information on group adaptation, analysis of long bone thickness, width, and histological structure can reveal patterns of metabolic activity and physiological disruption in adults. Because cortical bone is in a constant state of remodeling by resorption and deposition, introduction of any stress which seriously affects metabolism may alter the rate of remodeling (Stout and Simmons 1979). When rates of bone remodeling are in disequilibrium, bone can be lost instead of maintained. Conditions resulting in osteoporosis (bone loss) include metabolic disturbances, systemic disease, and nutritional stress (Garn 1970; Huss-Ashmore et al. 1982; Martin and Armelagos 1979; Ortner 1976).

Cross sections of bone shafts can be analyzed by calculating the percentage of the cortical area (bone thickness versus bone marrow cavity) and plotting this by age and sex (Garn 1970). Thin sections can also be made and viewed under a bright field microscope to reveal its microstructure (Martin et al. 1984). These measures assess the amount and quality of the cortical bone present and reflect the nutritional and health status of the individual. Huss-Ashmore and co-workers (1982) have demonstrated the usefulness of cortical bone analysis in the assessment of the quality and quantity of diet. Age-controlled samples from temporally sequential populations have been used to equate bone loss and increase remodeling activity with nutritional stress (Martin 1983; Richman et al., 1979; Stout 1979).

Sexual Dimorphism

Though common, analyses of the degree of sexual dimorphism are difficult to interpret for archaeological populations. In theory, a decrease in sexual dimorphism should be indicative of increased stress since the growing male is more susceptible to stress than the growing female (see Stini 1969, 1972, 1975). However, analysis of sexual dimorphism is confounded by potential genetic variation in the degree of dimorphism among populations and the likelihood that males are more protected from stress in many societies. Furthermore, in archaeological analysis the same traits which are used to determine sex are often used to access the degree of dimorphism, thus engendering circularity.

The standard method for assessment of sexual dimorphism involves the determination of male to female ratios for stature or for various measures of skeletal width and robusticity. More detailed multivariate analyses of sexual dimorphism have been performed by Van Gerven (1972) based on femoral measures and by Gustav (1972) based on pelvic measures.

While comparison of the degree of sexual dimorphism both among and within populations may prove to be useful, at present the results are difficult to interpret. For example, Larson (1982, Chapter 14 this volume) argues that an *increase* in sexual dimorphism is associated with increased stress. This results from the increased physical work load placed on females, which decreases their growth.

Other Cumulative Indicators of Stress

Other skeletal indicators may prove to be useful in providing data on cumulative levels of stress in archaeological populations. Angel (1978, 1982, and Chapter 3 this volume) argues that skull base height and pelvic inlet form are indicative of growth efficiency and nutritional stress. Angel may be correct in this assessment. However, his arguments should be supported by experimental data which at present are sparse. And, as with other growth assessment, the degree of genetic control must be considered.

Dental crowding should be indicative of nutritional or other chronic, severe stress since teeth will be less affected by chronic stress than alveolar bone size. Widdowson and McCance (1964) have demonstrated this effect in undernourished piglets and Trowell and co-workers (1954) have noted increasing crowding and impacted molars in severely malnourished children. Increased dental crowding may be indicative of severe and chronic stress in archaeological populations. However, we are unaware of the use of this potential indicator in any evaluation of health in prehistory.

Guargliardo (1982) has recently presented an argument that tooth size variation within a skeletal population may be indicative of environmental stress. This proposition is based on his observation that decreased tooth size is often associated with a mean earlier age at death. Individuals who are most stressed die earlier and their teeth fail to grow to their genetically determined potential size.

Fluctuating or random asymmetry refers to size differences in a bilateral organism in which one side is larger than the other, but in which no consistent pattern occurs (where, for example, some teeth on the right are larger and some smaller than their counterparts on the left; see Buikstra and Cook 1980, and Huss-Ashmore et al. 1982 for more detailed reviews of asymmetry). Asymmetries in long bone lengths and especially in dental crown measures have been used by a variety of authors as an index of stress in both living and prehistoric populations (cf. Bailit et al. 1970; DiBennardo and Bailit 1978; Niswander and Chung 1965; Perzigian 1977; and Perzigian et al., Chapter 13 this volume). Dental asymmetry is generally greater in populations which are under the greatest stress (Bailit et al. 1970). However, populations may also vary in the degree to which they are genetically predisposed to increased asymmetries (Niswander and Chung 1965). If asymmetries are due to stress, then

the critical period is likely to be in utero during the development of tooth buds (Bailit 1975). The postnatal environment may affect tooth size, causing asymmetries, but how this effect is produced is unclear. As noted, this condition frequently has been used as a measure of stress on a populational level. Its use likely will increase as the mechanisms by which asymmetries develop are better understood.

INDICATORS OF GENERAL AND EPISODIC STRESS

Harris Lines

Harris (1926, 1933) was one of the first researchers to study the mechanisms and causes of Harris lines. Harris lines, which consist of dense transverse lines visible in longitudinal sections or radiographs of longbones, are also referred to as "transverse trabeculae" (Platt and Stewart 1962), "transverse lines" (Park and Richter 1953), "radiographic transverse lines" (Harris 1933), and "lines and bands of increased radiopacity" (Garn et al. 1968). While the latter label is the most accurate description of the phenomena, we will refer to them as Harris lines or transverse lines for brevity and in agreement with the prevailing convention (Figure 2.2).

The mechanism by which Harris lines are formed involve a reduction in the thickness of the epiphyseal cartilage plate with increased resistance of the immature cartilage cells to capillary and osteoblastic penetration. Subsequently, a thin osteoblastic layer (primary stratum) is formed and replaces the more mature cartilage cells below the plate. Finally, horizontally oriented trabeculae are formed by the osteoblasts of the primary stratum (Garn et al. 1968; Park 1964; Park and Richter 1953; Steinbock 1976). Once formed, Harris lines may resorb and disappear. There is little agreement on how, when, and where resorption occurs. Hence, resorption introduces a potential source of error into analyses of Harris lines as indicators of past stressful experiences (cf. Garn et al. 1968; Park 1964).

There is little consensus about the cause of Harris lines (see Buikstra and Cook 1980; Huss-Ashmore et al. 1982, and Martin et al. in press for discussions on the meaning and use of Harris lines in paleoanthropology). The mechanisms of line formation require a period in which growth is arrested, followed by a period of growth recovery (Acheson 1959; Steinbock 1976). This process may take place within approximately one week (Steinbock 1976). Thus Harris lines should be indicative of stresses of approximately this duration. However, the clinical and experimental literature show only a weak association between known stressors and Harris line formation (see Marshall 1967). Published correlation

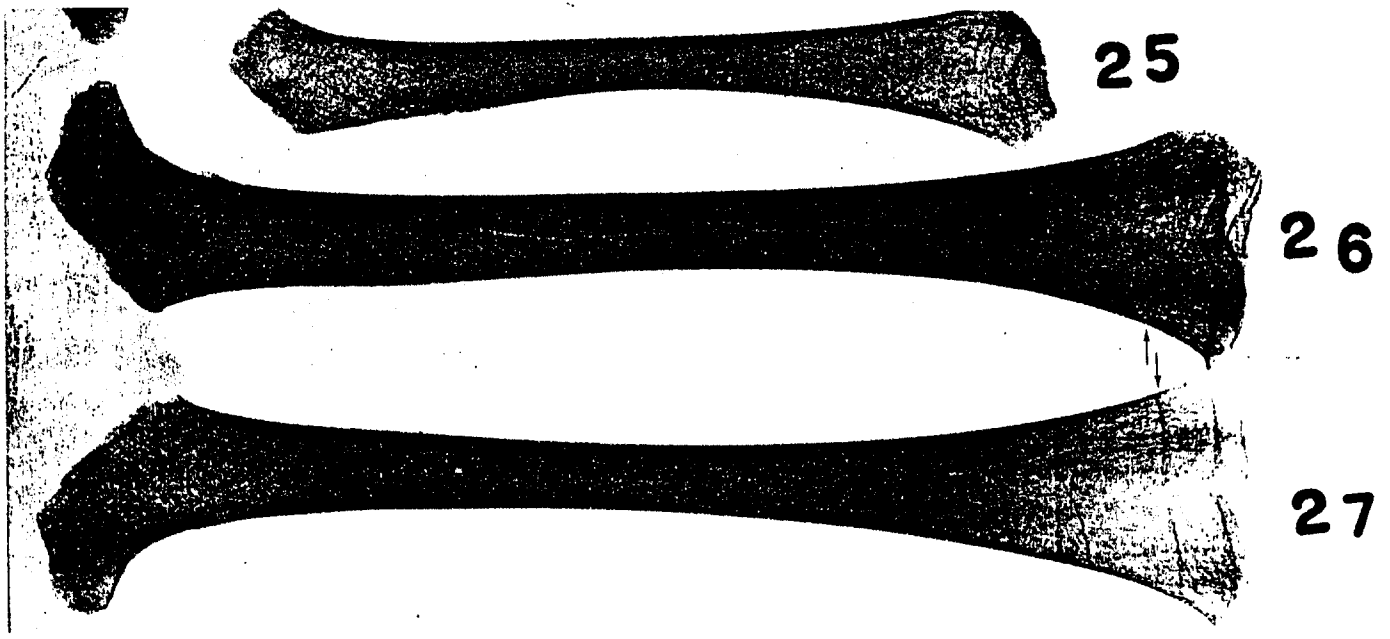

FIGURE 2.2 Harris lines in the distal femur of subadults from the Pueblo period at Black Mesa, Arizona (numbers are for identification).

coefficients for the associations between Harris lines and diseases range from .03 to .30 (Mensforth 1981).

Despite these low correlations, Harris line analyses have been common in paleopathology since Wells (1967) hailed the method as an exciting "new approach to paleopathology." The enthusiasm of Wells and others (Allison et al. 1974; Clarke 1978; Cook 1979; Goodman and Clark 1981; Gray 1967; McHenry 1968; and Woodall 1968) is likely to be a function of the ease of the method and the potential for gaining chronological information about episodic stresses. Harris line frequencies have been used to measure gender differences in stress (Wells 1967), interpopulation differences in stress (Goodman and Clark 1981), periodicity of stress (Cook 1979), and the chronology of stress during development (Clarke 1978). If Harris lines are a valid indicator of stress, then they are likely to indicate acute and episodic stress in contrast to the previously noted indicators of cumulative stress.

The standard method for analysis of Harris lines involves the production of radiographs of long bones in anterior-posterior position. Lines may be rated for thickness, degree of opacity, and amount that they traverse the diaphysis. However, we are unaware of a highly replicable method for scoring either the presence or absence or the quality of a line.

Chronologies of the time of development of lines have been developed based on the position of lines relative to the mid-shaft using established chronologies for the growth in length of long bones (see Goodman et al., Chapter 11 this volume). These chronologies can be adjusted for variations in growth as a function of age, sex, and body part but can not account for individual and population level variations in growth. Harris line analysis for adult long bones may provide a chronology of stress during the complete period of long bone growth. Analysis of subadult long bones may yield a chronology up to the age at death.

Interpretation of Harris line data must be undertaken with caution. Differences in frequency may be interpreted as a function of variation in degree of episodic stress. However, one must be aware of the low correlations between Harris lines and known stressors in the clinical and experimental literature as well as a series of inverse relationships between Harris lines and other stress indicators in paleopathological studies (Goodman and Clark 1981; McHenry and Schulz 1976). Similarly, Harris line chronologies may be useful in pointing to the distribution of stress over the growing years. However, interpretation of chronologies is also dependent on one's faith in the meaning of lines as well as a consideration of age-related host resistance and growth phenomena. Finally, evaluation of the frequency of Harris lines across studies is cautioned against because data concerning Harris lines are known to vary by observer, radiographic material and method employed, long bone observed, and age of sample (Martin et al. 1984). Harris lines are potentially an excellent source of data obtainable in no other way, but their interpretation should be supported by parallel analyses of other stress indicators.

FIGURE 2.3 Enamel hypoplasia of the maxillary central and lateral incisors in an adult from the Pueblo period at Black Mesa, Arizona.

Enamel Hypoplasias

Enamel hypoplasia (chronologic or linear hypoplasia or aplasia) is a deficiency in enamel thickness resulting from a cessation in amelogenesis (Sarnat and Schour 1941). Enamel hypoplasias are visible on tooth crown surfaces as lines, bands, or pits of decreased enamel thickness (Goodman et al. 1980; see Figure 2.3).

The mechanisms by which enamel hypoplasias develop is without controversy (Kreshover 1960; Osborn 1973; Rose et al. in press). Enamel matrix is formed by secretory ameloblasts. If these ameloblasts are disrupted to a degree that they lose their functional ability, then less matrix will be formed and the resulting enamel will be reduced. Since amelogenesis, the process of matrix formation, occurs along a coordinated front whose shape and timing have been determined, the age of development of hypoplasia may be deduced (Sarnat and Schour 1941). Finally, since enamel, once formed, is not resorbed or remodeled during life, it provides a permanent and unaltered chronologic memory of stress during its development.

The experimental basis for relating hypoplasias to periods of stress is a strong one. Hypoplasias have been associated with a wide variety of diseases and nutritional deficiencies (see Kreshover 1960; Pinborg 1982; Rose 1973; Rose et al. 1984).

With little variation, the associations with known stresses are high, with r^2 values around .5. Unexplained variation may be related to host resistance and methodological difficulties (Rose et al. 1984).

Hypoplasias have been examined for both deciduous and permanent dentition in prehistoric populations. The frequency of hypoplasias in the deciduous dentition is generally low, perhaps reflecting intrauterine protection (Blakey 1981; Sciulli 1977). The advantage of studying deciduous teeth is that they provide a record of stresses during their unique time of development--the first year of life and the last 5 months of prenatal development.

Enamel hypoplasias of the permanent dentition in prehistoric populations have been studied by a variety of authors in recent years (see Buikstra and Cook 1980; Huss-Ashmore et al. 1982; Rose et al. 1984). The usual method of analysis involves the recording of defects on a single tooth, usually the canine. Goodman and co-workers (1980) have shown that the canine may be a particularly good choice as it is highly susceptible to stress and has a long developmental period (Condon 1981; Rose et al. 1984).

Hypoplasia analysis includes a recording of the available enamel surface for observation and the position of hypoplasias on the enamel crown. Observation of hypoplasias may be aided by use of a binocular microscope. While hypoplasias differ in width, depth, and continuity (pits versus lines or bands), such characteristics are infrequently recorded. There is no standard definition of the minimum requirement for scoring a hypoplasia. The position of hypoplasias on the enamel crown may be recorded as distance from the cemento-enamel junction (in mm) and/or by estimation of the relative position of the defect on the crown.

An alternative to the single tooth analysis has been provided by Swärdstedt (1966), who studied all available permanent teeth except for the highly variable third molar. The two main advantages of this method are (1) an ability to check that the underlying stress is systemic rather than local in origin by showing that the same episode is recorded on different teeth, and (2) an extension of the chronology by use of teeth developing at slightly different times. The main disadvantage of this method is that it requires more time than the single tooth analysis. Goodman and co-workers (1980) proposed a compromise to the above with a "best teeth" analysis which includes the use of the maxillary central incisor and mandibular canine. In the Dickson Mounds study, 95% of systemic stresses were recorded on one or both of these teeth.

Hypoplasia frequencies have been used in archaeology to compare the frequency of stress by gender (Swärdstedt 1966), by status group (Cook 1981; Goodman et al. 1983), and by age class (Cook 1981; Goodman and Armelagos 1980; Swärdstedt 1966), as well as to compare populations (Cook 1976; Goodman et al. 1980). The comparison of age groups is of particular interest as authors have found that individuals with more hypoplasias die at an earlier age.

These data are evidence that hypoplasias record childhood events which are important to the survival of the individuals.

Hypoplasias have been used to reconstruct the chronological distribution of stress. Swärdstedt (1966) notes a peak period of stress around age two to four in a Swedish medieval population while Schulz and McHenry (1975) note a peak at age four to five in their California Amerindian sample. Goodman and co-workers (1980) noted that hypoplasias are more often separated by a year than by a half year. This is given as evidence of an annual cycle of stress.

In summary, enamel hypoplasias are a relatively valid and replicable indicators of infant-childhood stress. Further experimental work and more agreement on the minimum requirement for scoring defects would benefit interpretation. At present hypoplasias are a valuable method for evaluation of general stress occurring at early ages.

Enamel Microdefects

Enamel microdefects (pathological striae of Retzius, Wilson bands) are observable in longitudinal sections of teeth as a line or band running relatively perpendicular to enamel prisms which exhibits abnormal shape and prism bending (Rose et al., 1984) (Figure 2.4).

A microdefect is the result of a temporary disruption in amelogenesis. Matrix formation has a natural periodicity and under normal conditions will result in a series of incremental lines in enamel (Yaeger 1980). However, if the metabolism of the active, secretory ameloblasts is disrupted, then the matrix formed may be altered in thickness, prism direction, and protein content. The observable result of this disruption is a microdefect. Microdefects have been associated with a wide variety of disease and nutritional disturbances in clinical (Massler et al. 1941; Watson et al. 1964) and experimental studies (Rose and Pasley 1980). Though less research has been done on microdefects as compared to hypoplasias, the results are similar. It is likely that these two defects are different levels of analysis of the same disruption (Condon 1981). Hypoplasias may be associated with the severest grade of microstructural disruption.

Methods for analysis of microdefects include the preparation of undecalcified, longitudinal sections through tooth crowns. Recorded information is similar to that for hypoplasias (available enamel and location of defects relative to the cemento-enamel junction). A variety of operational definitions of microdefects have been employed (Rose et al. 1984) which vary mainly in their definition of the point at which a band is no longer considered to be due to normal processes. Rose's (1977) advocation of the term *Wilson bands* provides the most restricting definition of a microdefect. Wilson bands include a radical change in prism direction

FIGURE 2.4 Wilson band from a permanent canine (160×). Note the trough-like appearance of the band (Condon, 1981).

and abnormal prism structure. Condon (1981) and Rudney (1981), however, believe that a change in prism direction is sufficient to indicate systemic disruption.

In recent years a variety of studies have emerged on the appearance of microdefects in prehistoric populations (Clarke 1978; Condon 1981; Cook 1981; Jablonski 1981; Rose 1973, 1977, 1979; Rose et al. 1978; Rudney 1981). An exemplary series of studies of microdefects has been provided by Rose (1977) and co-workers (Rose et al. 1978). They demonstrate an increased frequency of Wilson bands in Dickson Mounds versus Gibson Mounds populations, a peak frequency of defects around 2 years of age, and an inverse relationship between Wilson bands and age at death.

Microdefect analysis is likely to increase in popularity in paleopathology. With a biologically meaningful and agreed upon minimum criteria for scoring a defect, microdefects provide a particularly sensitive indicator of stress. In comparison to hypoplasias, microdefects may record more disruption, including less severe and long-lasting ones. The major drawbacks of this method include the need to sacrifice teeth and the time and costs involved in the study.

Other Episodic Stress Indicators

Teeth provide a series of other episodic stress indicators which are less frequently used but may provide important information on stress. While enamel hypoplasias and microdefects are due to disruption in enamel matrix formation, enamel hypocalcifications are defects which result from a disruption in maturation of enamel (Yaeger 1980). These are visible as increased opacities in enamel and are often found in consort with hypoplastic defects (Blakey 1981). The common occurrence of hypoplasias and hypocalcifications is indicative of their being the result of the same metabolic disruption.

Dentin development is similar to enamel development. Dental matrix (predentine) is first formed and is quickly calcified (Avery 1980). Incremental lines (lines of von Ebner) are observable in dentin. Contour lines of Owen are accentuations of the normal incremental pattern and may be used to indicate metabolic stress (Avery 1980). Molnar and Ward (1975) have provided an overview of the potential application of dentin microdefects in anthropology. In theory, dentin should yield a set of indicators of stress which are as valid as enamel defects. However, dentin defects have been studied far less frequently, perhaps because there is no means of studying them without making histological thin sections.

INDICATORS OF SPECIFIC DISEASE STRESS

Porotic Hyperostosis

Porotic hyperostosis (lesions of the frontal, parietal, and occipital bones of the cranium) and cribra orbitalia (lesions on the superior border of the orbits) are manifest as a widening of the spongy diploe with a corresponding thinning of the outer dense cortical bone resulting in the appearance of surface porosity (see Figure 2.5). In severe cases, there is total obliteration of the bone surface with a lattice of trabecular overgrowth (see Figure 2.5). Mensforth and co-workers (1978) have presented the most thorough discussion of porotic hyperostosis in archeological populations. Steinbock (1976) provides information that is useful in the differentiation of the various factors which cause the lesion to be present in humans.

First described by Welcher in 1885, the condition has been attributed to a number of factors including thalassemia, hereditary anemias, sickle-cell anemia, and iron deficiency anemia (Moseley 1963). The alteration in skeletal tissue from these anemias is caused by the increase in red blood cell production which takes place in the marrow cavities of long bones and the diploe of flat

FIGURE 2.5 Porotic hyperostosis (cribra orbitalia) of the orbital surface.

bones. Because the cranial bones are so thin, they are often affected. As the diploe expands, the outer layer of bone becomes very thin and the inner trabecular bone is exposed. The thickened and porous bone has a sieve-like appearance.

In a landmark study, Hengen (1971) analyzed 459 human crania from various time periods and geographic areas and discussed the various possible explanations for the occurrence of porotic lesions. After casting out those explanations which did not fit most cases, he suggested that iron deficiency anemias fit most examples and that the lesion could be traced to the dietary habits. He further proposed that iron deficiency acted synergetically with infectious and parasitic diseases. Differential diagnosis could be made by examining the location of the lesion, its severity, and the age distribution of affected individuals. Numerous researchers have argued that porotic hyperostosis is due to nutritional stress in most cases in the New World since other explanations (malaria,

hemoglobin derived anemias) can not be applied to New World populations (El-Najjar et al. 1976; Lallo et al. 1977; Mensforth et al. 1978).

Carlson and co-workers (1974) furthered Hengen's hypothesis by suggesting that the interaction of cultural, environmental, and biological factors could account for the incidence of the pathology in prehistoric Sudanese Nubian populations. This study emphasized a consideration of the lesion with respect to age and found a higher incidence of lesions among infants and females of child-bearing ages. At about the same time El-Najjar and co-workers (1976) reached similar conclusions for two prehistoric Southwestern Amerindian populations. Both these studies suggest that porotic hyperostosis is a nonspecific pathology that reflects an anemic condition. High frequencies of infectious disease, a diet low in iron or one that inhibits iron absorption, and cultural factors such as weanling diarrhea, all increase the potential for porotic hyperostosis.

Studies conducted by Lallo and co-workers (1977) and Mensforth and co-workers (1978) expanded the previous research by examining the frequency and distribution of the lesions within specific age categories. Through the use of refined age categories, the clustering of lesions in younger children was shown to reflect the increased need for iron metabolism during periods of rapid growth and development. In addition, the evidence of healing of the lesion can provide important information on its impact on mortality. The evidence from remodeling (healing) suggests that the individual has survived the initial episode. Mensforth and co-workers (1978) were able to demonstrate that a large percentage of individuals did survive early stresses, while Huss-Ashmore and co-workers (1982) demonstrated that individuals with active porotic hyperostosis show a slight increase in mortality during early childhood.

The association of porotic hyperostosis and infectious diseases should also be considered. When both pathological conditions occur together in an individual, the infectious lesions are usually more severe (Lallo et al. 1977). Through the use of refined age categories and diagnostic distinctions of healed and unhealed lesions of both porotic hyperostosis and infectious reactions, Mensforth and co-workers (1978) were able to demonstrate that for the Libben population, infectious diseases were the initial pathological response which made individuals more susceptible to porotic hyperostosis.

Analyses of porotic hyperostosis have often been hindered by failure to consider the physical quality of the lesion (healed versus unhealed), failure to define precise and narrow categories, and failure to consider the synergistic relationship between host resistance, diet, and other variables such as infectious disease. However, when all factors are considered, and when the narrowest biologically meaningful age categories are used, porotic hyperostosis is a valuable marker of nutritional stress in skeletal populations.

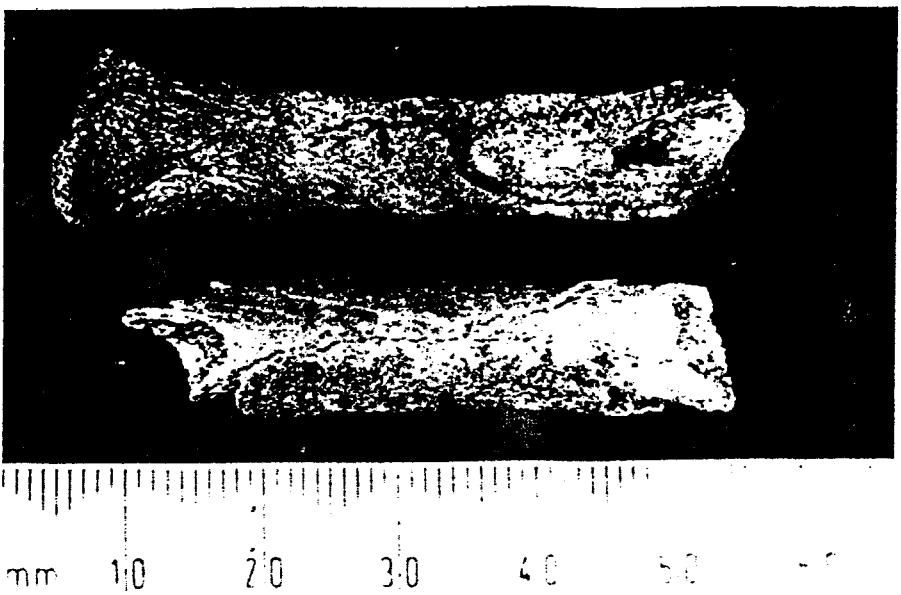

FIGURE 2.6 Periosteal reactions involving the long bone shafts.

Infectious Disease

Most examples of infectious disease in prehistoric skeletal remains are nonspecific; that is to say the lesions are caused by various kinds of microorganisms, but their exact etiology is unknown. Specific infectious diseases such as treponema (yaws/syphilis), tuberculosis, and leprosy, which can be differentially diagnosed, are much rarer (refer to Buikstra 1981; Ortner and Putschar 1981; Steinbock 1976). Nonspecific infectious lesions on bone are referred to as *periosteal reactions* (when the lesion is confined to the outer periosteal surface of bone, Figure 2.6) and *osteomyelitis* and *osteitis* (when the reaction courses throughout the bone tissue involving both the marrow and cortex). The latter reaction can be diagnosed only via radiographs; but the localization of the inflammatory process rarely occurs and there is usually some degree of involvement of all the anatomical components (Steinbock 1976).

Severe osteomyelitis and osteitis are caused by the spread of the microorganisms of *Staphylococcus* and *Streptococcus*. Periosteal reactions may also be caused by the organisms but other factors can result in a periosteal reaction (Greenfield 1975; Kunitz 1970). Depending on the virulence of the microorganism and the resistance

of the host, the infectious reaction may be an acute and localized one, or chronic and systemic (appearing simultaneously on many bones).

Periosteal reactions result from an elevation of the fibrous outer layer of the periosteum due to the compressing and stretching of blood vessels (Jaffee 1972). Subperiosteal hemorrage occurs which in turn reduces the blood supply to the bone. If the physiological disruption is severe and long term, the periosteal bone tissue will die (necrosis); otherwise, the periosteum will resume normal growth when the disruption is stopped.

Periosteal reactions account for a great majority of pathological alterations found in early human and animal bones (Steinbock 1976). In the New and Old World, infections may be found in every archaeological horizon and geographic location (refer to Jarcho 1966; Ortner and Putschar 1981; Steinbock 1976). Recent research has emphasized the synergistic interaction which infectious disease has with nutritional and degenerative disease. Often one pathophysiological state will predispose an individual to one or several other diseases.

Because infectious disease is so common in archaeological specimens, the interpretation of its meaning will be clear only when it is viewed within the larger cultural context and with respect to analysis of several indicators of stress. Thus, an effective analysis of infectious diseases of nonspecific origin requires several important considerations.

1. The narrowest biologically meaningful age categories should be used because broad age categories will obscure the range of susceptibility.
2. The analysis of the skeletal lesions should distinguish degrees of severity (light, moderate, and severe reactions), describe their location (single bone versus many bones, diaphysis versus epiphysis, long bones versus flat bones), and note any evidence of healing.
3. Consideration should be given to the synergistic reaction between infections, poor nutrition, and cultural factors.
4. The distribution of lesions across sex and age categories should be noted, as should differences in the age of onset, patterning, and frequency among cultural subgroups.
5. The distinction should be made between nonspecific periosteal reactions or osteomyelitis and specific disease entities such as tuberculosis and syphilis.
6. The lesions of infectious disease should be analyzed in conjunction with other stress indicators rather than being interpreted in isolation.

Lallo and co-workers (1978) examined the frequency of infectious disease for 595 burials from Dickson Mounds. They demonstrated that the incidence of infectious diseases increased dramatically as the group changed from a hunting-gathering economy to one based more fully on agriculture. The explanation of this increase rested on the fact that there was an increase in

population density which increased the number of potential hosts and facilitated the transmission of disease within a population. Further, reliance on a maize diet reduced nutritional adequacy. A synergistic interaction between malnutrition and infectious diseases resulted in a higher rate of morbidity and mortality.

Infectious disease can be a powerful indicator of physiological stress in a population, but its meaning in a broader sense can only come from an analysis considering the cultural and ecological context and employing several indicators of stress.

Trauma

Traumatic lesions have been classified by Ortner and Putschar (1981) as belonging to four major types: (1) fractures, (2) dislocations and displacements, (3) deformity induced artificially, and (4) disruption in nerve or blood supply. These types of injury are primarily caused by physical force or by contact with blunt or sharp objects. The cause of trauma can often be determined by analyzing the intensity and direction of the force. Interpretations concerning trauma are straightforward if the age, sex, and health status of the individual are known. If the traumatic lesion occurs with periosteal reaction and infectious inflammation, a severe condition which involves the soft tissue as well as the bone is implied. Steinbock (1976) has stated that simple fractures which do not break through the soft tissue and skin rarely become infected. The degree to which a trauma has healed provides a clue to the relationship between the traumatic event and the death of the individual.

Specific types of trauma often provide a direct inference about specific behavior patterns. Certain activities predispose individuals to certain types of accidental trauma. Moreover, various forms of interpersonal violence (warfare, scalping, mutilation, lacerations) and of surgical intervention (trephination, amputation) may be specifically identified (Ortner and Putschar 1981).

Fractures are the most common traumatic injuries encountered in archaeological populations. The response of bone to a fracture is immediate; vascularization and new bone formation begin within a few days after a break is made. Calcium salts are released from dead bone fragments and also from the living bone and are used in calcifying the callous matrix which forms a binding and connecting sheath around the two bone ends. Within two weeks, calcification is underway, and the internal remodeling and reorganization of the bone callus begins. The process can last for months or years, depending on the severity of the break (Steinbock 1976). Even a poorly aligned bone will eventually mend itself if infection does not set in. The rate of this repair is modified by age, type of fracture, degree of vascularization, amount of motion between the broken ends, and presence of infection (Steinbock 1976). Infectio

at the site of the bone can seriously hamper repair, and the determination of the timing of the fracture on archaeological specimens is rarely possible without determining the nature of the healing process.

One of the most thorough analyses of fractures at the population level is presented by Lovejoy and Heiple (1981) for the Libben skeletal collection. A quantitative approach to the analysis of long bone fractures was shown to be valuable in interpreting various behavioral aspects of the population. These authors found that most fractures occurred as a consequence of accident; the fracture rate was highest in the 10-25 and 45+ age grou; that care of patients was skillful; and that the chance of fracture was largely determined by accumulated years of risk in the population.

Degenerative Conditions

Osteoarthritis is among the oldest and most commonly known diseases afflicting humans. However, the paleopathological diagnosis is sometimes complicated. Measuring the amount of arthritic involvement with skeletal remains is problematic, but numerous researchers have attempted to assess it systematically (refer to Jurmain 1977; Ortner and Putschar 1981). While many factors may contribute to the breakdown of skeletal tissue, such as nutrition, genetics, and even viral infections, the primary cause of osteoarthritis is related to biomechanical wear and tear and functional stress. Biomechanical stress is most apparent at the articular surfaces of long bone joints and is referred to as *degenerative joint disease*. The patterning of degenerative joint disease has been linked to behavioral factors. Individuals who habitually engage in activities which put strain on the joint systems are more likely eventually to show degeneration (refer to Aegerter and Kirkpatrick 1968).

Degenerative joint disease is defined by changes in the articular surface areas of joint systems. Following the exposure of subchondral bone, the articular surface regions become pitted, with marginal lipping and erosion; eventually eburnation takes place. Eburnation is the formation of a very hard callus on bone surfaces which are rubbing together without being cushioned by lubricating fluids. Degenerative joint disease is not an inflammatory disease, but develops on the basis of aging changes and breakdown of the cartilage and lubricating fluid. The condition is slowly progressive, but is not found to occur in all older adults in the same form. Thus, the condition probably is the accumulation of years of alterations of the articular cartilage and breakdown of the joint system. Lifestyle and activity repertoire play an important role in either buffering an individual from arthritis or enhancing the chance that the condition will appear.

The analysis of degenerative joint diseases should consider the severity of the condition, distinguishing slight involvement from severe. There are numerous sets of criteria published to assist the researcher in devising a graded scale of severity of involvement (Aegerter and Kirkpatrick 1968; Jurmain 1977; Martin et al. 1979; Steinbock 1976). In addition, degenerative joint diseases are rarely confined to a single joint complex. The weight-bearing joints such as the hips and knees and those joints exposed to chronic trauma such as the shoulder and elbow are most frequently affected (Jurmain 1978; Martin et al. 1979). The pattern, distribution, severity, and onset by age class and sex in adults can be used to interpret the role of cultural activity in the etiology of degenerative joint disease.

Vertebral osteophytosis is another form of degeneration which is characterized by marginal lipping on the vertebral bodies, and has been associated with changes in the intervertebral disc (Chapman 1973). Commonly found in prehistoric and modern populations, this degeneration typically begins at 30 years of age and affects almost all individuals by 60 years of age (Steinbock 1976). The marginal lipping may range from a slight sharpness to complete fusion of the vertebral bodies. When the degree of osteophytosis is assessed, each vertebra should be divided into four quadrants and each quadrant assessed on both the superior and inferior margins using a scale which ranges from no lipping to extreme bony ridges and eversion at the margins (Chapman 1973). The bony ridges, or osteophytes, can grow to a great size.

As with other pathological processes in bone, maximum information can be obtained regarding the cultural implications of a disease if it is combined with analyses of other stress indicators. A preliminary study correlating the incidence of degenerative joint disease, osteophytosis, and periosteal reactions was undertaken for the Dickson Mound population (Martin et al. 1979). Individuals with multiple joint involvement showed a statistically higher percentage of periosteal reactions. Both infectious lesions and degenerative joint disease appeared to be a function of age, and the more severe arthritic involvements consistently showed more severe infectious reactions.

Dental Pathologies

Dental pathologies are frequently found in prehistoric populations. Among those which are most common are caries, periodontal disease, excess or abnormal attrition, abscessing, excessive calculus, and premortem loss (see Brothwell 1972 for standard scoring techniques and Ortner and Putchar 1981 for illustrations).

Dental pathologies are often interrelated. For example, periodontal disease (measured by degree of alveolar resorption), caries, attrition, and abscessing all may cause premature loss of teeth. Attrition and caries patterns are often interrelated

(Turner and Machado 1983). Both may be a result of diet and eating habits. However, the rate of attrition may affect the rate of caries formation (Armelagos 1969).

An increase in rate of caries through time has been cited as invariably associated with a shift to high carbohydrate and then to refined carbohydrate diets. Based on this relationship, some archaeologists and physical anthropologists have begun to use caries rates as an indicator of carbohydrate consumption where archaeological evidence for diet is insufficient (see Rose et al., Chapter 15 this volume). Turner and Machado (1983) have presented a similar argument for attrition patterns. These interpretations, however, may introduce circularity to the analysis. If caries or attrition are used to help determine dietary changes, then they certainly can not be used to assess the impact of the change.

Finally, while dental pathologies may be common, it is uncertain as to how much they add to the disease load of prehistoric populations. While some conditions may cause temporary pain and others may decrease chewing efficiency, it is likely that they are generally of less consequence to population level adaptation than most of the previously mentioned conditions.

Isotopic and Trace Element Studies

New chemical methods have recently been developed and tested which offer hope for more precise indication of dietary contents than has hitherto been possible. Trace element analysis may provide the most direct information concerning diet of individuals prior to death (Schoeninger 1979; Zurer 1983). From observations of differing amounts of trace elements such as iron, calcium, magnesium, lead, zinc, copper, and strontium, it may be possible to deduce the presence of dietary insufficiencies (Gilbert 1977).

Stable carbon isotopic analysis can provide an assessment of the principal dietary components (Schoeninger et al. 1983; Sillen and Kavanaugh 1982). These studies will contribute to current analyses of dietary change by establishing the presence of certain cultigens such as maize in the diet, segregating groups of individuals with differential access to these cultigens, and determining the proportion of specific cultigens, animal protein, and other foodstuffs in the diet (Bumsted 1981; Smith et al., Chapter 5 this volume; Norr, Chapter 18 this volume). Such analyses, however, are just beginning to make a contribution to paleonutrition research.

CONCLUSIONS: PATTERN, PROCESS, AND MULTIPLE INDICATORS OF STRESS

The purpose of this chapter has been to present a model for the interpretation of skeletal indicators of stress and to provid an overview of the use and meaning of a series of commonly used indicators. The various indicators may have partially overlappin etiologies; but they are not identical or equally useful. For these reasons, coupled with the inherent uncertainty of archaeolo gical research and paleopathological diagnosis, we advocate the use of several indicators (and thereby multiple confirmations) of stress.

Multiple stress indicators may be used to determine the degre and pattern of stress in populations. Is stress primarily chronic or acute, affecting children or adults, related to increased mortality or unrelated to mortality? By evaluating the pattern of stress within populations we may be able to better understand the conditions which are causative of the stress and evaluate likely responses to the stress.

The pattern and severity of stress can also lead to inferences about the causal conditions. As an example, the accumulation of indicators of stress occurring around the ages of two to four has lead Cook (Chapter 10 this volume) to an evaluation of weanling diet. Her conclusion is that the weanling diet in her maize agriculturalists was inadequate and led to the onset of a variety of stresses.

From an examination of a population's total stress load one may begin to make inferences about long-term consequences for culture and behavior. For example, mortality is traditionally high in the younger and older age segments of preindustrial populations. However, this mortality has little impact on a population's ability to maintain itself. If the middle age segment of a population shows signs of increased morbidity and mortality, however, then reproduction and production may be severely affected.

The skeleton is an important artifact or source of information. We have tried to illustrate the fact that bone and teeth may provide an important and interpretable record of events in individuals' lives and their responses to these events.

ACKNOWLEDGMENTS

We wish to acknowledge the following sources of financial support: NIDR Grant No. T-32-DE07047 (A.H.G.), NIH Biomedical Support Grant No. RR07048-17 (D.L.M. and G.J.A.), and NIH Biomedical Support Grant No. 632-509 (G.J.A. and G.C.).

REFERENCES

Acheson, R. M.
1959 The effects of starvation, septicaemia and chronic illness on the growth cartilage plate and metaphysis of the immature rat. *Journal of Anatomy 93*:123-130.
1960 Effect of nutrition and disease on human growth. In *Human growth*, edited by J. M. Tanner, pp. 73-92. Pergamon Press, New York.

Acheson, R. M., and G. B. Fowler
1964 Sex, socio-economic status and secular increase in stature. *British Journal of Preventative Social Medicine 18*:25-34.

Acsádi, G., and J. Nemeskéri
1970 *History of human life span and mortality*. Akademei Kiadó, Budapest.

Aegerter, E., and J. A. Kirkpatrick
1968 Orthopedic diseases (second ed.). W. B. Saunders, Philadelphia.

Allison, M., D. Mendoza, and A. Pezzia
1974 A radiographic approach to childhood illness in pre-Columbian inhabitants of southern Peru. *American Journal of Physical Anthropology 40*:409-415.

Angel, L. J.
1969 The basis of paleodemography. *American Journal of Physical Anthropology 30*:427-438.
1978 Pelvic inlet form: A neglected index of nutritional status. *American Journal of Physical Anthropology Abstract 48*:378.
1982 A new measure of growth efficiency: Skull base height. *American Journal of Physical Anthropology 58*:297-305.

Armelagos, G. J.
1969 Disease in ancient Nubia. *Science 163*:255-259.

Avery, J.
1980 Dentin. In *Orban's oral histology and embroyology* (ninth ed.), edited by S. N. Bhaskar, pp. 107-179. C. V. Mosby, St. Louis.

Bailit, H.
1975 Dental variation among populations. *Dental Clinics of North America 19*(1):125-139.

Bailit, H., P. L. Workman, J. K. Niswander, and C. J. MacClean
1970 Dental asymmetry as an indicator of genetic and environmental stress in human populations. *Human Biology 42*: 626-638.

Blakey, M.
1981 An analysis of hypoplasia and hypocalcification in deciduous dentition from Dickson Mound. In *Biocultural adaptation comprehensive approaches to skeletal analysis*. University of Massachusetts Department of Anthropology Research Reports No. 20, pp. 24-34.

Bocquet-Appel, J. P., and C. Masset
1982 Farewell to paleodemography. *Journal of Human Evolution* *11*:321-333.

Brothwell, D.
1972 *Digging up bones* (second ed.). Trustees of the British Museum, London.

Buikstra, J. (editor)
1981 *Prehistoric tuberculosis in the Americas*. Northwest University Archeological Program, Evanston, Illinois.

Buikstra, J., and D. Cook
1980 Paleopathology: An American account. *Annual Review of Anthropology* *9*:433-470.

Bumsted, M. P.
1981 The potential of stable carbon isotopes in bioarcheological anthropology. In *Biocultural adaptation comprehensive approaches to skeletal analysis*, University of Massachusetts, Department of Anthropology Research Reports No. 20, pp. 108-126.

Cannon, W. B.
1932 *The wisdom of the body*. W. W. Norton, New York.

Carlson, D. S., G. J. Armelagos, and D. P. Van Gerven
1974 Factors influencing the etiology of cribra orbitalia in prehistoric Nubia. *Journal of Human Evolution* *3*:405-410.

Chapman, F. H.
1973 Osteophytosis in prehistoric Brazilian populations. *Journal of Man* *8*:93-99.

Clarke, S. K.
1978 Markers of metabolic insult: The association of radioplaque transverse lines, enamel hypoplasias and enamel histopathologies in a prehistoric skeletal sample. Unpublished Ph.D. dissertation, Department of Anthropology University of Colorad, Boulder.

Condon, K. W.
1981 Correspondence of developmental enamel defects between the mandibular canine and first premolar. Unpublished M.A. thesis, Department of Anthropology, University of Arkansas Fayetteville.

Cook, D. C.
1976 Pathologic states and disease process in Illinois woodland populations: An epidemiologic approach. Unpublished Ph.D. dissertation, Department of Anthropology, University of Chicago.

1979 Subsistence based on health in prehistoric Illinois valley Evidence from the human skeleton. *Medical Anthropology* *4*:109-124.

1981 Mortality, age-structure, and status in the interpretation of stress indicators in prehistoric skeletons: A dental example from the lower Illinois valley. In *The archeology of death*, edited by P. Chapman and K. Randsborg, pp. 133-144. Cambridge University Press, London.

Craig, J. O.
1963 The height of Glasgow boys: Secular and social influences. *Human Biology 35*:524-539.

DiBennardo, R., and H. Bailit
1978 Stress and dental asymmetry in a population of Japanese children. *American Journal of Physical Anthropology 48*: 89-94.

Dickerson, J. W., and R. A. McCance
1961 Severe undernutrition in growing adult animals: 8. The dimensions and chemistry of long bones. *British Journal of Nutrition 15*:567-576.

Dubos, R.
1965 *Man adapting*. Yale University Press, New Haven.

El-Najjar, M., D. J. Ryan, C. Turner, and B. Lozoff
1976 The etiology of porotic hyperostosis among the historic and prehistoric Anasazi Indians of Southwestern United States. *American Journal of Physical Anthropology 44*: 477-488.

Engfeldt, B., and S. Hjertquist
1961 Vitamin D deficiency and bone and tooth structure. *World Review of Nutrition and Dietetics 2*:187-208.

Frisancho, A. R., and S. Garn
1970 Childhood retardation resulting in reduction of adult body size due to lesser adolescent skeletal delay. *American Journal of Physical Anthropology 33*:325-336.

Garn, S. M.
1970 *The earlier gain and later loss of cortical bone in nutritional perspective*. C. C. Thomas, Springfield.

Garn, S. M., A. B. Lewis, and R. S. Kerewsky
1965 Genetic, nutritional and maturational correlates of dental development. *Journal of Dental Research 44*:228-242.

Garn, S. M., C. G. Rohman, M. Behar, F. Viteri, and M. A. Guzman
1964 Compact bone deficiency in protein-calorie malnutrition. *Science 145*:1444-1445.

Garn, S. M., A. R. Lewis, and D. L. Polacheck
1959 Variability of tooth formation. *Journal of Dental Research 38*:135-148.

Garn, S. M., F. N. Silverman, K. P. Herzog, and C. G. Rohman
1968 Lines and bands of increased density: their implication to growth and development. *Medical Radiography and Photography 44*:58-89.

Gilbert, R.
1977 Applications of trace element research to problems in archeology. In *Biocultural adaptation in prehistoric America*, edited by R. Blakely, pp. 85-100. University of Georgia Press, Athens.

Goodman, A. H., G. J. Armelagos, and J. C. Rose
1980 Enamel hypoplasias as indicators of stress in three prehistoric populations from illinois. *Human Biology 52*: 515-528.

Goodman, A. H., and G. A. Clark
1981 Harris lines as indicators of stress in prehistoric Illinois populations. In *Biocultural adaptation comprehensive approaches to skeletal analysis*, University of Massachusetts Department of Anthropology Research Reports No. 20, pp. 35-46.

Goodman, A. H., N. A. Rothschild, and G. J. Armelagos
1983 Social status and health in three prehistoric populations from Dickson Mounds, Illinois. *American Journal of Physical Anthropology 60*(2):199 (abstract).

Gray, P. H.
1967 Radiography of ancient Egyptian mummies. *Medical Radiography 43*:34-44.

Green, S., S. Green, and G. J. Armelagos
1974 Settlement and mortality of the Christian site (1050 A.D.-1300 A.D.) of Meinarti (Sudan). *Journal of Human Evolution 3*:297-311.

Greenfield, G. B.
1975 *Radiology of bone disease* (second ed.). Lippincott, Philadelphia.

Guargliardo, M.
1982 Tooth crown size differences between age groups: A possible new indicator of stress in skeletal samples. *American Journal of Physical Anthropology 58*(4):383-390.

Gustav, B.
1972 Sexual dimorphism in the adult boney pelvis of a prehistoric human population from Illinois. Unpublished Ph.D. dissertation, Department of Anthropology, University of Massachusetts, Amherst.

Harris, H. A.
1926 The growth of long bones in children, with special reference to certain bony striations of the metaphysis and to the role of the vitamins. *Archives of Internal Medicine 38*:785-806.
1933 Bone growth in health and disease: The biological principles underlying the clinical, radiological and histological diagnosis of perversions of growth and disease in the skeleton. Oxford University Press, London.

Hengen, O. P.
1971 Cribra orbitalia: Pathogenesis and probable etiology. *Homo 22*:57-75.

Himes, J. H.
1978 Bone growth and development in protein-calorie malnutrition. *World Review of Nutrition and Dietetics 28*:143-187.

Huss-Ashmore, R.
1981 Bone growth and remodeling as a measure of nutritional stress. In *Biocultural adaptation comprehensive approaches to skeletal analysis*. University of Massachusetts Department of Anthropology Research Reports No. 20, pp. 84-95.

Huss-Ashmore, R., A. H. Goodman, and G. J. Armelagos
1982 Nutritional inference from paleopathology. *Advances in Archeological Method and Theory* 5:395-474.
Jablonski, K. A.
1981 Enamel calcification patterns of the Averbuch site (40DV60), Nashville, Tennessee. Unpublished M.A. thesis, department of Anthropology, University of Tennessee, Knoxville.
Jaffee, H. L.
1972 *Metabolis, degenerative and inflammatory disease of bone and joints.* Lea and Febiger, Philadelphia.
Jarcho, S. (editor)
1966 *Human palaeopathology.* Yale University Press, New Haven.
Johnston, F. E.
1976 Hereditary and environmental determinants of growth in height in a longitudinal sample of children and young of Guatemalan and European ancestry. *American Journal of Physical Anthropology 44*:469-476.
Jurmain, R. D.
1977 Stress and the etiology of osteoarthritis. *American Journal of Physical Anthropology 46*:353-365.
1978 Paleoepidemiology of degenerative joint disease. *Medical College of Virginia Quarterly 14*:45-56.
Kunitz, S.
1970 Disease and death among the Anasazi. *El Palacio 76*:17-22.
Kreshover, S.
1960 Metabolic disturbances in tooth formation. *Annals of the New York Academy of Sciences 85*:161-167.
Lallo, J.
1973 The skeletal biology of three prehistoric American Indian populations from Dickson Mounds. Unpublished Ph.D. dissertation, Department of Anthropology, University of Massachusetts, Amherst.
Lallo, J., G. J. Armelagos, and R. P. Mensforth
1977 The role of diet, disease and physiology in the origin of porotic hyperostosis. *Human Biology 40*:471-483.
Lallo, J., G. J. Armelagos, and J. C. Rose
1978 Paleoepidemiology of infectious disease in the Dickson Mounds population. *Medical College of Virginia Quarterly 14*:17-23.
Larsen, C. S.
1982 The anthropology of St. Catherines Island. 3. Prehistoric human biological adaptation. *Anthropology Papers of the American Museum of Natural History 57*(3).
Lewis, A. B., and S. M. Garn
1960 The relationship between tooth formation and other maturational factors. *Angle Orthodontics 30*:70-77.
Lovejoy, C. O., and K. G. Heiple
1981 The analysis of fractures in skeletal populations with an

example from the Libben Site, Ottowa County, Ohio. *American Journal of Physical Anthropology 55*:529-541.

Lovejoy, C. O., R. S. Meindl, T. R. Prysbeck, T. S. Barton, K. G. Heiple, and D. Kotting
1977 Paleodemography of the Libben site, Ottawa County, Ohio. *Science 198*:291-293.

Maresh, M.
1955 Linear growth of long bones of extremities from infancy through adolescence. *American Journal of Diseases of Childhood 89*:725-742.

Marshall, W. A.
1967 Problems in relating the presence of transverse lines in the radius to the occurrence of disease. In *The skeletal biology of earlier human populations*, edited by D. R. Brothwell, pp. 245-261. Pergamon Press, Oxford.

Martin, D. L.
1983 Paleophysiological aspects of bone remodeling in the Meroitic, X-Group and Christian populations from Sudanes Nubia. Unpublished Ph.D. dissertation, Department of Anthropology, University of Massachusetts, Amherst.

Martin, D. L., and G. J. Armelagos
1979 Morphometrics of compact bone: An example from Sudanes Nubia. *American Journal of Physical Anthropology 51*: 571-578.

Martin, D. L., G. J. Armelagos, and J. R. King
1979 Degenerative joint disease of the long bones in Dickson Mounds. *Henry Ford Hospital Medical Journal 27*:60-63.

Martin, D. L., A. H. Goodman, and G. J. Armelagos
1984 Skeletal pathologies as indicators of quality and quantity of diet. In *The analysis of prehistoric diet*, edited by J. Mielke and R. Gilbert, in press. Academic Press, New York.

Massler, M., I. Schour, and H. G. Poncher
1941 Developmental pattern of the child as reflected in the calcification pattern of the teeth. *American Journal of Diseases of Childhood 62*:33-67.

McCance, R. A., and E. M. Widdowson
1962 Nutrition and growth. *Proceedings of the Royal Society of Britain 156*:326-337.

McHenry, H.
1968 Transverse lines in long bones of prehistoric California Indians. *American Journal of Physical Anthropology 29*: 1-17.

McHenry, H., and P. Schulz
1976 The association between Harris line and enamel hypoplasia in prehistoric California Indians. *American Journal of Physical Anthropology 44*:507-512.

Mensforth, R. P.
1981 Growth velocity and chondroblastic stability as major factors related to the pathogenesis and epidemiological

distribution of growth arrest lines. *American Journal of Physical Anthropology* Abstract *54*:253.

Mensforth, R. P., C. O. Lovejoy, J. W. Lallo, and G. J. Armelagos
1978 The role of constitutional factors, diet and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Medical Anthropology 2*(1):1-59.

Mielke, J., and R. Gilbert (editors)
1984 The analysis of prehistoric diets. Academic Press, New York, in press.

Molnar, S., and S. Ward
1975 Mineral metabolism and microstructural defects in primate teeth. *American Journal of Physical Anthropology 43*: 3-17.

Moore, J., A. Swedlund, and G. J. Armelagos
1975 The use of life tables in paleodemography. *American Antiquity Memoirs No. 30*:57-70.

Moseley, J. E.
1963 The paleopathologic riddle of "symmetrical osteoporosis." *American Journal of Roentgenology 95*:135-142.

Niswander, J. D., and C. S. Chung
1965 The effects of inbreeding on tooth size in Japanese children. *American Journal of Human Genetics 17*:390-398.

Ortner, D. J.
1976 Microscopic and molecular biology of human compact bone: An anthropological perspective. *Yearbook of Physical Anthropology 20*:35-44.

Ortner, D. J., and W. G. Putschar
1981 Identification of pathological conditions in human skeletal remains. *Smithsonian Contributions to Anthropology*, No. 28.

Osborn, J. W.
1973 Variations in structure and development of enamel. *Oral Science Review 3*:3-83.

Park, E. A.
1964 The imprinting of nutritional disturbances on the growing bone. *Pediatrics* (Supplement) *33*:815-862.

Park, E., and S. Richter
1953 Transverse lines in bone: the mechanics of their development. *Bulletin of The Johns Hopkins Hospital 93*:234-248.

Perzigian, A.
1977 Fluctuating dental asymmetry: Variation among skeletal populations. *American Journal of Physical Anthropology 47*(1):81-88.

Pinborg, J. J.
1982 Aetiology of developmental enamel defects not related to fluorosis. *International Dental Journal 32*(2):123-134.

Platt, B. S., and R. A. McCance
1964 Severe undernutrition in growing and adult animals. *British Journal of Nutrition 18*:393-408.

Platt, B. S., and R. J. C. Stewart
1962 Transverse trabeculae and osteoporosis in bones in experimental protein-calorie deficiency. *British Journal of Nutrition 16*:483-495.

Richman, G. A., D. J. Ortner, and F. P. Schulter-Ellis
1979 Differences in intracortical bone remodeling in three aboriginal American populations: Possible dietary factors. *Calcified Tissue Research 28*:209-214.

Rose, J. C.
1973 Analysis of dental micro-defects of prehistoric populations from Illinois. Unpublished Ph.D. dissertation, Department of Anthropology University of Massachusetts, Amherst.
1977 Defective enamel histology of prehistoric teeth from Illinois. *American Journal of Physical Anthropology 46*:439-446.
1979 Morphological variations of enamel prisms within abnormal stria of Retzius. *Human Biology 51*:139-151.

Rose, J. C., G. J. Armelagos, and J. Lallo
1978 Histological enamel indicators of childhood stress in prehistoric skeletal samples. *American Journal of Physical Anthropology 49*:511-516.

Rose, J. C., K. W. Condon, and A. H. Goodman
1984 Diet and dentition: developmental disturbances. In *The analysis of prehistoric diets*, edited by J. Mielke and R. Gilbert, in press. Academic Press, New York.

Rose, J. C., and J. N. Pasley
1980 Stress and dental development: An experimental paleopathological model. *American Journal of Physical Anthropology 52*:272 (abstract).

Rudney, J.
1981 The paleoepidemiology of early childhood stress in two ancient Nubian populations. Unpublished Ph.D. dissertation, Department of Anthropology University of Colorado.

Sarnat, B. G., and I. Schour
1941 Enamel hypoplasia (chronological enamel aplasia) in relation to systemic disease: A chronologic, morphological and etiologic classification. *Journal of the American Dental Association 28*:1989-2000.

Schoeninger, M. J.
1979 Diet and disease states in Calcatzingo: Some empirical and technical aspects of strontium analysis. *American Journal of Physical Anthropology 51*:295-310.

Schoeninger, M. J., M. J. DiNiro, and H. Tauber
1983 Stable nitrogen isotope ratios of bone collagen reflects marine and terrestrial components of prehistoric diet. *Science 220*:1381-1383.

Schulz, P. D., and H. McHenry
1975 The distribution of enamel hypoplasia in prehistoric California Indians. *Journal of Dental Research 54*(4):913.

Selye, H.
1950 *Stress: The physiology and pathology of exposure to systemic stress.* Acta, Inc., Montreal.
1971 Hormones and resistance, part 1. Springer, New York.

Shore, L. R.
1935 Polyspondylitis marginalis osteophytica. *British Journal of Surgery 22*:850-863.

Sciulli, P. W.
1977 A descriptive and comparative study of the deciduous dentition of prehistoric Ohio Valley Amerindians. *American Journal of Physical Anthropology 47*:71-80.

Sillen, A., and S. Kavanagh
1982 Strontium and paleodietery research: A review. *Yearbook of Physical Anthropology 25*:69-90.

Steinbock, R. T.
1976 *Paleopathological diagnosis and interpretation.* C. C. Thomas, Springfield, Illinois.

Stewart, R. J. C.
1975 Bone pathology in experimental malnutrition. *World Review of Nutrition and Dietetics 21*:1-74.

Stini, W.
1969 Nutritional stress and growth: Sex differences in adaptive response. *American Journal of Physical Anthropology 31*:417-426.
1972 Reduced sexual dimorphism in upper arm muscle circumferences associated with protein-calories deficient diet in South American populations. *American Journal of Physical Anthropology 36*:341-352.
1975 Adaptive strategies of populations under nutritional stress. In *Biosocial interactions in population adaptation*, edited by E. S. Watt, F. E. Johnston, and G. W. Lasker, pp. 19-39. Mouton, The Hague.

Stout, S. D.
1979 Histomorphometric analysis of archeological bone. Unpublished Ph.D. dissertation, Department of Anthropology, University of Washington.

Stout, S., and D. J. Simmons
1979 Use of histology in ancient bone research. *Yearbook of Physical Anthropology 22*:228-249.

Sundick, R. I.
1978 Human skeletal growth and age determination. *Homo 29*: 228-249.

Swärdstedt, T.
1966 *Odontological aspects of a Medieval population in the province of Jamtland/Mid-Sweden.* Tiden-Barnangen Tryckerien, Stockholm.

Swedlund, A. C., and G. J. Armelagos
1969 Une recherche en Paleodemographie: La Nubie Soudanaise. *Annales Economies Societies Civilisations* Ser. 4 *24*:1281-1298.

1976 *Demographic anthropology.* W. C. Brown, Dubuque, Iowa.

Tanner, J. M.
1977 Human growth and constitution. In *Human biology,* (second ed.), edited by G. H. Harrison, J. S. Weiner, J. M. Tanner, and N. A. Bannicot, pp. 301-385. Oxford University Press, New York.
1978 Fetus into man. Harvard University Press, Cambridge, Massachusetts.

Thomas, R. B.
1973 Human adaptation to a high Andean energy flow system. Pennsylvania State University Occasional Papers in Anthropology No. 6.

Trowell, H. C., J. N. Davies, and R. F. Dean
1954 *Kwashiorkor.* Arnold, London.

Turner, C., and L. M. Machado
1983 A new dental wear pattern and evidence for high carbohydrate consumption in a Brazilian Archaic skeletal population. *American Journal of Physical Anthropology 61*(1): 125-130.

Ubelaker, D. H.
1978 *Human skeletal remains: Excavation, analysis and interpretation.* Aldine, Chicago.

Van Gerven, D.
1972 Sexual dimorphism in the adult human femur. Unpublished Ph.D. dissertation, Department of Anthropology, University of Massachusetts, Amherst.

Van Gerven, D. P., and G. J. Armelagos
1983 "Farewell to paleodemography?" Rumors of its death have been greatly exaggerated. *Journal of Human Evolution 12*: 353-360.

Watson, A., M. Massler, and M. Perlstein
1964 Tooth ring analysis in cerebral palsy. *American Journal of Dentistry 107*:370-384.

Welcher, H.
1885 Cribra orbitalia. *Archive für Anthropologie 17*:1-18.

Wells, C.
1964 *Bones, bodies and disease.* Praeger, New York.
1967 A new approach to paleopathology: Harris lines. In *Disease in antiquity,* edited by D. R. Brothwell and S. T. Sandison, pp. 390-404. Thomas, Springfield, Illinois.

Widdowson, E. M., and R. A. McCance
1964 Effects of nutrition and disease on growth. *British Dental Journal 117*:326-330.

Wing, E. S., and A. B. Brown
1980 *Paleonutrition: Method and theory in prehistoric foodways.* Academic Press, New York.

Woodall, J.
1968 Growth arrest lines in long bones of the Cases Grandes population. *Plains Anthropologist 13*:152-160.

Yaeger, J. A.
1980 Enamel. In *Orban's oral histology and embryology* (ninth ed.), edited by S. N. Bhaskar, pp. 46-106. Mosby, St. Louis.

Zurer, P. S.
1983 Archeological chemistry. *Chemical Engineering News 61*: 26-44.