ORTHOPEDIC PROBLEMS OF THE WETHERILL MESA POPULATIONS

MESA VERDE NATIONAL PARK, COLORADO

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Mug House, view from the south.

Image of human burial removed from the electronic edition.

A typical Mesa Verde burial in the trash slope of Mug House.

PUBLICATIONS IN ARCHEOLOGY 7G · WETHERILL MESA STUDIES

ORTHOPEDIC PROBLEMS OF THE WETHERILL MESA POPULATIONS

MESA VERDE NATIONAL PARK, COLORADO

BY JAMES S. MILES M.D.

NATIONAL PARK SERVICE · U.S. DEPARTMENT OF THE INTERIOR
WASHINGTON 1975

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PREFACE

This study of human skeletal remains from archeological excavations on Wetherill Mesa, Mesa Verde National Park, was performed as an adjunct to the usual anthropological studies. The objective was to engage the services of a clinical orthopedist to identify the disease processes demonstrated by these bones and skeletons, primarily in hopes that such an evaluation would define more clearly the life, habits, and problems of the Indians who once inhabited the area.

The Park and Its Setting

Mesa Verde National Park is located on a towering, relatively flat table land, overlooking the Montezuma Valley in southwestern Colorado. The northern portion of the mesa rises as a precipitous bluff 1600 feet above the valley floor. The top of the mesa, which is covered by an evergreen, juniper, pinon forest, slopes gently to the east and the south, and is broken by large canyons into many finger-like projections.

The many cliff dwellings on the mesa were well known to the Indians of the region, but were not discovered by early Spanish or American visitors to the area. The first known record of the dwellings was made in 1874 by William H. Jackson, the photographer of the Hayden Expedition. Although other cliff dwellings were discovered by subsequent government survey parties, the major credit for exploration of the mesa and discovery of the larger cliff dwellings belongs to Richard Wetherill and his brothers. The Wetherills were ranchers living in the Mancos Valley. While searching for stray cattle, they discovered Cliff Palace and other major ruins in December of 1888. Later they found and recorded all of the major cliff dwellings, and probably most of the smaller ones, in the Mesa Verde area. The inexpert, but, for the time and place, intelligent excavations of the Wetherill brothers were supplemented by the more professional work of Nordenskiold of Sweden. The collections of materials and the excavations excited great interest and culminated in the formation of Mesa Verde National Park in 1906. Under the direction of J. W. Fewkes and others, the major cliff dwellings of the Chapin Mesa were excavated and stabilized. As the number of visitors to the park increased, the enjoyment of the individual visitor decreased because of crowding, and the archeological monuments of the Park began to suffer "visitor erosion." The National Park Service then made plans to excavate a similar series of dwellings on another of the mesa projections, and thus the Wetherill Mesa Archeological Project was begun in 1958. Over 800 archeological sites were found by the Project's survey, and the sites were chosen carefully to tell the story of Mesa Verde inhabitants and to yield the greatest in research values.

The Early Inhabitants

The precise time of arrival of the Pueblo Indians on the Mesa Verde is unknown. It is believed, however, that they were there during the early portion of the "Basketmaker II" period. This term chosen is descriptive of the pre-Pueblo culture between approximately A.D. 1 and A.D. 450–600. These people were skilled in weaving baskets and used tools and instruments of stone, bone, wood and shell. They were almost exclusively agriculturalists, cultivating corn and squash. They do not appear to have had pottery or the bow and arrow.

Sometime after A.D. 600, the basket maker culture in the Four Corners area advanced significantly with the introduction, probably from the south, of pottery, the bow and arrow, and bean agriculture. Although the earlier Basketmaker II homes (pit houses of varying complexity) have been found in southern Colorado, none have been located in the Mesa Verde. The house structures of the Basketmaker III people, were semisubterranean pit houses of one or two rooms with a heavy superstructure, low walls and a flat roof, tightly thatched and covered with earth. These pit houses were disseminated over the mesa top, in the open, near the cultivated fields and, apparently more rarely, in the large alcoves where the cliff dwellings were later built. This modification of the basketmakers' existence made by the introduction of pottery, beans, the bow and arrow, and the enlarged pit houses, took place in the period before A.D. 750.

In the latter part of the 8th century, the Indians began to abandon their pit houses and constructed vertical-walled, above ground, flat-roofed houses, built in curving contiguous rows. To this, or a more developed grouping of rooms, the Spanish later applied the name of "pueblo," and the Indians who built in this fashion have been called the Pueblo Indians. The development of the Pueblo I period, as it is called, was gradual and took place between the years of A.D. 750 and 900. During this period, the kiva appeared as a modification of the pit house and was used as a ceremonial room. It remained entirely, or almost entirely, subterranean and was circular. The Indians continued to be primarily agriculturalists, although the bow and arrow probably permitted more successful hunting.

The next period, Pueblo II, roughly A.D. 900 or 1075 or 1150, depending on the area and cultural criteria applied, was generally one of increasing population and architectural

sophistication. Agricultural techniques that were thoroughly attuned to the area were either perfected or introduced. Walls were now made primarily of stone; adobe mud was used as mortar instead of being a major component of the wall. House blocks were not built in a long curving row, but were more tightly planned with the kivas in front (usually to the south). Water control devices, check dams, terraces and ditches are indicative of practical engineering knowledge. This was apparently the most successful period of occupation in much of the Four Corners area.

Archeologists use the year A.D. 1100 as a convenient break for the change to the next period: Pueblo III (A.D. 1100–1300). This is the "classic period," but it also was a decadent and terminal one in the Four Corners area. Modifications of construction indicate an increasing need for security. Double walls replaced single walls in house construction, tower structures appeared, compact pueblos replaced the previously open houses. For about 100 years the Indians continued to live on the mesa top near their fields, although these architectural changes suggest a period of social unrest or change. The pressure may have come from nomadic groups outside the area, or, more likely, there may have been quarrels among the people themselves. Probably developing, and perhaps competitive theocracies, soil loss and exhaustion, and periods of dryness and coolness, with shorter growing seasons may have culminated in undue pressure on the economy and social structure. About A.D. 1200 the Indians intensified an already initiated move to abandon their mesa top dwellings and again moved into the caves. The spectacular cliff dwellings of the mesa were built in the next few years. These cliff dwellings were more easily defended, but certainly they were far from ideal: the fields of the Indians were exposed and distant on the mesa top, as well as on nearby talus slopes. The water supply of the caves was inadequate.

There is good evidence of a population decline at the end of the Pueblo II period. Late in the 13th century the exodus from Mesa Verde and, indeed, from the entire Four Corners area was accelerated, and by 1300 the region must have been essentially abandoned. The Indians probably left in small groups, traveling to pueblos south along the Rio Grande. Certainly, the last 24 years of the 13th century were characterized by a drought. The prolonged dry period may have been the telling blow to the Indians, although it did not appear to be particularly severe. At any rate, there are no known tree ring building dates for Mesa Verde after the late 1200's, and 1300 is therefore taken as a fairly accurate date for the termination of Pueblo occupation there.

Archeological Investigations

Chosen for excavation on Wetherill Mesa were many archeological sites that tell the story of the inhabitants of the mesa. Pit houses of the Basketmaker III period, the earliest found on the mesa, are scattered over many portions of the mesa top. These are deeply buried and usually must be located by soil auger. Some pit houses have been found in the caves, and, in this respect, Step House with its Basketmaker III pit houses (dated in the 600's) is of particular interest. It is apparent the early Indians of the Basketmaker III period lived in the Step House alcove, that it was then abandoned, only to be reoccupied in the 12th century.

Two pit houses of the Basketmaker III or Modified Basketmaker (A.D. 450–750 or 800) were excavated on Wetherill Mesa (Site 1644), but no human burials were found. The dwellings of Long House, Mug House (frontispiece A), Adobe Cave and Step House, are typical of the classic period, the late 12th- and 13th-century reoccupation of the caves by the pueblo people. They are all cliff dwellings, varying in size and complexity. Exceptions to these generalizations are found in most sites: Step House, and in some evidences of earlier inhabitation (Pueblo II) in Mug House, and in a Basketmaker III pit house below the later structures in the eastern end of Long House alcove.

Between the Basketmaker pit houses and the late classic Pueblo III sites (such as Long House) are a series of excavations, all of which yielded skeletal material. Badger House is a translation from the Navajo, "Kin Naaschiti," and was so named because of the numerous badger holes in the midden immediately south of the house block. It was occupied during Pueblo I, II and III periods and is a particularly valuable site. The earlier evidence was largely destroyed by the latter occupation, primarily the long Pueblo II period. The Pueblo III use of the site was brief, but a house block, or tower and kiva were built. Numerous burials were found at Badger House.

Big Juniper House, which likewise yielded human remains, was also occupied at least twice for long periods. Both Pueblo II and early Pueblo III occupations were distinguished, but they were rather thoroughly intermingled by later builders. Re-use of building stone and various structural additions left the site an archeological puzzle, which, though comprehensible to the archeologist, was too complex for visitor interpretation.

Two Raven House was excavated in 1962. It is also a Pueblo II and early Pueblo III site, but the Pueblo II remains were predominate, and the Pueblo III material stands out with greater clarity than it did at Big Juniper. Unusual structures were noted at Two Raven House, including a stockade of widely spaced posts at the eastern edge of the cite. Burials were found at this site.

Archeological excavations of Site 1676 were finished during the summer of 1963. It was a huge dwelling, but several separate excavations sufficiently delineated both the areal and temporal extent of the settlement. Few burials were found there, a condition not uncommon in Pueblo I excavations. As with all of the sites described above except Long House, Step House, Mug House and the Adobe Cave, this community was a mesa top pueblo. Several other small excavations, some of which yielded skeletal material, were made.

From the outset of the excavations, the decision was made to obtain all possible information from materials removed. Accordingly, all specimens from the debris, rooms, and kivas were studied, classified and catalogued. The tools, implements and articles of manufacture were thoroughly studied, as is customary in modern scientific archeology. Architecture, settlement pattern, and all possible remnants of the prehistorical period at Mesa Verde were analyzed. Cultivated plant specimens were not merely identified but also were evaluated on the basis of their growth characteristics, indicating plant disease, human agricultural advancement and effects of weather. Soil columns were analyzed for mineral and nitrogenous content. Pollens were studied for indications of natural plant growth or cultivated materials, for floral change and possible concomitant climatic change. Animal remains were studied to indicate the patterns of animal use, both domesticated and wild. The bones and plant remains aided in a comprehension of basic ecological change, if any, between the past and the present.

Human remains were thoroughly examined to ascertain the physical and health characteristics of the inhabitants themselves. The remains were examined by a physical anthropologist, by a parasitologist, and by an orthopedist. It was hoped this study of the human bones might shed some light upon the physical being of the inhabitants of Mesa Verde, and also might indicate some of the reasons for the various moves and migrations made by the Indians.

Burial habits of the Indians were quite simple (frontispiece B). Apparently the dead were buried singly in shallow graves, with very few personal articles and effects. Numerous graves were found in the various archeological sites and, although the number of graves found is not adequate for the estimated population of the mesa, there is no indication that these burials were selective or that other burial habits might have influenced the sample. There was no evidence of cremation. It is probable that deaths of mesa inhabitants in places remote from their dwellings resulted in burials away from the mesa. Mummification was not practical. The few mummies found on the mesa are the result of simple dessication and do not represent any change in burial methods.

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ILLUSTRATIONS

EDITOR'S NOTE

The human skeletal remains discussed by Dr. James S. Miles came from 16 sites on Wetherill Mesa. Many elements belonged to specific burials, whereas many others were simply miscellaneous bones. The latter are identified in text and captions by catalogue numbers, e.g., 12345/718. The first number is the serial number assigned to a given specimen, and the second is the accession number of a given site. Accession and site numbers (and in some cases, site names) follow:

700	Site 1200, Long House
702	Surface survey sites (yielding pottery and non-pottery artifacts)
703	Site 1229, Mug House
704	Site 1205
706	Site 1452, Badger House
708	Site 1228, Adobe Cave
172*	Site 1228, Adobe Cave
175*	Site 1228, Adobe Cave
709	Site 1285, Step House
710	Site 1595, Big Juniper House
711	Site 1645, Two Raven House
712	Site 1227
713	Site 1644
714	Site 1676
715	Site 1230
716	Site 1253
717	Site 1575
718	Site 1291
719	Site 1801

* Accession numbers applied to earlier excavation sites.

1 INTRODUCTION

All of the human skeletal remains removed during the excavations were submitted for the study. The bones of a single burial were collected and catalogued as a unit. Miscellaneous bones were also identified and catalogued. For preservation, most of the bones were dipped in a transparent and non-radio-opaque plastic material. A select number of the bones were not dipped in this plastic. These bones were saved in the hope that research methods might be developed in the future which would permit additional studies such as chemical analysis or blood typing. No attempt was made to articulate any of the burials, although some of them were in such excellent condition that articulation would have been relatively easy.

It is impossible to estimate accurately the number of burials represented by the miscellaneous bones. The physical proximity of bones excavated by the archeologists was taken as an indication of a single burial or, conversely, of multiple burials. Obviously, the bones of a single individual might have been scattered by design or by accident. Although many of these miscellaneous bones could have presumably been parts of a single skeleton, no attempt was made to group them since the accuracy of such a single burial would have been highly questionable.

All of the bones were also examined by a physical anthropologist. When the skeletons were complete, fairly accurate anthropological and orthopedic evaluations could be made. Very little could be determined with the miscellaneous bones except in regard to individual abnormalities. Many of the bones were subjected to roentgenography. Roentgenograms were obtained of obviously normal bones as well as of most of the abnormal ones. The purpose of radiologic examination of presumably normal bones was to find lesions which might have gone undetected during preliminary inspection, and also to establish a norm of the changes which would have been produced in these bones by erosion and the elements. Many of the abnormal bones were also photographed. Microscopic sections were made of some of the bones. These sections were very few in number and they added very little information because of the absence of cellular remains. Only the osteones were evident, but no decisions could be made as to which osteones were enlarging or which were being reabsorbed. Roentgenograms could indicate almost as accurately the cortical and trabecular patterns, and the processes involved.

The abnormalities of the bones, joints, and skeletons were identified, and a probable clinical cause (etiology) was determined. In some instances, the morphology of the abnormality permitted a clear cut determination of the disease or process. Roentgenograms aided greatly, for the radiologic appearance of a disease, process, or lesion is often quite characteristic. A discussion of probable causes for an abnormality is presented for those cases in which the etiology is uncertain. Clinical experience and judgement were necessary in assigning a most probable cause. Such an assessment can not exclude the rare, the unusual, or improbable disease; but theories are built on probabilities—not possibilities.

In some instances, there may also have been dual causes (etiologies) which were probabilities. For example, a joint that had been damaged by infection would still function in a limited, deranged fashion. Such an abnormal function would have led to premature or excessively severe degeneration. In this instance, the probable causes would have been both infection and degeneration. These etiologies would have been serial in order. If both etiologies were apparent, both were recorded and tabulated.

In other instances, the abnormality could be identified, but a probable cause could not be assigned. This diagnostic problem is due either to an incomplete understanding of the disease process which produced the abnormality, or the disease itself may have been caused by one of several equally likely diseases. An example of an incompletely understood bone abnormality or disease is Paget's Disease. Here, the process may be identified morphologically or roentgenographically, but no clinical cause has ever been determined. The disease is known, but it remains a mystery. An example of an abnormality or disease which could have had many causes is periostitis of the bone. Here, the periosteum (the fibrous tissue envelope about a bone) is irritated or damaged, and responds by building layers of reactive bone on the host bone cortex. This reaction is quite non-specific and may be due to pyogenic infection of low grade virulence, to tuberculosis, to syphilis, to repeated irritation of minor trauma, or even to minor, hidden fracture. When the probable cause of an abnormality could not be determined, it was simply listed as being of uncertain cause.

A few abnormalities were so bizarre as to be outside the author's clinical experience and knowledge. The advice of numerous other orthopedists and radiologists was sought in the examination of these specimens and roentgenograms. The consultants could only confirm the confusion. These abnormalities are discussed, classified as "mysteries," and are tabulated as being of "uncertain causation."

In fairness to myself and to the orthopedic and radiologic consultants, it must be added that the absence of soft tissues

and surrounding structures contributed to the confusion. Had these tissues and structures been present, the additional information might have made a diagnosis possible since many disease prosesses involve tissues other than bone.

The absence of these soft tissues and structures made it impossible for our assessment of the general health (or lack of such) of the Wetherill Mesa populations. Diseases of the intestines, lungs, kidneys, heart, and arteries, including arteriosclerosis, remain unknown, and this study could give no additional clues as to their presence or absence. In addition, professional diagnostic work could not expand beyond speculation because of the lack of soft tissues. For example, it was possible only to speculate that there might have been soft tissue abscesses and sinuses in those persons with osteomyelitis.

The various causes or etiologies were then grouped in a number of large categories. Such etiologic categories are commonly used by present day physicians. The etiologic categories are applicable not only in the field of orthopedics, but in other medical and surgical specialties. They are useful in the study and teaching of disease.

The following etiologic categories were used:

1. Disturbances of prenatal influence
2. Disturbances secondary to metabolic abnormalities
3. Abnormalities of growth

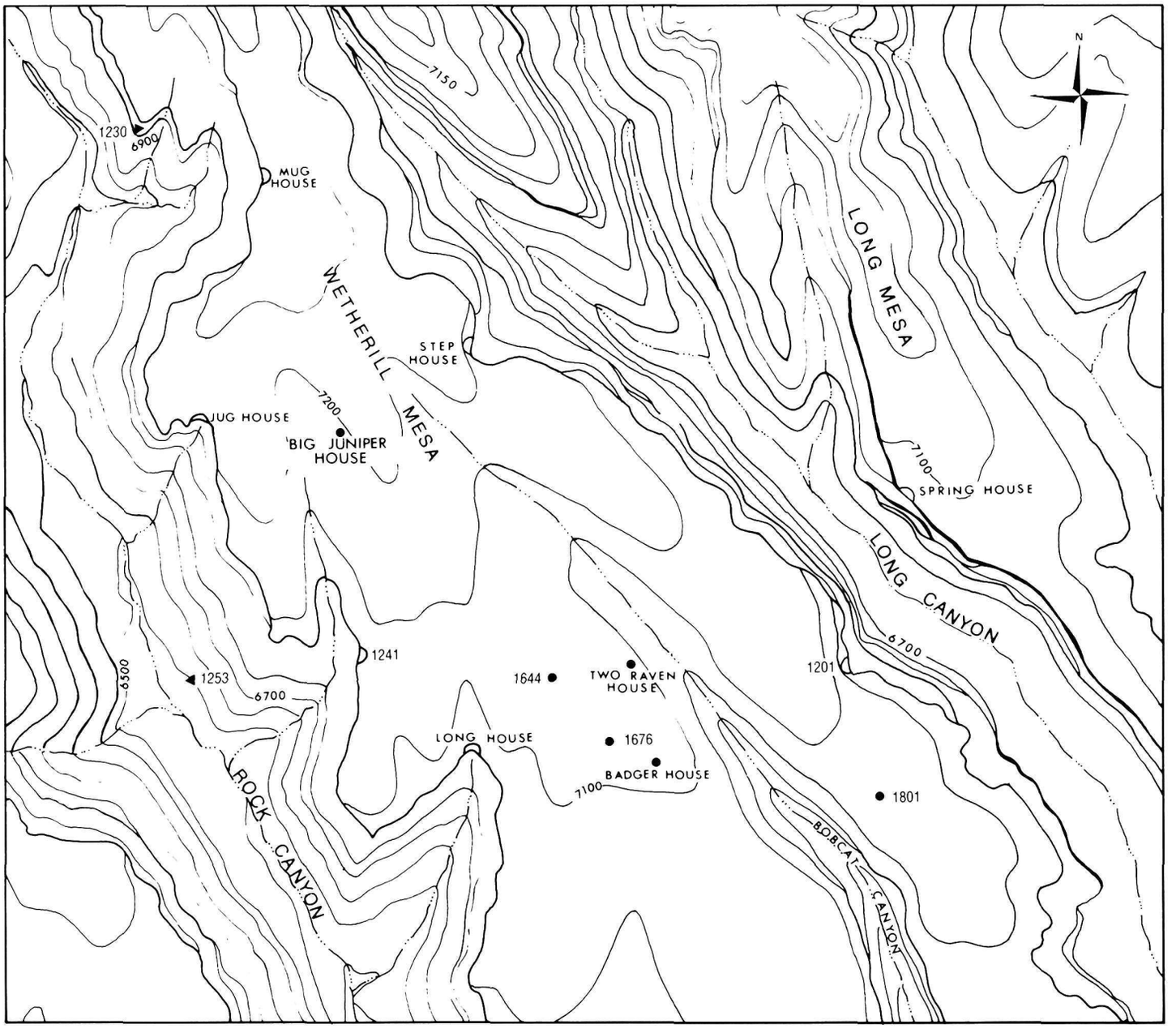


Figure 1.—Map showing selected sites on Wetherill Mesa.

4. Degenerative processes
5. Infections
6. Trauma
7. Disturbances secondary to vascular interruption
8. Disturbances secondary to neurologic defects
9. Neoplasia
10. Diseases of unknown or uncertain origin

Determination of the age of the individuals of the burials presented some problems. The state of preservation of the bones and the degree of completeness of the skeletons obviously affected the estimation of the ages. The ages of "subadults", the infants, children and adolescents, were estimated with much greater accuracy than were the ages of the adults since the immature individuals could be fitted into standard "bone age" tables if the skeletons or parts were relatively complete. Most human bones develop in cartilage models, and at amazingly regular ages, ossification centers begin in these cartilage models, one in the center of the "bone" (the diapligses) and usually one on either end (the epiphyseal ossification center). These areas of osseous tissue grow and enlarge in the process of enchondral ossification. As a child grows in height, the "bones" enlarge and elongate through the growth plates or epiphyseal cartilages, the remnants of the original cartilage model between ossification centers. These cartilage discs are usually situated at one or both ends of the long bones. The cartilage grows in a longitudinal direction, and the bone of the shaft (diaphysis) also elongates in a longitudinal direction. It would seem that the bone of the diaphysis is trying to "catch up" with the longitudinal growth of these epiphyseal cartilages. This zone of the "catching up" of bone growth is called the metaphysis of the bone. Not only is the bone of the metaphysis replacing the older more mature cartilage in the process of enchondral ossification, but there is also extensive remodelling of the shape and diameter of the long bones. In general, the transverse diameters of the epiphyseal cartilage plates is much greater than the transverse diameters of the diaphyses of long bones. Finally, the cortex of the diaphysis is much thicker than the cortex of the metaphyseal area, and in the diaphysis, the spicules of spongy bone (the trabeculae or cancellous bone) of the metaphysis are replaced by a marrow space (the medullary cavity) which is filled with blood cell precursors or fatty marrow. Again, at amazingly regular ages, the bone of the metaphysis catches up with the cartilage growth of the epiphyseal plates. The bone of the metaphysis now unites with the bone of the end or end of the long bone. This final process is termed epiphyseal closure. Now the long bone consists almost entirely of osseous tissue surrounding the medullary cavity. Remodelling of the bone and longitudinal growth cease. The only cartilage that remains of the primary cartilage model is that cartilage (called articular cartilage) which coats the end of a long bone inside a joint. Thus, the ends of the two long bones within a joint glide on two articular cartilage surfaces. This articular cartilage is an amazing tissue. The friction developed in the gliding of one such surface is the lowest coefficient of friction known to man. It is far lower than any man has been able to provide in any of his most highly developed and best oiled machines. It is even less than that of ice on ice.

The time of closure or disappearance of the epiphyseal cartilage is not related to the ultimate stature of the individual. Whether the person is destined to be five feet in height or six and one half feet in height, the closure of a specific epiphyseal cartilage occurs at the same age in the two individuals. Obviously, the rate of epiphyseal cartilage growth and the rate of metaphyseal enchondral ossification is much greater in the taller person. The regularity of the age of appearance of the ossification centers, their enlargement, and the age of closure of certain epiphyseal cartilages have led to the construction of "bone age" tables. For example, at age seven months, an infant should have a set number of these centers in the bones of the wrist and foot. If an infant has more than the usual number of centers, his bone age is greater than his chronologic age. This is a rare occurrence. Reasoning in reverse fashion, the number of ossification centers present can be counted in the roentgenogram or in the gross examination, and the bone age of the person can then be determined. At the other end of the growth scale, the "open" and closed epiphyseal cartilages may be seen radiographically or grossly, and the bone age of the adolescent determined.

Application of present day bone age tables to the Wetherill Mesa populations may be subject to question. However, these tables are probably more than reasonably accurate, for they seem to fit the persons of many diverse racial and cultural backgrounds of today's populations. At any rate, these tables are the best in existence, and they are probably adequate for this study.

The age estimations for the adults of this study presented a much greater problem. The standard method used by most physical anthropologists is based upon the morphology of the symphysis pubis and the degenerative changes in the peripheral joints. As previously noted, the intraarticular ends of long bones are covered with articular cartilage. While this articular cartilage has amazing physiologic properties, it is also particularly vulnerable. It begins to degenerate very early in life, and for some unknown reason, the degenerated or aged articular cartilage is replaced by bone. The articular cortices of the bone become thickened and deformed, the congruity of the articular surfaces is destroyed, and spurs develop about the margins of the joint. This whole process is known as degenerative arthritis, osteoarthritis, or simply "arthritis".

In the symphysis pubis, the cartilage of the symphysis is replaced by peculiar little ridges or rings. Rarely, all of the cartilage of the symphysis is replaced by bone, and the two halves of the pelvis anteriorly become a single united bone.

It is well known that degenerative arthritis appears at grossly different ages in adults. A person of 40 may be

completely disabled by this arthritic process while a person of 90 may be amazingly spry and mobile. Factors other than age affecting the degenerative process of articular cartilage include climate, diet, disease, injury, racial and cultural characteristics. Thus, it is apparent that the bone age determinations in adults that are based upon degenerative changes are much less accurate than the bone age determinations of sub-adults that are based upon ossification center appearance, growth, and closure. However, such estimations for adults are the best available, and are probably accurate enough for the study. Since all of the bone age determinations were made by the same physical anthropologist, the errors of individual observation were minimized.

Determining the sex of the individual also presented some problems. Such determinations are made according to the configuration and development of the pelvis and other bones which differ markedly in adult males and females. The size of the bones might be of some help, but it obviously could not be the only indicator of sex. Male and female infant and children's bones are quite indistinguishable. Not until adolescence and puberty do these distinguishing changes take place in the pelvis and other bones. Thus, the sex of infant and children specimens could not be ascertained, and the sex of the specimen was simply recorded as unknown. In some adolescents, the determination of sex could be made. Of the sub-adult specimens, only eight presented sufficient sex identifying characteristics. In the adult, the skeletal characteristics are usually distinct enough to determine the sex. Only 12 of the adults had sufficient damage to the skeletal material to prevent such identification. Of the remaining adults, 50 were males, and 41 were females.

The number of skeletal remains was significant. There were 179 easily identified individual burials. Some of these were complete while others had only a few bones missing. Some bones were fragmented by trauma following the burial, by erosion, or by both. The 179 individual burials listed could be identified, however, as distinct burial attempts. There were 366 additional burials which also varied greatly in completeness and quality of preservation. In a few instances, only single bones were present. In other instances, a fairly sizable number of bones were in close proximity and could be assumed to be a single individual since there were no other adjacent burials. In some instances, scattered bones were found. Such incomplete burials probably do not indicate a like number of individuals. Scattering of the skeletal remains might have resulted in the bones of an individual being found in widely separated areas, and thus being listed as different specimens with different catalogue numbers. Approximately 40,000 bones were examined in this study.

The burials were listed by site of provenience, although the individuals of a particular site may have come from cultures of different periods. For example, specimens removed from the Juniper House could have been either from the Pueblo II or Pueblo III periods. Concurrently, burials from the top of the Wetherill Mesa could have come from several different cultural periods.

In appendix 1, the archeological sites, the cultural ages, and the number of burials and bones are listed. The sites of earliest occupancy are listed first. Some of these sites represented archeological jumbles where one period of habitation was followed by a subsequent period of habitation. These archeological details are presented elsewhere in other reports of this archeological project on Wetherill Mesa.

In appendix 2, the abnormalities of the skeletal material are listed by site or provenience. Sex and age (if known) are also listed. In general, the sites are arranged by their cultural ages with the earlier sites placed first. The abnormalities are also listed by their etiologic group. The numbers and types of abnormalities are perhaps significant in themselves. No importance, however, may be attached to percentages of abnormalities in relation to the total volume of material studied. Such figures would be significant only if all the skeletons were complete and in an excellent state of preservation. The gross number of such abnormalities in each category is of more interest clinically than statistically.

Because of the small numbers of individuals involved, it was impossible to compare and contrast the diseases of one cultural period with another. One cannot state that the Indians were more healthy in one period than another.

2 ETIOLOGIC CATEGORIES

Disturbances of Prenatal Origin

Strictly speaking, the disturbances of prenatal origin are those which are genetically controlled. However, the science of genetics is as yet quite incomplete and does not permit definite conclusions regarding the genetic causation of some abnormalities. A number of abnormalities have been included in this category which are presently believed to be of genetic influence. It is very likely that research may clarify the etiology of these diseases or processes or abnormalities, removing some from the category of prenatal genetic causation, or adding still more to this category.

Unfortunately, some of these diseases or processes have been termed congenital and this has given the impression that any disease or process present at birth was of genetic control. It is now known that many are lesions that were acquired *in utero*. The most commonly known example is of blindness in the infant resulting from the viral disease rubella of the pregnant female during the first three months of her pregnancy when the eyes of the fetus are being formed. Similarly, other diseases or processes may be acquired by the fetus. In the orthopedic field, a process that quickly comes to mind is congenital dislocation of the hip. At the present time, this process is believed to be caused by many cumulative factors. Only one of these factors is the genetic influence.

The prenatal disturbances may be single, simple isolated lesions, such as extra fingers or toes, or they may be of a more generalized and spectacular nature with abnormalities of the entire skeleton and many other tissues. Osteogenesis imperfecta exemplifies the latter type of disturbance. Not only is there a generalized abnormality of the skeleton, but the disturbance also produces blue sclerae, deafness, and other problems.

In addition, it may well be that some of these problems may be due to an altered growth pattern which follows the original abnormality. For example, a single bone, such as the patella, usually develops from a single ossification center. The bone occasionally develops from two centers—a genetic abnormality. Subsequently, the two ossification centers usually unite with continued growth, but occasionally, the two centers may persist into adult life—a failure of the anticipated normal growth process.

The most frequent disturbances of prenatal origin in this study were those related to the fusion of secondary ossification centers to primary centers. Persistent accessory ossification centers for the patella were seen in six instances. Clinically, these would be termed "bipartite patellae". Two individuals presented bilateral deformities (fig. 2a, b, and c; and 2866/172, not shown). The second patellae of the third and fourth individuals (17187/702, and 41049/714) were missing, and thereby prevented determination of the bilaterality of the process in these two persons. It is common clinical experience that bipartite patellae are most frequently bilateral, and that the accessory or secondary center is usually in the upper, outer pole of the patella.

There were two examples of congenital fusion of vertebrae (fig. 3a and b; and 41051/718, not shown). These fusions were through the lamina and facets posteriorly. Presumably, the normal segmentation of the somites of the fetus and subsequent union of the vertebrae did not take place in this portion of the vertebral column. There were numerous examples of fusion of vertebral bodies and the facets secondary to degenerative arthritis. In the two instances in which the fusion was thought to be prenatal in origin, the degenerative changes of the bodies and of the facets were too insufficient to be considered the cause of the fusion.

There were several other examples of fusion abnormalities. Figure 4 depicts fusion of ribs 1 and 2 caused by a prenatal genetic abnormality. There are arthritic changes about the rib heads. Such changes are secondary to the rib fusion which produced abnormal immobility of these ribs, incongruous motion between the heads of the ribs and the vertebral bodies, and incongruous motion between the rib necks and transverse processes of the vertebrae. Incongruous or asymmetric motion in a joint causes premature degenerative arthritis.

Fusion of three phalanges of the small finger, left hand is demonstrated in figure 5a and b. The absence of the normal configuration of the condyles and bases of the phalanges indicates fusion of prenatal or genetic disturbance rather than a fusion secondary to arthritis, injury, or disease. Failure of fusion of the transverse processes of the first and second sacral vertebrae can be seen in figure 6. There probably was a similar condition of the second and third sacral vertebrae since the lower portion of the second sacral vertebra does not have the appearance of fracture or erosion, but of separation through cartilaginous syndesmosis. Figure 7 reveals the presence of persistent accessory ossification for the odontoid which had united only precariously with the body of the odontoid. The two projections inferior to the facet joint surfaces (right larger than left) are pieces of clay holding the specimen for photography.

Additional specimens of fusion abnormalities not illustrated in this publication were: partial closure of the left coronal suture and six lumbar vertebrae (27247/708); fusion

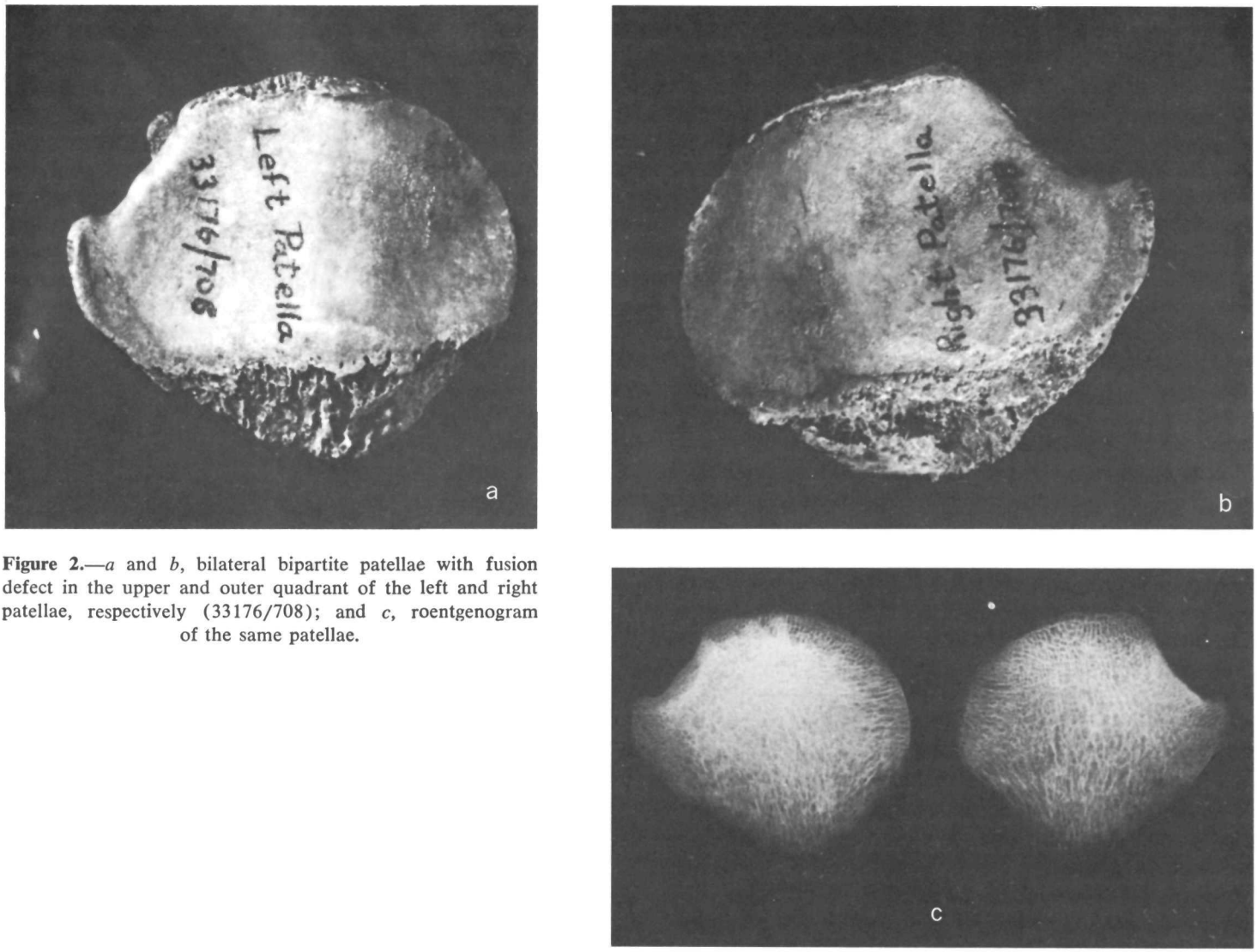


Figure 2.—*a* and *b*, bilateral bipartite patellae with fusion defect in the upper and outer quadrant of the left and right patellae, respectively (33176/708); and *c*, roentgenogram of the same patellae.

of the first ribs to the sternum bilaterally (33176/708); a bifid rib (41052/719); an accessory ossification center for the cuboid (33179/708); unilateral sacralization of the fifth vertebrae (36770/711); and sacral spina bifida (36771/711).

Miscellaneous abnormalities of prenatal disturbance were: an infant (25436/703) from Mug House with a 45° anteversion of the femoral necks—far more than normal for an infant this age; perforation of the olecranon fossa (36859/711); lateral bridge of C1, (36760/711); multiple vascular foramina in the isthmus of L5 (36761/711, 36770/711, 24033/703, 2866/172, 83895/710, 24030/703); perforation of the blade of the scapula (32890/710); omohyoid bone (13665/700); and division of the vertebral artery foramina (23694/703).

Of great significance was the total absence of the spectacular, generalized, skeletal abnormalities of prenatal origin. No examples of achondroplasia, osteogenesis imperfecta, osteopetrosis or dwarfism were found. Such diseases are of dominant inheritance, and had one case appeared in this population, many others would probably have been present also. It is also possible that cultural habits may account for the absence of generalized, dominant, fulminant abnormalities. Such infants often have a very limited life expectancy, and in modern times are kept alive only with heroic medical and surgical treatment. Some contemporary populations, aware of the limited life expectancy and great physical disability of these infants and children, continue to practice infanticide. Obviously, a group or tribe struggling for its very existence must have the active and positive contribution of each individual to this struggle. A deformed child or disabled infant would require the aid and assistance of many able-bodied adults, and would thus create a great deficit in the latter's contributions to the struggle for the group's existence. The remains of infants thus destroyed would quickly disintegrate since their bodies were largely soft tissue and cartilage. Very small osseous portions would also disappear. Other rare syndromes involving a generalized prenatal disturbance of cartilage and bone formation were not observed. Such an

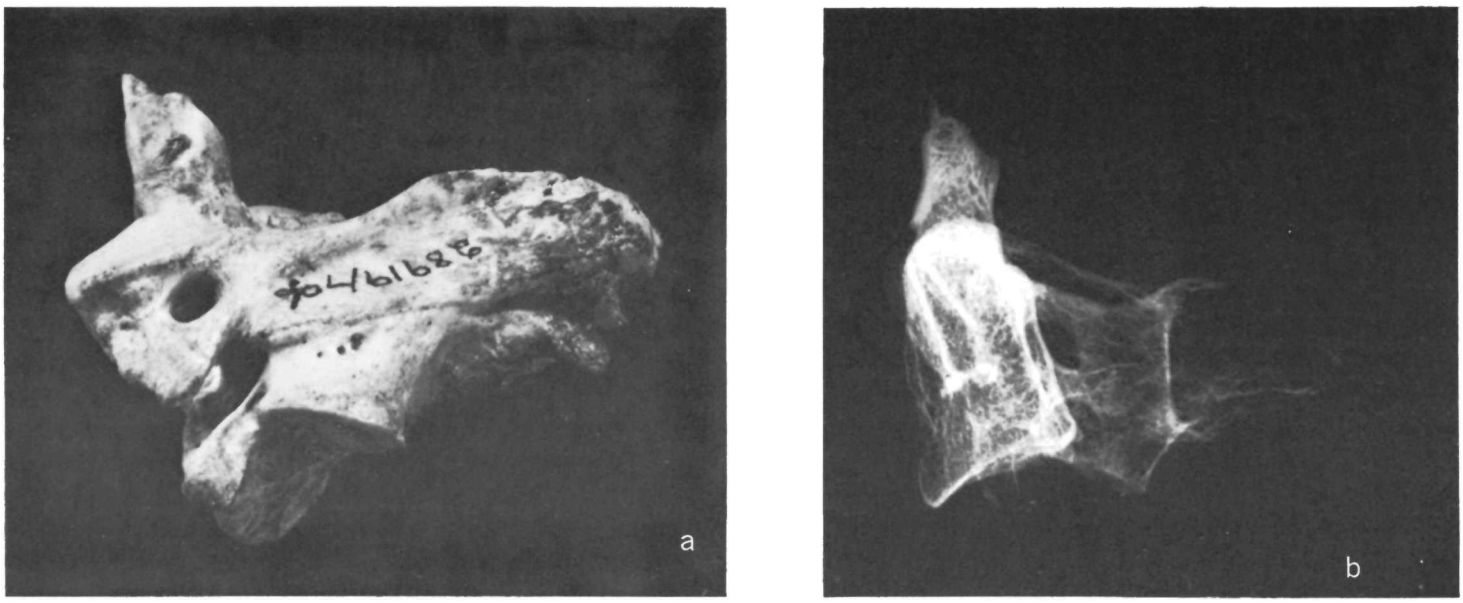


Figure 3.—*a*, Congenital fusion of cervical vertebrae 2 and 3 (28919/706); and *b*, roentgenogram of the same cervical vertebrae.

absence was probably due to the small number of individuals available for study. It would seem that recessively inherited traits would appear in increased numbers in a population which was so small and probably so inbred as this one. Occasional mutant abnormalities of genetic disturbance were notably absent, again probably due to the small number of burials.

Also of significance in this study was the absence of examples of congenital dislocation or dysplasia of the hip. It would thus appear that these problems were either very rare or were unknown to this group. This phenomenon is of great clinical interest since numerous medical studies now indicate an extremely high incidence of congenital dislocation or dysplasia of the hip in a number of present Indian tribes.

Current medical thought believes that congenital dislocation or dysplasia of the hip is caused by several factors of prenatal hormonal and environmental nature. Numerous studies reveal the high incidence of such hip problems in certain racial groups and populations, and their absence in others. The exact nature of the inheritance of this problem has not been satisfactorily determined because of the many diverse manifestations of hip abnormalities. Some of these are so minor that they escape detection clinically and are only evident in roentgenograms. The overwhelming preponderance of these hip problems in female infants suggests at least some hormonal influence on their causation and development. In addition, racial or familial customs may contribute greatly to the development of the problem. An example of a probably harmful practice is the use of the cradle board which causes immediate and full extension of the infant's hips. In contrast to this is the practice of carrying the infant on the mother's back in "piggy-back" fashion. The latter is believed beneficial since it places the hips in flexion and abduction.

Figure 4.—Congenital fusion of the two mid-thoracic ribs (13659/700).

Other factors of prenatal abnormality or growth nature are believed to influence the occurrence and severity of congenital dislocation and dysplasia of the hip. Current medical thought regarding anteversion of the femoral neck is of interest in this respect. The angle of anteversion is measured by sighting along the long axis of the femoral shaft and relating the bicondylar axis of the two femoral condyles to the longitudinal axis of the femoral neck. Normal present day infants (Indian and Caucasian) have an angle of anteversion of the femoral neck of about 25° to 30°. It decreases in a normal present day adult to less than 20°. It is also known that infants with congenital dislocation or dysplasia of the hip have an excessively high angle of anteversion. It is not known whether this increased anteversion is the cause or the result of the hip problem. Interestingly, a number of examples of this disturbance were seen in the Wetherill

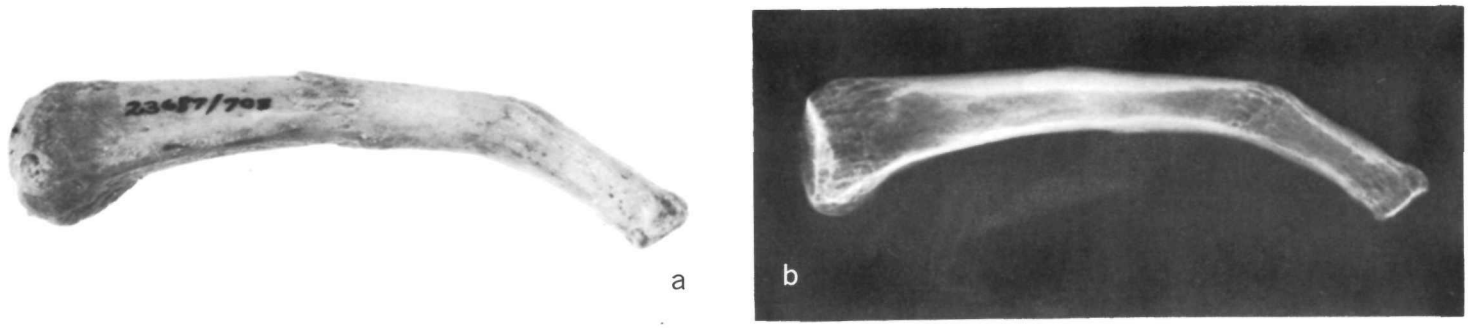


Figure 5.—*a*, Congenital fusion of three phalanges of the small finger, left hand (23687/703); *b*, roentgenogram of the same phalanges.

Mesa population. Since none of the individuals demonstrated dislocation or dysplasia of the hip, one might reason that this condition was the result of dislocation rather than the cause. Examples of excessive anteversion will be discussed in the section on disturbances of growth.

One of the mysteries of anthropologic literature now seems close to solution. Since the work of Hooton (1930, pp. 316–319) on the Pecos Indians, the term "osteoporosis", and more specifically, "symmetrical osteoporosis", has been an accepted diagnosis. Hooton essentially was describing a disease process which affected the skulls of infants and children. The disease destroyed the outer table of the skull and produced widening and thickening of the trabeculae of the diploe. Roentgenographically, this produced a sunburst-like effect of radiating trabeculae of the reactive bone. Hooton apparently chose the term porosis because of the destruction of the outer table of the skull. However, it appears quite obvious that the disease process is primarily one of the tissue of the medulla, and clinicians now consider the roentgenographic appearance to be quite typical of the erythroblastic anemias. These anemias are hereditary, probably of autosomal recessive inheritance.

Six examples of the erythroblastic anemia process were found in this population (41051/718, 32899/710, 33539/710, 33554/710, 24026/703, and 18352/703). One of these is illustrated in figure 8a and b. The irregular, patchy loss of the outer table of the skull of the infant with pitting of the surface is quite striking. The diploe are not enlarged, and the inner surface is normal. Their genetic nature is suggested by the fact that three occurred in Big Juniper House and two in Mug House. It would be very interesting to know the familial relationships of these five infants. It is quite probable that those in Big Juniper House were related; the two in Mug House might also have been related. It is also possible that the families of the two sets were related. The disease process was rather deadly since the six infants represent a rather high percentage of the infant burials found on Wetherill Mesa.

Disturbances of Metabolism

These disturbances are secondary to abnormalities of the metabolic state of the individual and the specific tissue. Generalized dietary excesses or starvation are difficult to evaluate in skeletal remains only. Hypertrophy of bone

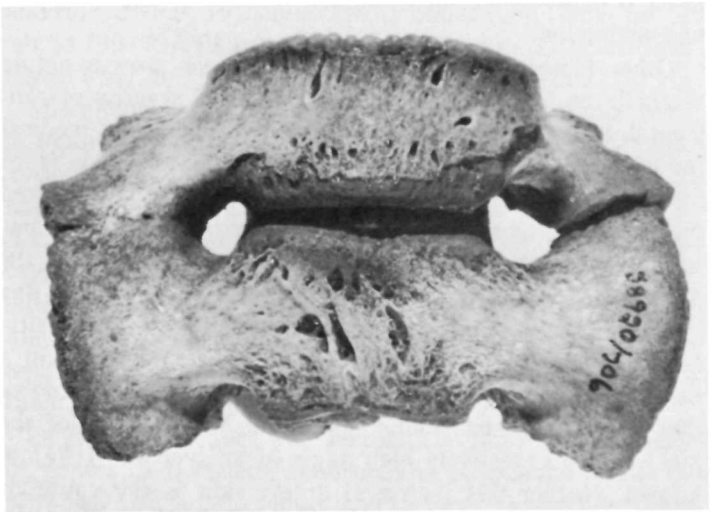


Figure 6.—Failure of fusion of the transverse processes of the first and second sacral vertebrae, (38920/706).

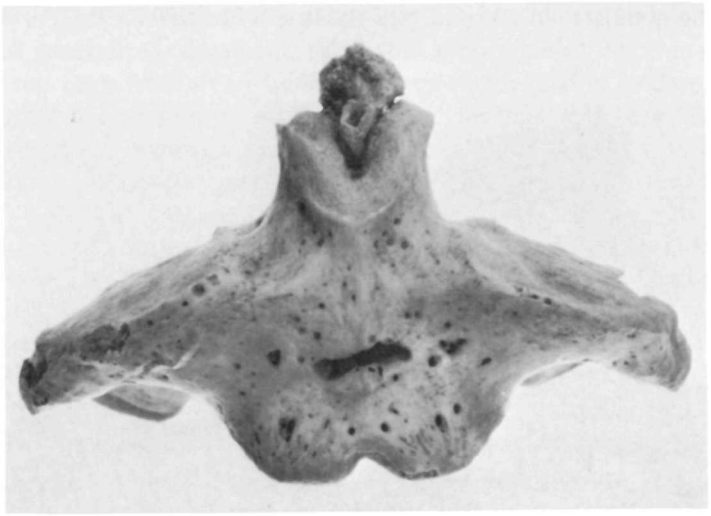


Figure 7.—Persistent accessory ossification center for the odontoid (35143/706).

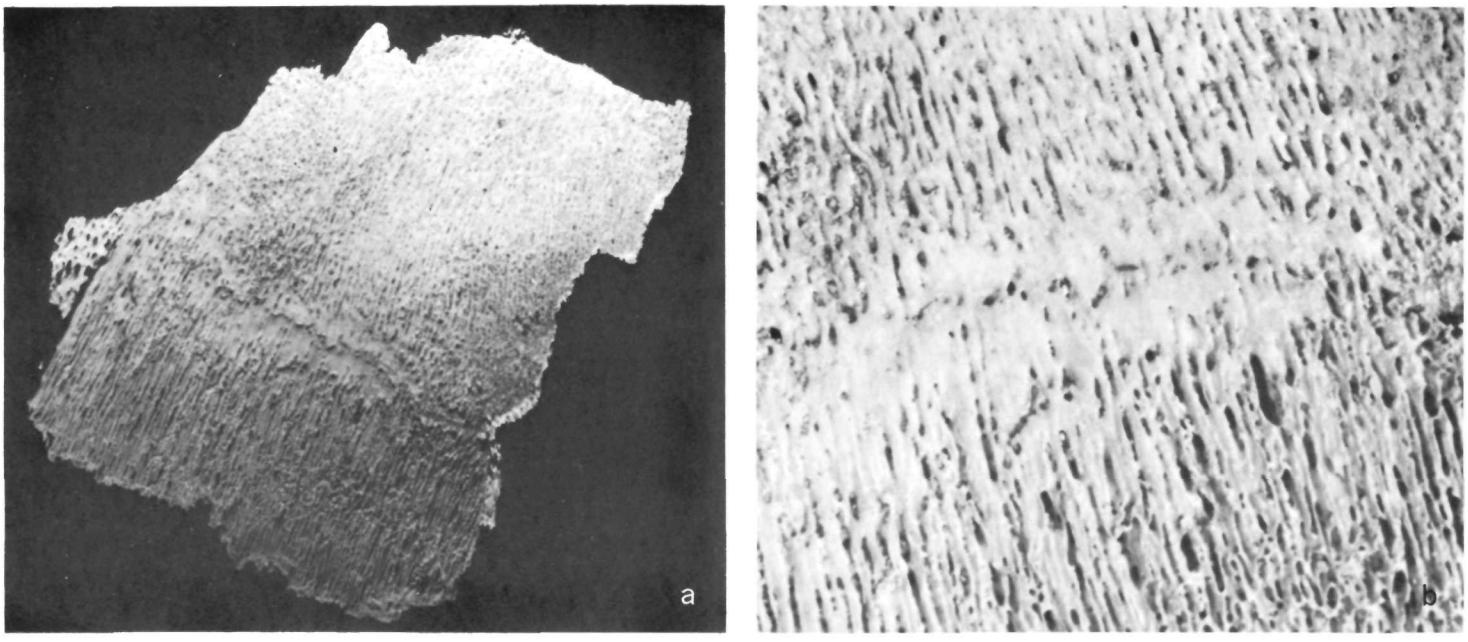


Figure 8.—*a*, Fragment of the parietal bone of an infant with erythroblastic anemia; and *b*, detail of the same bone fragment showing thinning and erosion of outer table of skull.

would provide evidence of obesity. However, the obesity would have to be of such great magnitude as to markedly increase the structural stresses on bone. Other factors such as increased muscle mass and heavy work would produce similar changes. In starvation, the bone appears to have a "high priority", maintaining its integrity while such soft tissues as fat stores and muscle mass are depleted. Starving persons are frequently referred to as being "skin and bones" —a tacit acknowledgment of this selective preservation of the two tissues.

Specific isolated deficiencies or excesses are often evident in the skeleton. Excess fluoride or lead ingestion is often easily detected grossly and radiographically in the teeth and skeleton. Other heavy metals, such as strontium and radium also are selectively stored in bone. The excessive intake of mineral water from a spring or spa often leaves these trace metals in the bone.

Other specific dietary deficiencies are due to lack of adequate vitamin intake, to hormonal imbalance, or deficiency in the endocrine system. Man is one of the few animals unable to synthesize vitamin C. Therefore, deficiency of intake of this vitamin will result in scurvy. Vitamin deficiencies are frequently multiple due to starvation or deprivation. Ancient sailors on long voyages were frequently deprived of many vitamins, and in addition to scurvy, became ill with beri-beri (vitamin B deficiency), and osteomalacia (vitamin D). All of these deficiencies may be detected in the skeleton. Hormone deficiency or imbalance is more difficult to detect, for there are frequent checks and balances for the endocrine system. Thus, one hormone may have a specific or primary function with regard to bone, but in the deficiency of this hormone, others may have a like secondary influence, and the original primary deficit be undetectable. Additionally, one hormone deficiency due to one endocrine glandular dysfunction would lead to complex, interrelated defects which would be very confusing even with present day sophisticated hormone assay techniques. Nevertheless, we can state that hyperthyroidism, hyperparathyroidism, hypercortisonism (adrenal), and estrogen deficiency of sufficient magnitude produce a rather nonspecific osteoporosis. The reverse endocrine disturbances, however, do not usually produce osteosclerosis (the opposite change in bone). We should be able to detect the osteoporosis of the former disturbances, but would only have to guess which endocrine gland was involved by secondary inferences. For example, estrogen deficiency in a postmenopausal female might be recognizable. Similar estrogen deficiency in an adolescent girl would result in the persistence of a pelvis with male configuration. Such an estrogen deficient adolescent girl might be indentified as an adolescent male because of the shape of her pelvis.

It is significant that no evidence of metabolic disturbances of bone secondary to endocrine gland dysfunction or vitamin deficiency was observed in any of the skeletal remains. The most common, present day metabolic bone disease is osteoporosis, either of a localized or a generalized nature. Osteoporosis is usually a roentgenographic diagnosis based upon diminished density of bone to the x-ray. About 25 percent of the bone mass must be absent before roentgen evidence will be significant. Considerable variation in the roentgenographic density of these bones was noted, but it was not felt to be of significance because of appreciable variations in preservation. Variation in densities of the bones of a single skeleton might be of significance. However, vary-

ing exposure of the skeleton's different parts to the elements could produce such density changes. Localized, individual density changes in the bones might indicate disuse, trauma, infection, or the residuals of infantile paralysis. If the osteoporotic changes are severe and generalized, secondary changes in the bone such as fracture or deformity would indicate the presence of osteoporosis.

One example of generalized osteoporosis was present in this series. This burial, Number 24, of Long House, was simply catalogued as an "old man". This skeleton was in a good state of preservation. Severe degenerative arthritis was evident in the spine and many peripheral joints (fig. 9a). The photograph is of the twelfth thoracic vertebra and the five lumbar vertebrae. The arthritis of the vertebral column had produced complete ankylosis with bridging across the intervertebral discs of the twelfth thoracic vertebra and the five lumbar vertebrae on the right side, but circumferential bridging of the bodies by osteophytes was absent on the left side (fig. 9b). The photographs and roentgenograms demonstrated the degree of the degenerative process and the porosis of the vertebral bodies. The osteoporosis was best seen in the roentgenogram (fig. 9c). The lumbar discs were enlarged and produced biconcavity of the adjacent cortical surfaces of the vertebral bodies (ballooning of the discs). The first lumbar vertebra was compressed anteriorly, thus indicating a pathologic fracture secondary to the osteoporosis. Moderately advanced degenerative changes were apparent in the facet joints posteriorly and in the sacro-iliac joints. Neither the facet joints nor the sacro-iliac joints demonstrated the degree of degenerative arthritis that was apparent about the vertebral bodies anteriorly. The sacro-iliac joints did not demonstrate spontaneous ankylosis. Degenerative arthritis changes were seen in the major peripheral joints, including the hips, knees, ankles, feet, and elbows, and a minimal central protrusion of the acetabulum was seen on the right side.

The type of arthritis present in Burial Number 24 is a matter for conjecture. That the visible roentgenographic changes represented an advanced degenerative process can not be questioned, but such arthritic changes might have resulted from a "mixed" arthritis. The most common mixture would be composed of rheumatoid arthritis or rheumatoid spondylitis, and secondary degenerative arthritis. The extent of the porosis in this old man and the rather striking limitation of the porosis to the spine with subsequent ankylosis would support the diagnosis of rheumatoid spondylitis. The lack of involvement of the fingers and toes, which is frequently the case with rheumatoid spondylitis, also favors such a decision. Other findings in this individual, however, make the diagnosis of rheumatoid spondylitis rather doubtful. One of the common physical abnormalities of rheumatoid spondylitis is limitation of respiratory function secondary to ankylosis of the joints between the vertebrae and the ribs.

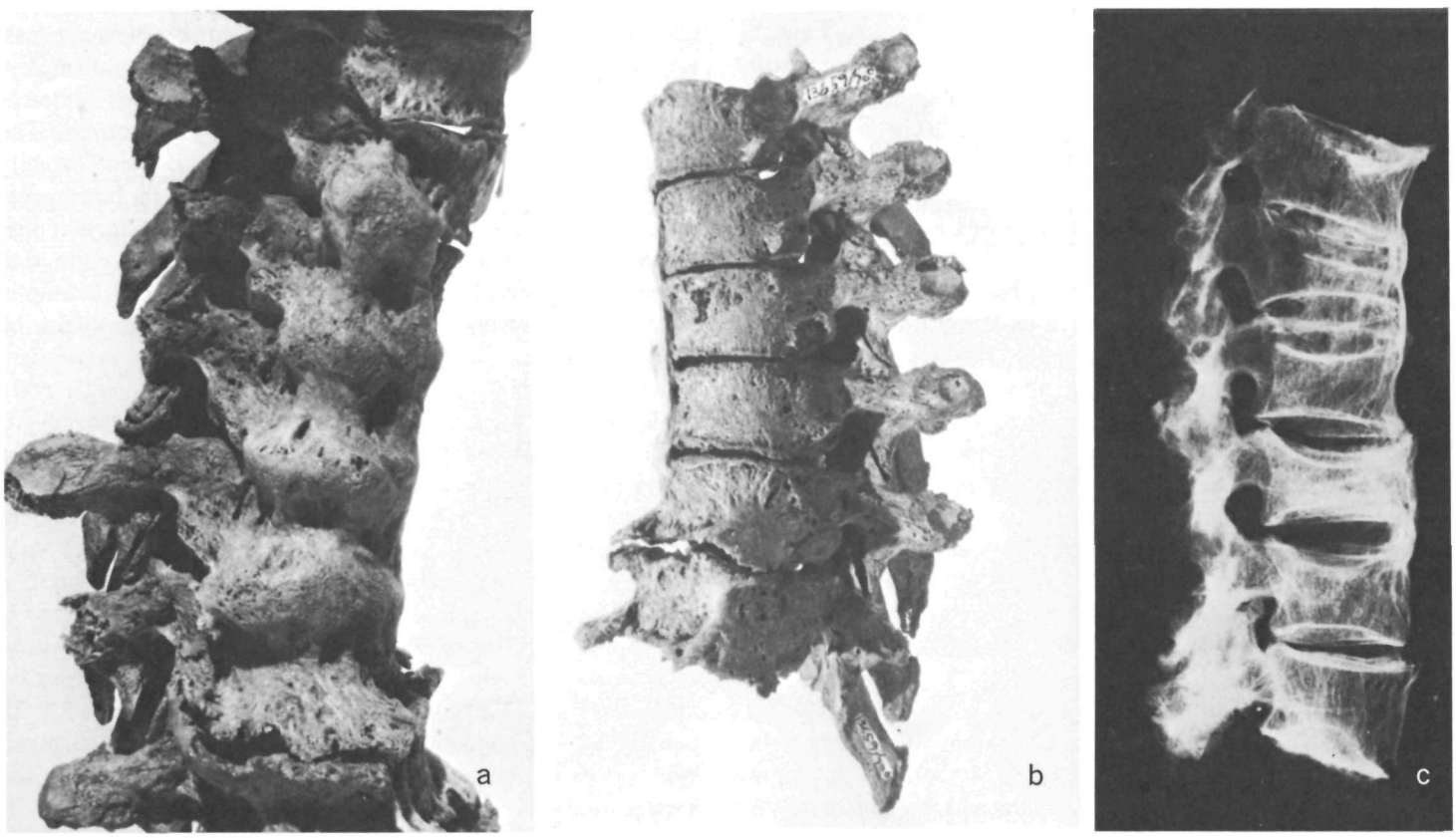


Figure 9.—*a*, Advanced arthritis of the thoracic vertebra, T12, and all five lumbar vertebrae as seen from the right side (13659/700); *b*, osteophyte bridging not extending completely around the vertebrae (left side); *c*, roentgenogram of the same thoracic and lumbar vertebrae (right side).

It would be anticipated that such ankylosis would have been present in this individual had this been rheumatoid spondylitis, particularly in view of the extent of the osteophyte formation laterally in the thoracic spine. Fragments of the rib heads and necks would have remained fused to the vertebrae. Commonly, ankylosis of the sacro-iliac joints is also evident in males with ankylosing rheumatoid spondylitis. Arthritic involvement of knees, ankles, subtalar joints, and elbows is unusual in pure rheumatoid ankylosing spondylitis.

A diagnosis of primary rheumatoid arthritis was likewise difficult to support. The sex of the individual, the absence of involvement of the hands and feet, the more extensive spinal changes, and the absence of porosis of the major long bones of the upper and lower extremities favor a diagnosis other than rheumatoid arthritis. It would thus appear that the more defensible diagnosis would be osteoporosis associated with simple degenerative arthritis peripherally. The course of events would have been: severe degenerative arthritis of the peripheral joints leading to great disability; the disability of the extremities leading to markedly decreased activity producing osteoporosis of the spine; the osteoporosis of the spine leading to pathologic compression fracture of the vertebral bodies, and the fractures leading to abnormal spinal mechanics which in turn accelerated the degenerative arthritis of the spine.

Other causes of the porosis in this individual must be considered. It could well be the osteoporosis of senility in view of the old age of the individual. It probably was not the osteoporosis of endocrine origin since this is almost exclusively seen in the postmenopausal female. There is great argument as to whether or not there is a male menopause. There were no other evidences of debilitating diseases or processes in this old man. We are left with the simple conclusion that the elderly male (Burial 24 of Long House) had severe osteoporosis which resulted in compression fracture of the lumbar vertebrae ballooning of the discs, and probably secondary degenerative arthritis of the spine with a rather more advanced degenerative arthritis of the peripheral joints.

No evidences of vitamin deficiencies such as rickets or osteomalacia were seen in this study. A similar lack of these deficiency diseases was noted in the studies of Hrdlicka (1908, pp. 82, 190) and Hooton (1930, pp. 317–320). It would appear that these ancient Indians probably had diets adequate in vitamin D or its precursors. Man is able to synthesize vitamin D from its precursors in the presence of adequate sunlight—an ingredient abundantly present on Mesa Verde.

Disturbances of Growth

Because growth is such a remarkable characteristic of man, and because such growth from a single celled organism to an extremely complex system of organs takes place in a few years, many disturbances may take place with devastating results. It is most remarkable that so many humans develop "normally". Any abnormality in growth may produce simple size variation, deformity or faulty organ specialization. Localized or generalized growth disturbances may be present.

Some of the factors which influence growth are disease and trauma. Since growth and specialization are much more rapid in the fetus, the effect of a disease or injury to a pregnant female may produce spectacular or devastating results in a fetus. The familiar blindness of the fetus produced by rubella in the pregnant female is well known. It is also known that this disease must be in the first trimester of pregnancy when the eyes are in the stage of differentiation.

The most frequent abnormalities of growth found in this group were rotational deformities about the long axis of the lower extremity. Excessive anteversion of the femoral neck was present in thirteen individuals. The anteversion of the femoral neck is determined by relating the long axis to the femoral neck of the bicondylar axis of the femur. It is measured by examining the femur from either end, essentially viewing it along the long axis of the shaft. The angle of the anteversion of the femoral neck is not to be confused with the angle of inclination. The angle of inclination is the angle produced by the long axis of the femoral neck and the long axis of the femoral shaft. This angle is measured by examining the femur in the anterior-posterior projection. Increase in the angle of inclination is called coxa valga; decrease in this angle is called coxa vara.

Current adult populations (Indian and Caucasian) demonstrate an angle of anteversion of the femoral neck of about 18° to 20°. Any angle over 25° in the adult is abnormal. Infants have an even greater degree of anteversion with 30° not being abnormal. The angle of anteversion thus decreases with growth, and the adolescent child decreases the angle of anteversion to equal that of the adult.

The greatest degree of anteversion of the femoral neck found in this study was in a female teenager, 32883/710, of Big Juniper House. The anteversion of each of her femoral necks measured 50°. Figure 10 demonstrates normal and

Figure 10.—Examples of normal (33176/700) and excessive (13639/700) anteversion of the femoral neck.

excessive anteversion of two femoral necks. The femur on the left demonstrates a normal anteversion of 20° while the femur on the right presents an anteversion angle of 35°. Ten of the 13 individuals with excessive anteversion were adults, one was a teenager, another a child of nine, and the last was an infant. It is apparent that the normal decrease of the degree of anteversion did not occur in a significant number of these persons. None of these individuals demonstrated dysplasia of the hip despite their persistent excessive anteversion. The frequent association of excessive anteversion of the femoral neck and dysplasia of the hip is commented upon in the section on prenatal disturbances.

Abnormal external rotation of the distal tibia was noted in nine instances. The angle of external rotation is determined by viewing the tibia from either end along its long axis. The relationship of the biocondylar axis is measured against the axis of the distal end of the tibia. However, there are no good landmarks for the measurement of such axes, and therefore, an angle of external rotation is not as accurately determined as is the angle of anteversion of the femur. It is generally conceded that 20° of external rotation of the tibia is within normal limits, and anything over 25° is abnormal.

Excessive external torsion of the distal end of the tibia was noted in nine instances. The greatest degree of external torsion of the tibia was 45° bilaterally, (fig. 11). Small remnants of articular cartilage remain on the distal tibial surface. The medial malleolus appears enlarged, but the bone otherwise appears normal. Since many of these skeletons were quite complete, it was possible to describe the relationship between excessive external torsion of the tibia and excessive anteversion of the femoral neck. Three of the individuals with excessive femoral neck anteversion also had excessive external torsion of the tibia. Ten of the persons with excessive anteversion of the femoral neck had no increase of external torsion of the tibia. Four of the nine individuals with excessive external tibial torsion had normal femoral anteversion angles. In two of the individuals with excessive external tibial torsion, the femoral neck anteversion angle could not be measured because of postmortem damage to the femur or because of the absence of the femur. It would thus appear that there is no relationship between the two abnormalities.

External torsion of the tibia is a puzzling abnormality. It is frequently thought to be secondary to muscle imbalance that follows a neurologic disease such as poliomyelitis. However, rather extensive residuals of the disease must be present in a child with rather severe muscle imbalance in order to produce the torsional deformity. It is also thought that it might be the result of posture such as squatting or sitting crosslegged. If such were the case, the incidence of external torsion would then be almost universal in the population. The rarity of this abnormality suggests, however, that it was a true growth deformity, and not simply due to cultural habit.

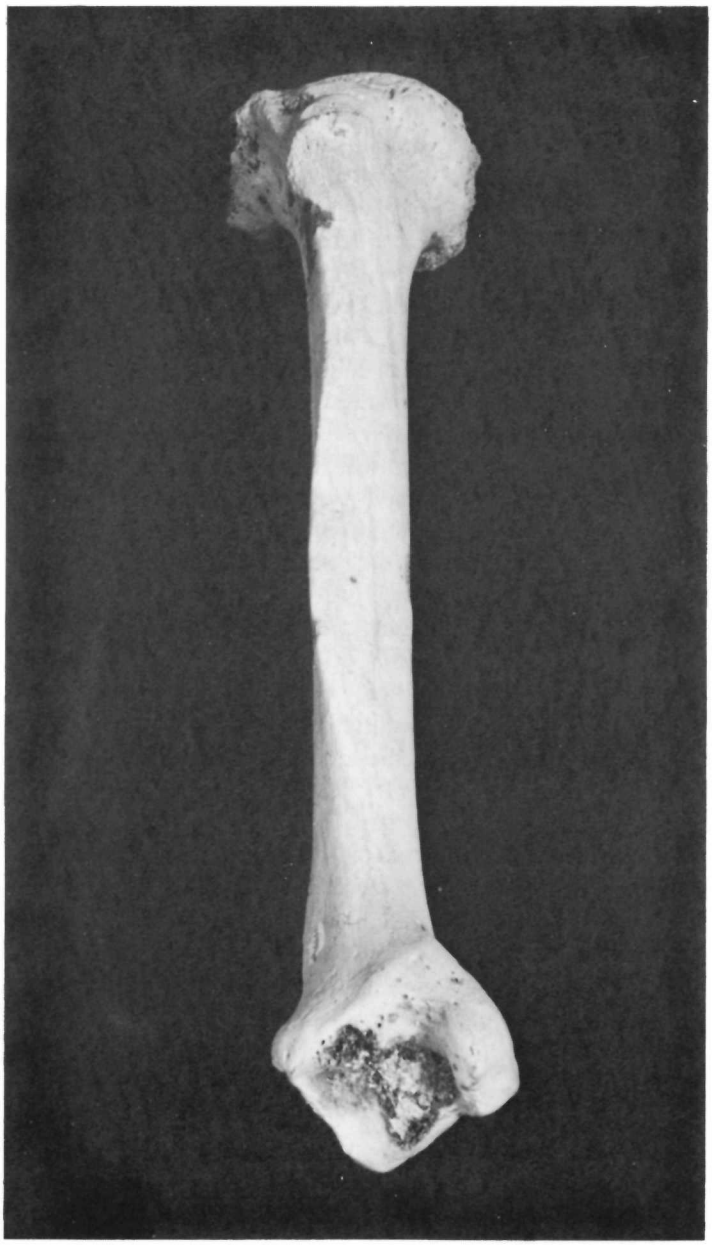


Figure 11.—External torsion of the tibia (19494/700).

Another example of rotational disturbance is the relationship of the angle of the neck and body of the talus. This angle is measured by viewing the talus from its superior surface. The angle is measured by the intersection of two lines representing the long axis of the body of the talus and the long axis of the neck of the talus. Fifteen degrees of medial deviation of the axis is accepted as normal. The neck-body angle of the talus measured at 38° in one individual (fig. 12). The talus had articulated with a tibia (shown in fig. 11) with 45° of external torsion. The two deformities tended to compensate for each other.

The one example of an increased angle of diversion of the body neck of the astragalus found at Mesa Verde is of interest in the light of a report by Irani and Sherman (1963,

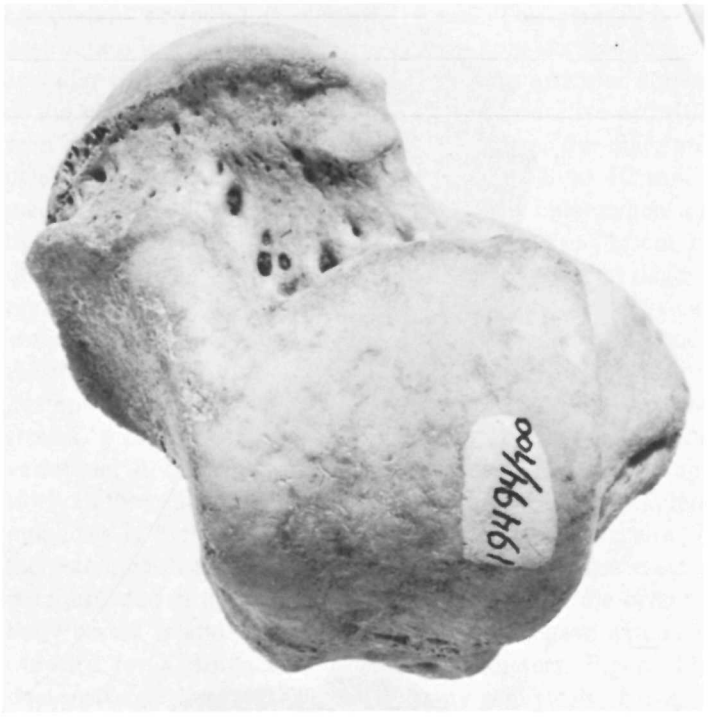


Figure 12.—Increased angle of divergence of the talar neck and body (19494/700).

pp. 45–52) linking such an abnormality with club foot deformity (talipes equinovarus). Such an increase in the neck-body angle was the only consistent abnormality found in the examples of clubfeet dissected by the two researchers. The Mesa Verde individual did not demonstrate any other osseous deformities of clubfeet, and it must therefore be assumed that this sole example merely represented an isolated growth deformity.

No instance of the presently common varus and valgus deformities of the extremities was noted. Talipes equinovarus, metatarsus adductus, genu vara or genu valga, and coxa vara or valga were not seen. Most deformities of the feet are primarily the result of soft tissue contractures. Bony deformities are not evident until such contractures have been present for a long time. Skeletal evidence of such abnormalities may not have appeared because of high infant mortality. The other currently common growth abnormalities in both Indian and Caucasian adolescents, such as idiopathic scoliosis (spinal curvature) and epiphyseolysis (slipped capital femoral epiphysis), were also notably absent.

Degenerative Processes

All tissues of the body degenerate or wear out with time or excessive usage. However, the degeneration proceeds at markedly different rates in different individuals. Some persons may show early degeneration of the arteries with resultant myocardial infarction or cerebral thrombosis. Other persons may show the earliest degenerative signs in the skeletal system. In this system, the cartilage wears out much more rapidly than the bone, and with more disabling consequences. The bone attempts to repair the joint damage, and degenerative arthritis results.

The incidence of degenerative arthritic disease is quite striking in the Wetherill Mesa people. Such arthritic changes varied from small marginal osteophytes to spectacularly large osseous bridges across joints. The most common joints involved were those of the vertebral column. The incidence of degenerative arthritis of the facet joints equaled the incidence of hypertrophic spurs about the bodies of the vertebrae. No distinction could be made between the incidence of facet arthritis and osteoarthritis of the vertebral bodies ("osteoarthrosis"). The degenerative process produced a solid osseous fusion about a number of intervertebral discs in quite a few of the individuals. Fusion of the lamina and the facet joints posteriorly, caused by osteophyte formation, was present in all of the individuals with interbody fusion. Thus, it would appear that there is no justification for the attempt to separate osteophyte formation anteriorly between the vertebral bodies and about the intervertebral discs from osteophyte formation posteriorly about the facet joints and the laminae. All changes apparently represent the same degenerative process.

The following joints involved in degenerative arthritis are listed in order of frequency: spine, 58; knee, 12; shoulder, 12; hip, 11; temporo-mandibular, 4; subastragalar, 4; ankle, 4; elbow, 3; sterno-clavicular, 1; interphalangeal joints of toes and fingers, 8. The numbers are probably not significant in themselves since they include observations on the miscellaneous bones and incomplete skeletons. Other joints almost surely would have been involved had the skeletons been complete. The numbers would have been significantly altered since most persons experience degenerative changes in more than one joint.

Most significant, however, is the high incidence of degenerative arthritis in individuals over the age of 35. Of 28 such individuals, 23 demonstrated degenerative arthritis with the spine involved in almost every case. The absence of degenerative change in four of the other five individuals (28909/706, 28913/706, 28916/706 and 28922/706) could not be documented because they presented insufficient skeletal material. The vertebrae in these four were either absent, or the articular surfaces of the knees, shoulders, and hips were destroyed by post-mortem changes. The fifth individual over the age of 35 without arthritis was estimated to be a 45-year old male (34634/703). His skeleton was nearly complete, and no arthritis was found in the spine, hip joints, and shoulder or other peripheral joints. He was apparently an exception in a population with an almost 100 percent rate of degenerative arthritis by the age of 35. In addition, many younger individuals also possessed rather severe degenerative changes.

Minimal marginal osteophyte formation is seen about the glenoid fossa of the scapula in figure 13a. These osteophytes

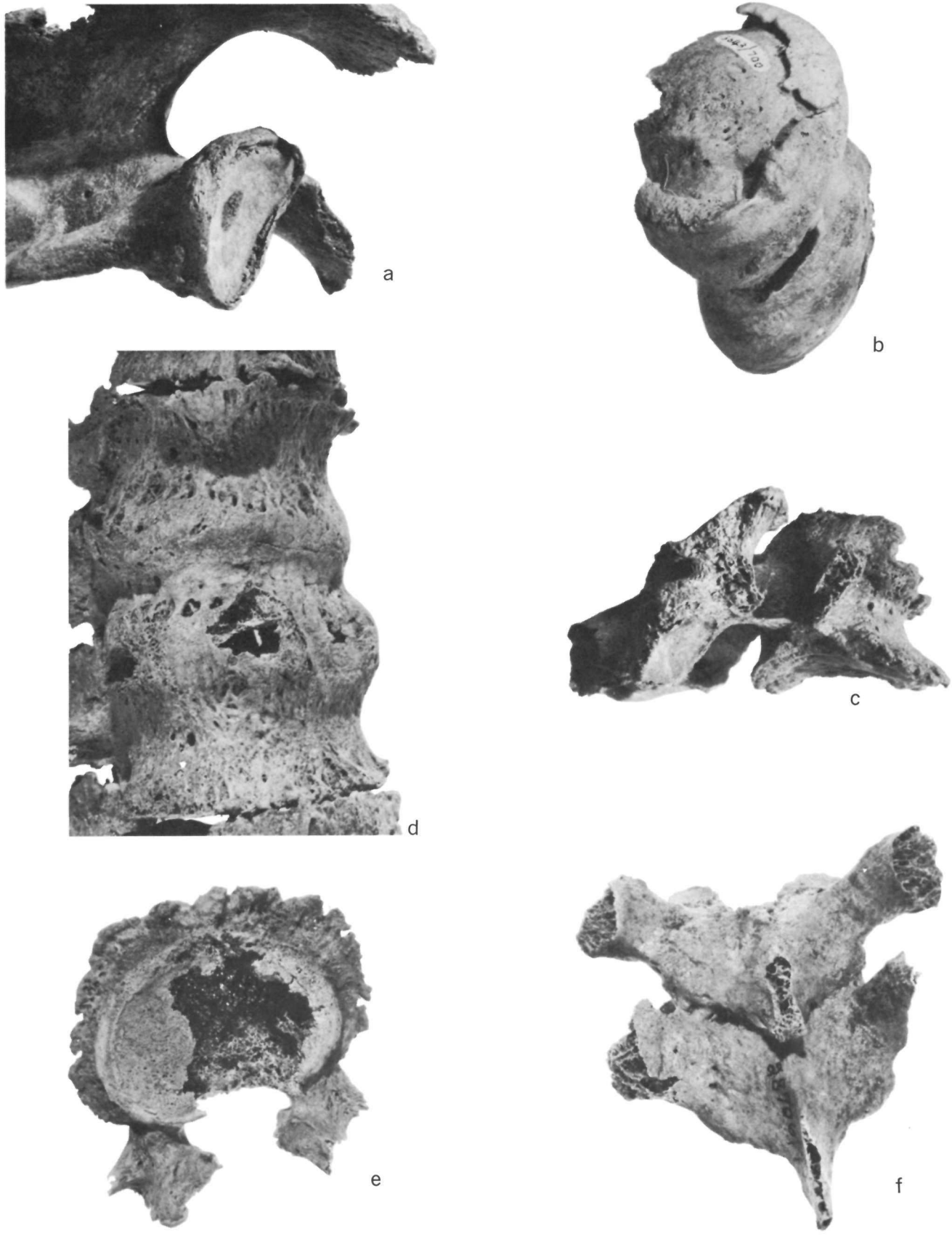


Figure 13.—*a*, Minimal degenerative arthritis of the glenoid (13643/700); *b*, degenerative arthritis of the femoral condyles (13643/700); *c*, degenerative arthritis of cervical spine (13643/700); *d*, degenerative arthritis of the lumbar spine (13659/700); *e*, degenerative arthritis of the lumbar vertebrae (28062/706); *f*, degenerative arthritis with fusion of two thoracic vertebral lamina (28906/706).

completely surround the glenoid fossa. The central bone destruction in the glenoid fossa points to considerable loss of articular cartilage resulting in damage to the articular cortex of the glenoid. More spectacular is the degenerative arthritis seen about the knee joint in figure 13b. Here, the marginal osteophytes about the femoral condyles are 8 to 10 millimeters wide and have produced considerable enlargement of the joint itself. The marginal osteophytes are also present in the intercondylar area of the femur. More advanced degenerative arthritis is seen in figure 13c. Here, the two cervical vertebrae are solidly fused together. Fusion anteriorly about the intervertebral disc is solid and is matched by the fusion posteriorly through the laminae. Figure 13d also demonstrates massive osteophyte formation about the lumbar vertebrae. A quite solid ankylosis has been produced anteriorly in the central three lumbar vertebrate, while incomplete ankylosis is present above and below the area. The size of the osteophytes about some vertebral bodies is more clearly demonstrated in figure 13e. Here, the contour of the original body cortex is apparent, and the osteophytes have extended outward for a distance of 6 to 10 millimeters. Figure 13f demonstrates the equally solid bony ankylosis between lamina of two thoracic vertebrae. The similarity of the interlaminal body fusion posteriorly with the fusion about the intervertebral disc anteriorly is clearly visible. Damage to the processes is post-mortem. The left lamina of the inferior vertebra has a number of perforating vascular foramina.

Commonly accepted theory holds that degenerative arthritis most frequently affects individuals living in cold and damp climates. Conversely, it is believed that warm, dry climates exert a beneficial effect. No support for this opinion is found in the statistics of the degenerative changes of the Wetherill Mesa populations. The incidence of degenerative arthritis was as high in those individuals who lived on the sunny mesa top as it was in those Indians who lived within the alcoves. It is probable that the latter worked on the mesa top and were able to enjoy essentially the same climate as those Indians who lived primarily in the mesa top pueblos. In addition, one cannot categorically state that warm, dry climates decrease the incidence of degenerative changes, but that they merely decrease the symptoms which are produced by these changes.

No statement can be made about the incidence of degenerative processes in the soft tissues since none of these tissues were preserved. It is not known if the Indians suffered from bursitis, tendonitis, or fasciitis. There is, however, indirect evidence of degenerative changes in the intervertebral discs. The normal aging process of the human intervertebral disc includes loss of distinction, grossly and microscopically, between the nucleus pulposus and annulus fibrosus; dehydration of both portions of the disc; and subsequent loss of vertical height of the disc. This latter change would be grossly and roentgenographically evident as a narrowing of the disc space, subluxation of the facet joints, and approximation of the spinous processes (kissing spines).

An example of such a change is seen in Burial 12 of Long House. Evidence presented by the posterior elements of the two lumbar vertebrae suggests just such a sequence of events. The degeneration of the disc produced narrowing of the disc space and subluxation of the facet joints. This was evident in the erosion of inferior lamina by the projecting inferior articular process of the superior vertebra, and the corresponding oblique erosion of the two spinous processes resulting from contact between the spines. The posterior elements under discussion have been photographed in three different positions to better illustrate the processes of degeneration and secondary changes. In figure 14a, the facet joints are in a normal position with normal spacing between the lamina and spinous processes. Normal intervertebral disc height and stability are assumed. The superficial erosion of the more superior portion of the inferior laminae is evident. In figure 14b, subluxation of the facet joints is minimal, but the approximation of the posterior elements is already apparent. The vertebrae would assume this position with minimal loss of intervertebral disc height and integrity. In figure 14c, the

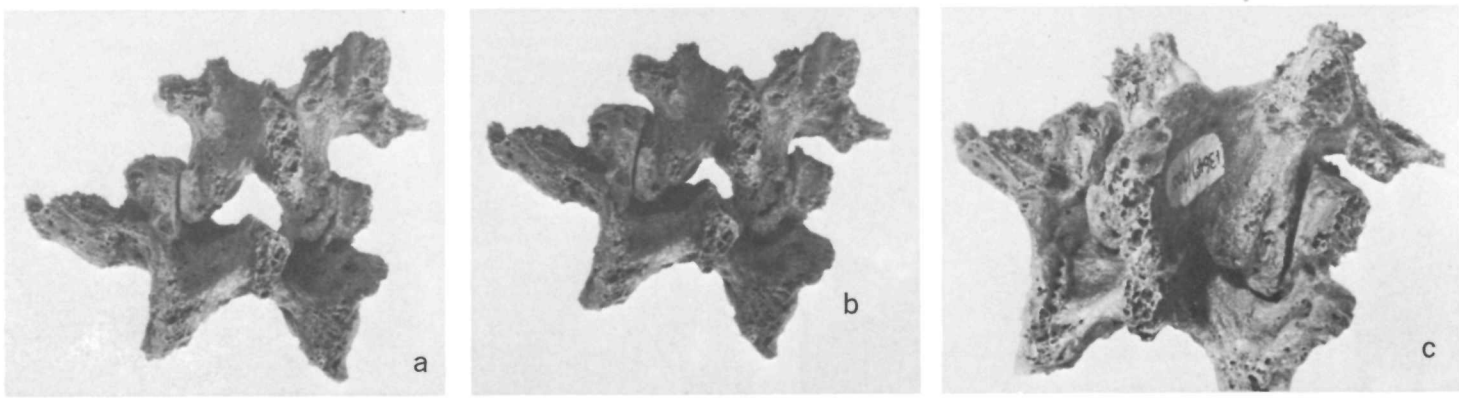


Figure 14.—*a*, Lumbar process with normal articulation of the facets (13647/700); *b*, minimal subluxation of the same lumbar facets; *c*, more marked subluxation of the same lumbar facets.

subluxation is more marked, and contact of the posterior elements is evident. This position would only be found with almost complete loss of disc material. The gliding of the inferior articular processes of the superior vertebra has quite obviously produced the superficial erosion of the inferior vertebral lamina. Movements of flexion and extension of the individual would have accentuated the erosions.

Six individuals demonstrated mild osteophyte formation and degenerative change over the medial aspect of the metatarsal head of the great toe, changes which are almost always associated with bunion formation. Figure 15a presents a dorsal view of the two great toes of a single individual showing the metatarsals and proximal phalanges. The valgus angulation of the phalanges—slight to moderate in the right toe, and moderate in the left—is readily seen. A lateral view (fig. 15b) of one of the metatarsals shown in the preceding figure demonstrates the osteophytes of degenerative arthritis on the medial surface of the metatarsal head. The osteophytes extend proximally on the head and indicate the presence of an overlying bursitis—the bursa of a bunion. Figure 15c presents the same great toes from the medial looking laterally. The degenerative arthritic osteophytes are well demonstrated on the dorsal and plantar surfaces of the metatarsal heads and the plantar aspects of the bases of the phalanges. In all probability, this individual demonstrated hypertrophic bursae over the medial aspect of the metatarso-phalangeal joints of the great toes. The degenerative process of the metatarsal heads was secondary. The presence of such bunion formation would be of great clinical interest since almost surely these individuals did not wear rigid, constrictive, and pointed shoes that are so commonly implicated in the formation of bunions in present day populations.

Infections

Clinically, infections of bone and joints result from invasion by two types of organisms: pyogenic or pus forming organisms, and granulomatous organisms. The pyogenic organisms produce pus as a result of cellular or tissue necrosis from bacterial toxins. The granulomatous organisms do not have such toxic action, and the host tissues react by producing granulation tissue or "proud flesh". Examples of pyogenic bacteria are the staphylococci and streptococci. Granulomatous organisms are the tubercle bacillus, syphilis, and the fungi. Infections of bones are called osteomyelitis. They may be exogenous in nature, i.e., the bacteria may enter the bone directly from the outside as in a compound (open) fracture, or they may be hematogenous in nature and enter the bone via the blood stream. Osteomyelitis is most common in the metaphysis or diaphysis of the bone. Infection of a joint is termed arthritis and may either be pyogenic or granulomatous. Tuberculosis, in almost 99 percent of the cases, is tuberculous arthritis. Syphilis is a granulomatous infection of the diaphysis in almost all cases. These infections had a grave prognosis in the days before chemotherapy. Hematogenous infections commonly involved many bones in infants and children, and often they were lethal.

Only five instances of bone and joint infections were found in this survey. There were two classical examples of pyogenic osteomyelitis of the femur. Figure 16a, b, and c shows an osteomyelitis of the distal metaphysis of the right femur with sinus, sequestrum, and involucrum formation. The second example (not shown), Burial M3, Mug House (28884/175), was a female adult with osteomyelitis of the proximal left femoral shaft. Both of these infections present gross and roentgenographic appearances of chronic osteomyelitis.

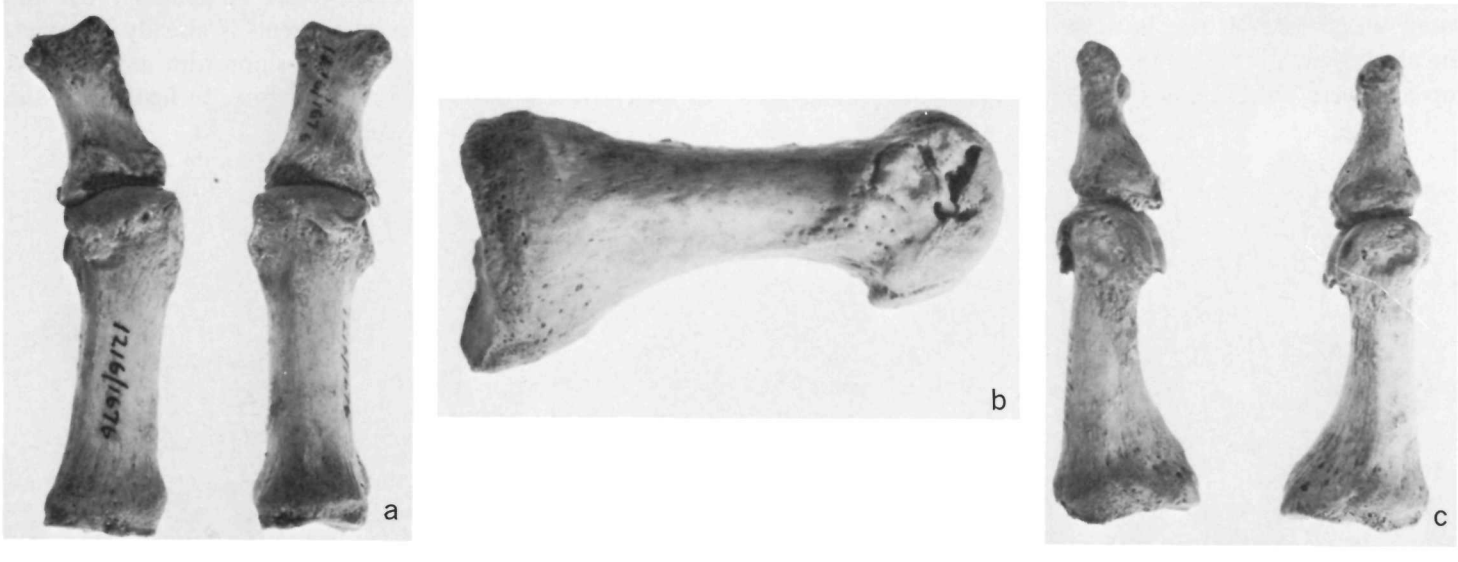


Figure 15.—*a*, Dorsal view of the articulated metatarsals and proximal phalanges of the two great toes (41050/714) showing the hallux valgus deformities of "bunions"; *b*, lateral view of the metatarsals in figure 14a demonstrating osteophytes of degenerative arthritis and bunions; *c*, view of the articulated metatarsals and phalanges of the two great toes shown in figures 14a and b.

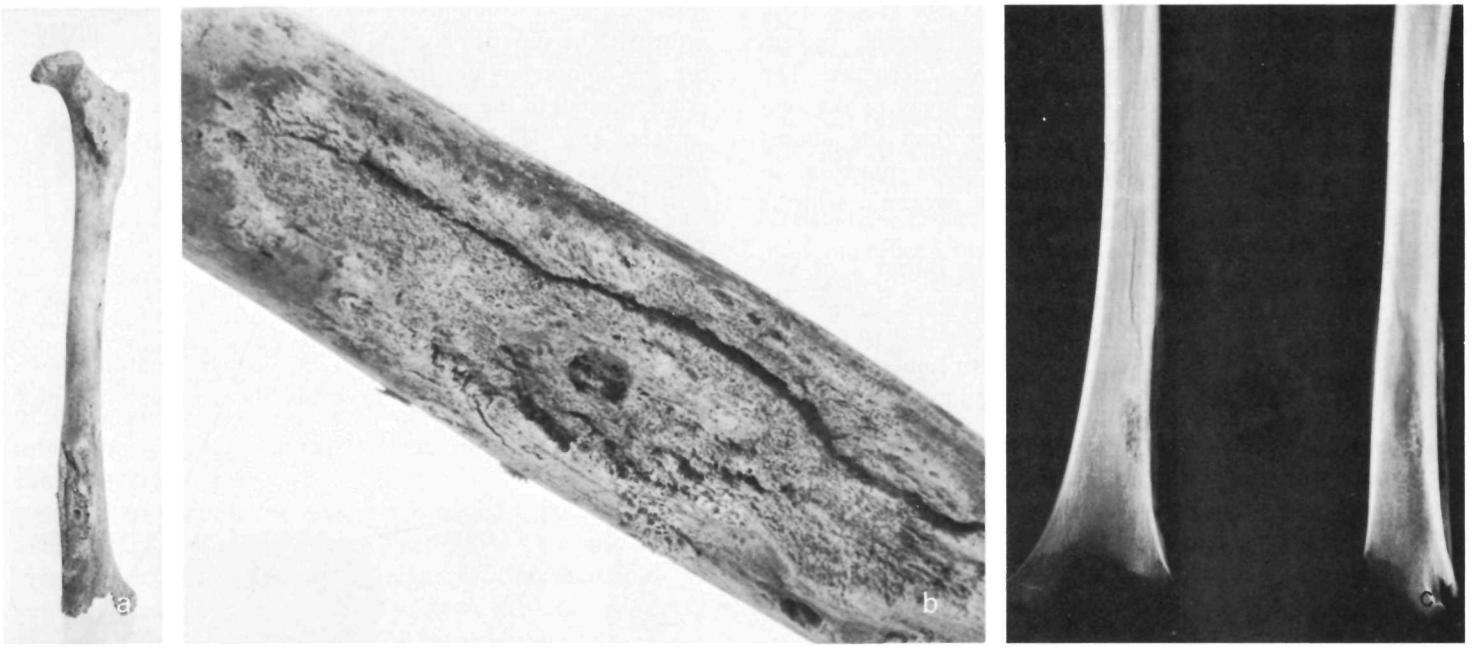


Figure 16.—*a*, Pyogenic osteomyelitis of the distal femoral metaphysis (20069/700); *b*, detail of the area of infection; *c*, roentgenogram of the same femur.

The third example of infection, in a 37 year old male from Badger House (fig. 17a and b), was protrusio acetabuli, i.e., protrusion of the floor of the acetabulum into the cavity of the pelvis. It is probable that this represents a pyogenic infection of the hip joint, rather than a granulomatous infection such as tuberculosis or syphilis. Tuberculous infection of the hip joint is quite common. However, tuberculosis usually is very destructive, and produces a widened, enlarged, and shallow acetabulum. Central protrusion of the acetabulum into the pelvis is not as common in tuberculous arthritis as in pyogenic arthritis. More common in tuberculosis of the hip is complete destruction of the anterior, superior, and posterior borders of the joint. Such destruction produces an enlarged and shallow acetabulum (wandering acetabulum), with migration of the cup of the acetabulum superiorly and subluxation of the femoral head into the

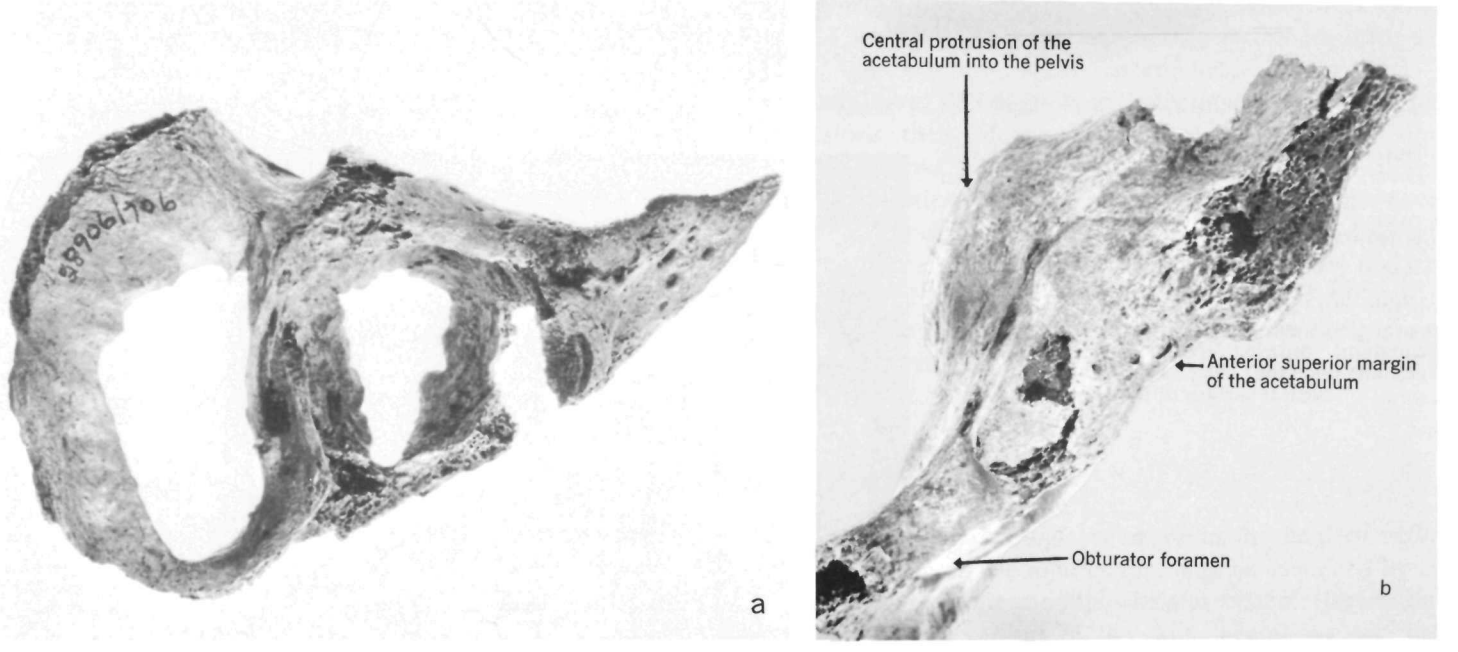
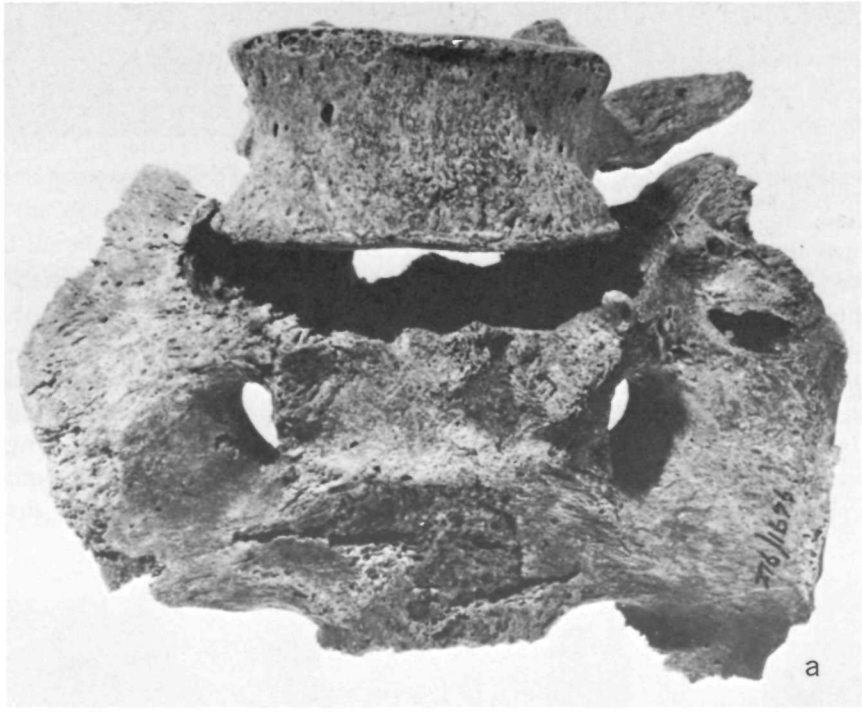
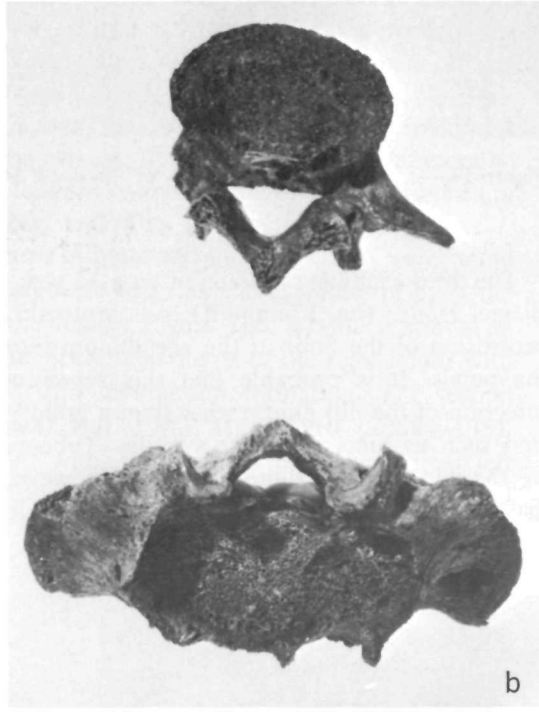


Figure 17.—*a*, Pyogenic arthritis of the left hip, lateral view (28906/706); *b*, superior view of the same hip showing protrusio-acetabuli.

newly formed socket superiorly. In the Badger House man, the borders of the acetabulum were still present, though damaged, and the cup of the acetabulum was deepened. The femoral head demonstrated only minimal areas of destruction typical of pyogenic infection, rather than the almost total destruction so often seen in tuberculous infection. In all probability then, this individual had a pyogenic arthritis of the hip joint.

The fourth example of infection was in Burial 2 of Site 1676. The lumbosacral joint of this adult male between 30 and 35 years of age presented extensive destructive changes (fig. 18a). The inferior surface of the fifth lumbar vertebra demonstrated minimal erosion and irregularity and a very small fringe of osteophytes due to secondary degenerative arthritis. The superior cortical surface of the first sacral vertebra was completely destroyed, and there was a large irregular crater present in the superior two-thirds of the centrum of the vertebra (fig. 18b). The crater was ringed with a fringe of osteophytes of spectacular size. The inferior surface of the fifth lumbar vertebra and the superior surface of the first sacral vertebra were not congruous, an indication that some intervening tissue must have been present. The size of this space between the fifth lumbar and the first sacral vertebrae was much larger than the normal intervertebral disc. The space was probably filled with pus or granulation tissue. The disease process, therefore, probably represents an infec-





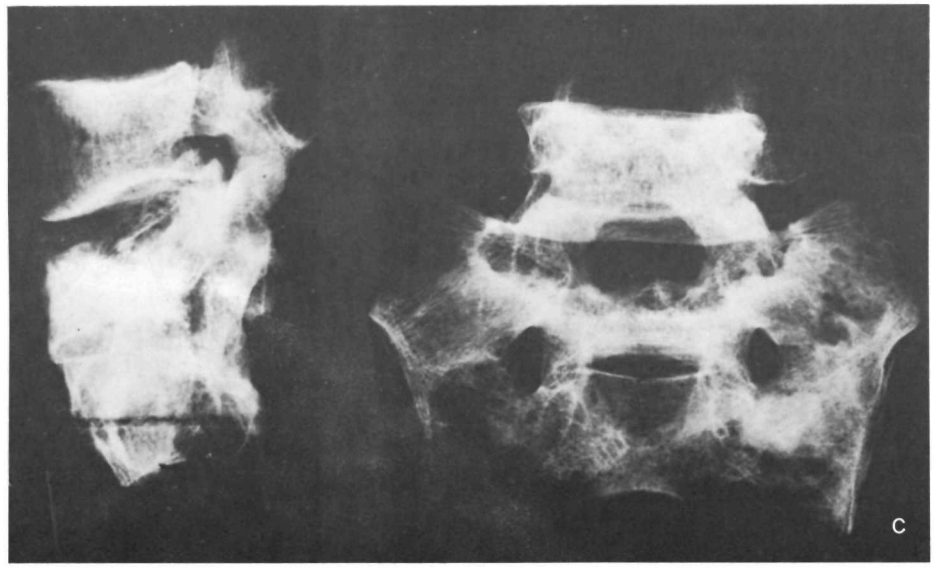


Figure 18.—*a*, Anterior-posterior view of the fifth lumbar vertebra and upper sacrum showing an essentially normal lumbar vertebra and the destruction of the superior surface of the sacrum (41049/714); *b*, the normal inferior surface of the fifth lumbar vertebra (upper photo) and the destruction of the superior and anterior surfaces of the first sacral vertebra (lower photo); *c*, lateral (left) and anterior-posterior (right) roentgenograms showing the normal fifth lumbar vertebra and the destruction of the superior cortical surface of the sacrum.

tion of the disc space with secondary degenerative and reparative changes involving the lumbosacral joint. The activity of the infection subsided or became quiescent, and the degenerative arthritis became prominent. Although the bacteriology of the infection would certainly be in doubt, the most likely probabilities are tuberculosis and pyogenic infection. Involvement of the lumbosacral joint with infection of either kind is quite rare and certainly would have been more rare in an individual of this age. Gibbus formation, so typical of tuberculosis of the higher vertebral bodies, would not be expected at this level of the vertebral column. The minimal damage of the fifth lumbar vertebra, the lack of wedging of the vertebra, and the absence of involvement of other areas of the spine favor a diagnosis of pyogenic infection. Roentgenograms of this lumbosacral joint are shown in figure 18c.

The final example of infection is in a 24 year old female of Mug House. The process involved the temporomandibular joints (fig. 19). The left condyle of the mandible was severely eroded and reduced to a wafer-thin projection medially. The remnant of the condyle was less than 2 mm. in thickness. The neck of the left mandible was also shortened and thinned. The right condyle had been similarly damaged and was approximately one half its normal size. There was minimal osteophyte formation of degenerative arthritis on the condyle. The temporal portions of the joints were extremely shallow and eroded. When articulated, the mandible had no stability on the base of the skull, and this made markedly anterior-posterior motion possible. Although the cartilaginous menisci of these joints were not present at this late date, it is probable that both menisci had either been destroyed or extensively damaged by the disease during the life of the individual. It is conceivable that the changes in the two temporomandibular joints could be the result of a fracture of one condyle with simple degenerative changes in the other. However, the change on the left represents destruction rather than deformity. The destructive process may have been the result of either a pyogenic arthritis or a rheumatoid arthritis of the temporomandibular joint. There was no evidence of rheumatoid arthritis of other joints such as that found in the juvenile form of the disease (Still's Disease). Single mandibular joint involvement would be most unusual for rheumatoid arthritis. The best judgment, therefore, is that this process represents one of pyogenic infection of the temporomandibular joint with simple but extensive secondary degenerative changes in the right joint.

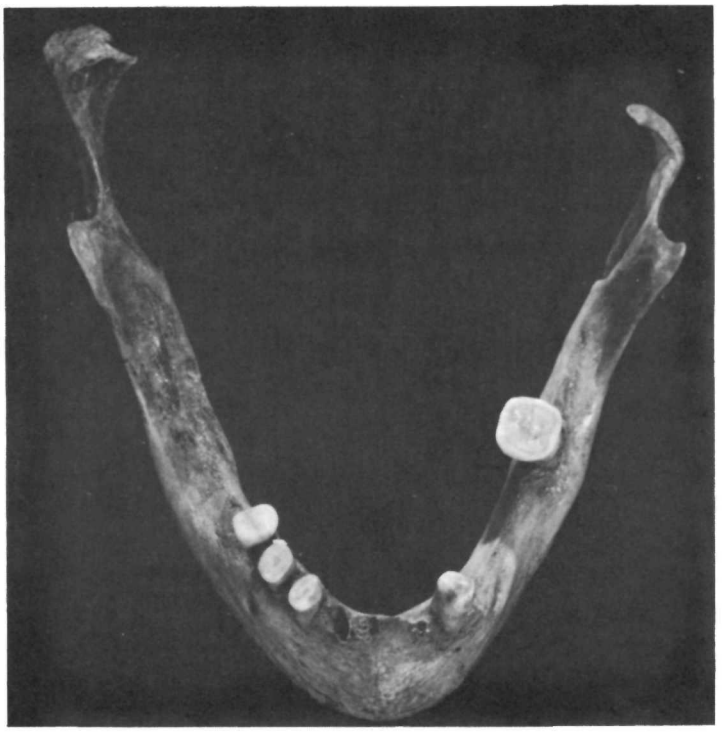


Figure 19.—Mandible with neck and condylar changes of uncertain nature (23686/703).

No support for the theory of the New World origin of syphilis was found in this study. All of the skeletons examined were pre-Columbian, and none exhibited evidence of syphilis. The only two questions were in two individuals who were simply recorded as "mysteries". These two will be discussed in the section dealing with diseases of uncertain origin.

Equally significant is the fact that no clear examples of osseous tuberculosis were found. The suspectibility of Indians to tuberculosis within the period of written history is well known. Hrdlicka (1908, pp. 208–212), in his survey of the Indian population in the American Southwest, found an extremely high incidence of both pulmonary and osseous tuberculosis. The only two examples of infection which could possibly be tuberculous were both discussed previously—one, the 37 year old male of Badger House with an infectious process in the hip, and the other, the 32 year old male from Site 1676 with destruction of the lumbosacral joint. Skeletal involvement with tuberculosis is almost always a process of arthritis, while tuberculous osteomyelitis is extremely rare. Although both individuals could have had tuberculosis, their infections were probably pyogenic rather than tuberculous. The diagnosis probably could have been made with fair certainty had some of the soft tissue remained. Tuberculous granulomas and abscess formations are quite typical pathologically, and they frequently undergo calcification. Had there been a tuberculous abscess with calcification in either individual, the calcified portions surely would have remained in as good a state of preservation as had the bones.

Trauma

Included in this etiologic category in the field of orthopedics would be all the injuries that may be sustained by the various tissues of the muscluo-skeletal system: lacerations, contusions and abrasions of the skin, lacerations and ruptures of the tendons, sprains of the ligaments, strains (partial

or compete ruptures) of the muscles, dislocations of the joints, and fractures of the bones. All of the above injuries are the result of a force acting upon the tissues. Since all of the tissues other than bone had disintegrated, only the evidences of fracture remained. It is therefore impossible to state how traumatic a life the people on Wetherill Mesa lived.

Support for the popular conception that these primitive peoples were injured and left maimed and crippled is lacking. It is probable that our current preoccupation with the treatment of injury and disease—doctors, ambulances, hospitals, etc.—and the lack of such refinements in these primitive peoples have led to such a conclusion. In reality, current populations live with the threat of much greater forces of injury, such as in automobile wrecks, plane crashes, and the like. It is probable that minor forces threatening the Wetherill Mesa peoples did exist, but that all evidences for sprains, strains, ruptures, and contusions disappeared with the disintegration of the tissues. Perhaps the greatest forces encountered by these peoples were in falls from rocky heights. The small number of fractures is due probably to the caution of the people and the slower pace of the life they lived.

Evidences of fracture were found in 24 bones involving 19 different individuals. All of the fractures were healed or healing, with some presenting evidences of malunion. There was no evidence of infection about any of the fractures, and it would thus appear that all were of the closed variety with no complications. Relative absence of fractures of major external force indicates that these people lived a rather quiet life without frequent warfare, and that they did not sustain many serious falls from the cliffs and mesas where they lived.

There were nine fractures of the vertebral bodies, most of which represented simple anterior compressions of flexion injury (fig. 20). All of the anterior compression fractures presented maximum stability, and therefore probably caused no neurologic complications. Stability of fractures of the vertebra is determined on the basis of injury to the anterior and posterior elements, those elements anteror or posterior to the spinal cord. The anterior elements would include the vertebral bodies, the intervertebral discs, and the anterior and posterior longitudinal ligaments. The posterior elements include the pedicles, the transverse processes, the superior and inferior facet processes and joints, the laminae, the spinous processes, the interspinous ligaments, the facet joint capsules, and the paravertebral musculature. The injury may produce damage to tissues either anteriorly, posteriorly, or both. When only anterior or posterior tissue integrity is lost through injury, the vertebral column is considered to be "stable." When both anterior and posterior tissue integrity are lost through injury, the vertebral column is considered to be "unstable." The individual is either paraplegic, or potentially so. Most of the vertebral fractures were near the thoracolumbar junction, a site injured with strong forward flexion and often seen in present populations. One thoracic vertebral fracture (fig. 21) presented lateral wedging probably from a rotational force rather than a simple flexion force. Such lateral wedging is frequently associated with instability of the spine and spinal cord damage. This vertebral fracture had healed satisfactorily, and the remaining portions of the skeleton did not indicate any porosis, the presence of which would have indicated paralysis.

The most interesting and threatening vertebral fracture involved the lumbar vertebrae of an adult male of Big Juniper House (fig. 22a). This man had had a rotational fracture-dislocation of two lumbar vertebrae. The pieces of white paper seen in the figure were placed on the anterior surfaces of the vertebrae to demonstrate this rotation. The fracture involved not only the vertebral bodies anteriorly but also portions of the posterior elements. The two bodies had fused solidly together anteriorly, and this fusion had produced good stability. The remaining portions of the skeleton did not reveal any evidence of paralysis. On the left are seen huge osteophytes which have united the three vertebrae. These spurs are much larger than the usual ones seen in degenerative change.

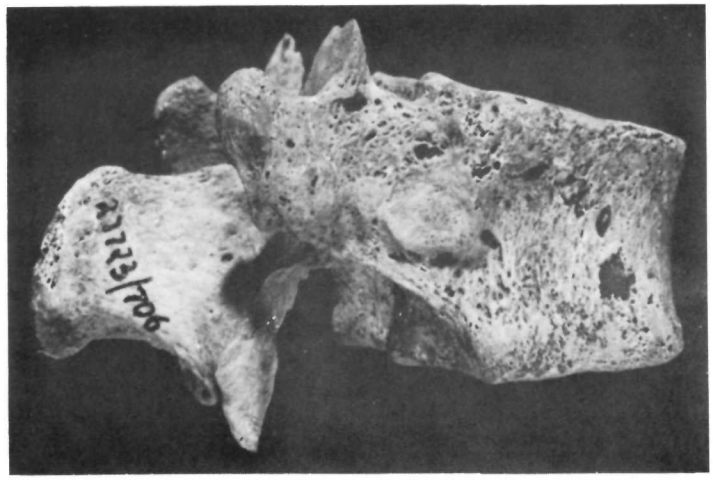


Figure 20.—Minimal compression fracture of the thoracic vertebra, lateral view (27723/706).

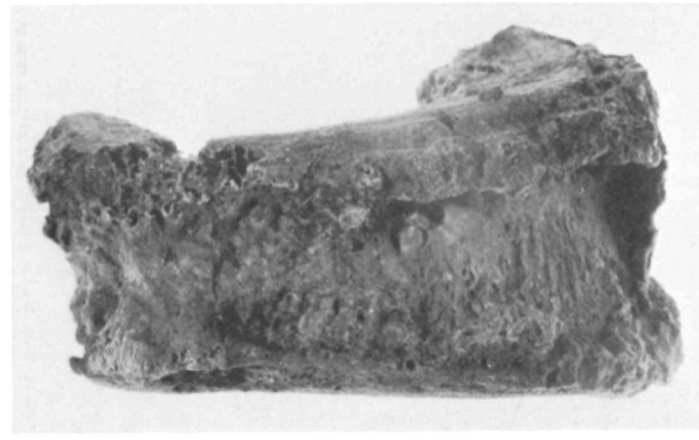


Figure 21.—Frontal view of a lateral wedge fracture of a thoracic vertebra (32895/710).

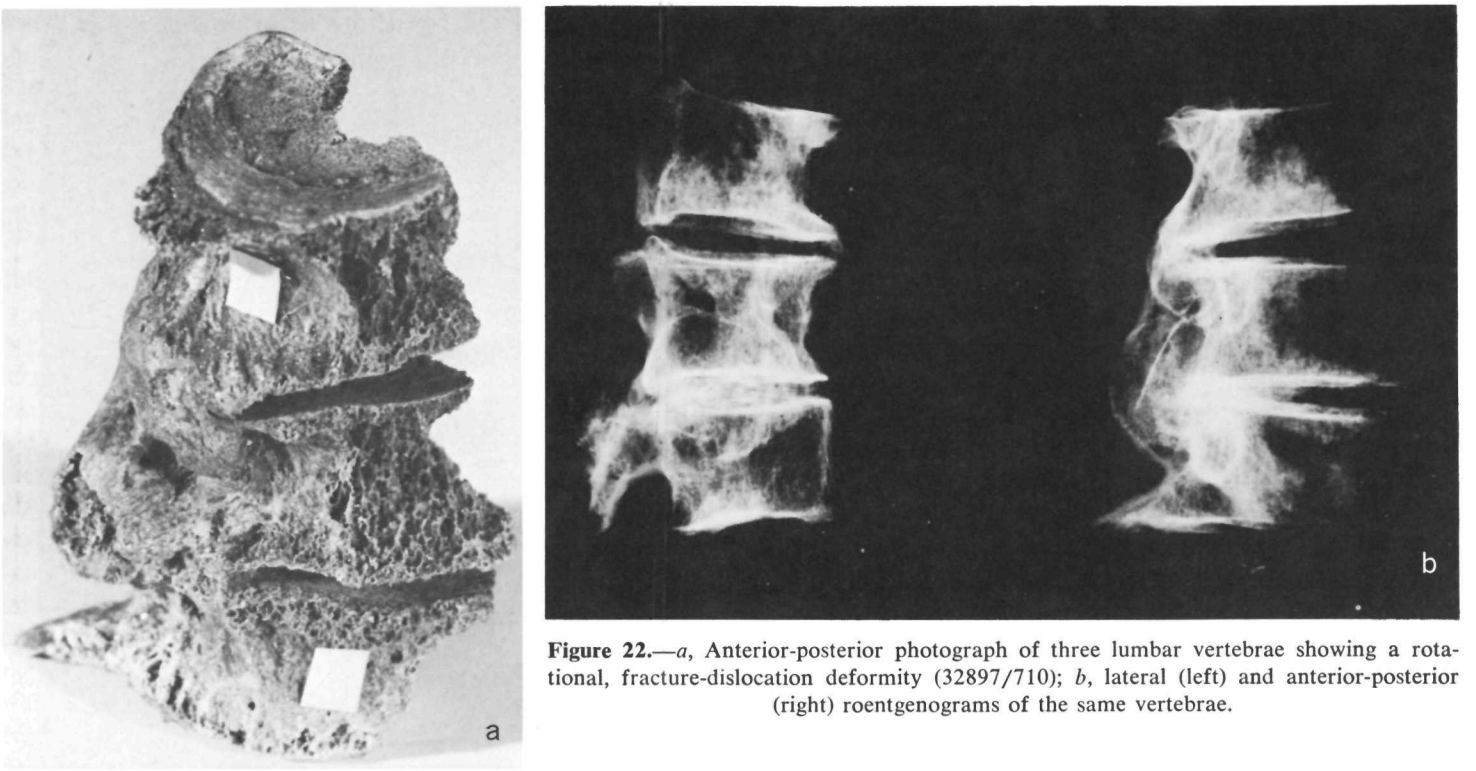


Figure 22.—*a*, Anterior-posterior photograph of three lumbar vertebrae showing a rotational, fracture-dislocation deformity (32897/710); *b*, lateral (left) and anterior-posterior (right) roentgenograms of the same vertebrae.

They actually appear to be reactional callus developing in a fracture-dislocation hematoma. Unfortunately, most of the posterior elements have disappeared, but the amount of rotation could only have been found with unilateral facet dislocation. The facets of the left posterior were probably interdigitated in reverse fashion, while those on the right posterior had remained in a subluxated position. This retention of proper digitation of some of the posterior elements probably maintained some stability of the spine and protected the neural elements in the neural canal. This would explain the lack of evidence of paralysis in the legs. At this level, only the conus or cauda equina would have been injured, and had paralysis occurred, it would have been limited to the feet and legs below the knee.

In the roentgenograms of these same vertebrae (fig. 22b), the size and density of the callus uniting the lower two vertebrae is apparent. The superior vertebra is angulated about 30° on the middle vertebra. The three pedicles on the right posterior of the right-hand film are visible, and the superior pedicle is displaced laterally. It is unfortunate that the left posterior elements have disappeared, but such might be expected if they were fractured and fragmented, and thus less able to stand the ravages of time.

Non-vertebral fractures included four of the clavicle (28907/706, 244033/703, 32890/710, 19122/703). In figure 23, a healing fracture of the right clavicle is shown. Roentgenograms indicated that the healing fracture was probably six to eight weeks old at the individual's death. The medial end demonstrates a minimal angulation deformity, and its discoloration is probably due to blood pigment of the fracture hematoma.

There were five rib fractures, 28925/706, 24033/703, and 2886/175. Three of the fractures occured in one infant found in Mug House (2886/175). None of the rib fractures demonstrated significant deformity, and it is probable that adjacent soft tissues such as the pleura and lung were not damaged. These fractures were probably the results of falls or blows on the chest wall.

A healed sternal fracture, 28906/706, (fig. 24) was found in specimen number 286906/706. The anterior surface is to the left, and the residual angulation deformity of the lower one-third of the sternum can be seen.

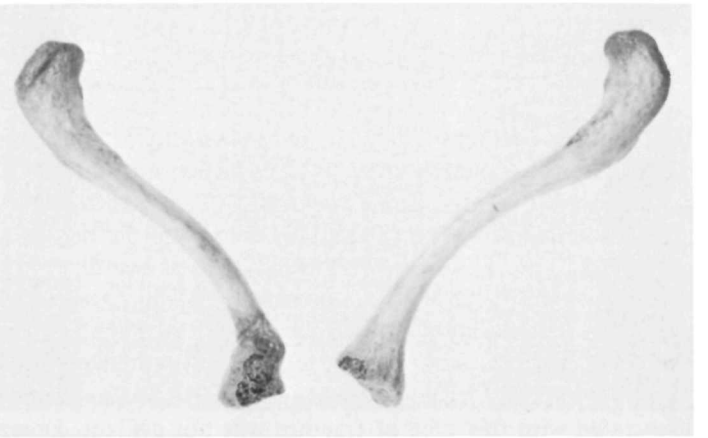


Figure 23.—Healing fracture of the left clavicle.

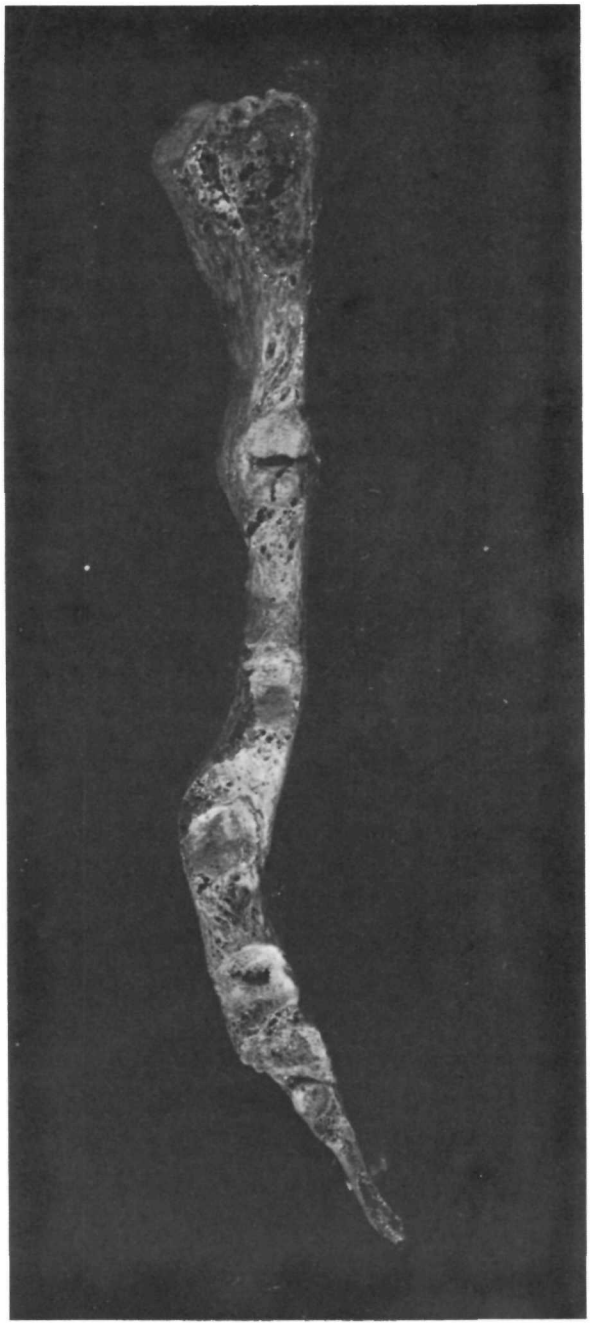


Figure 24.—Healed fracture of the sternum (28916/706).

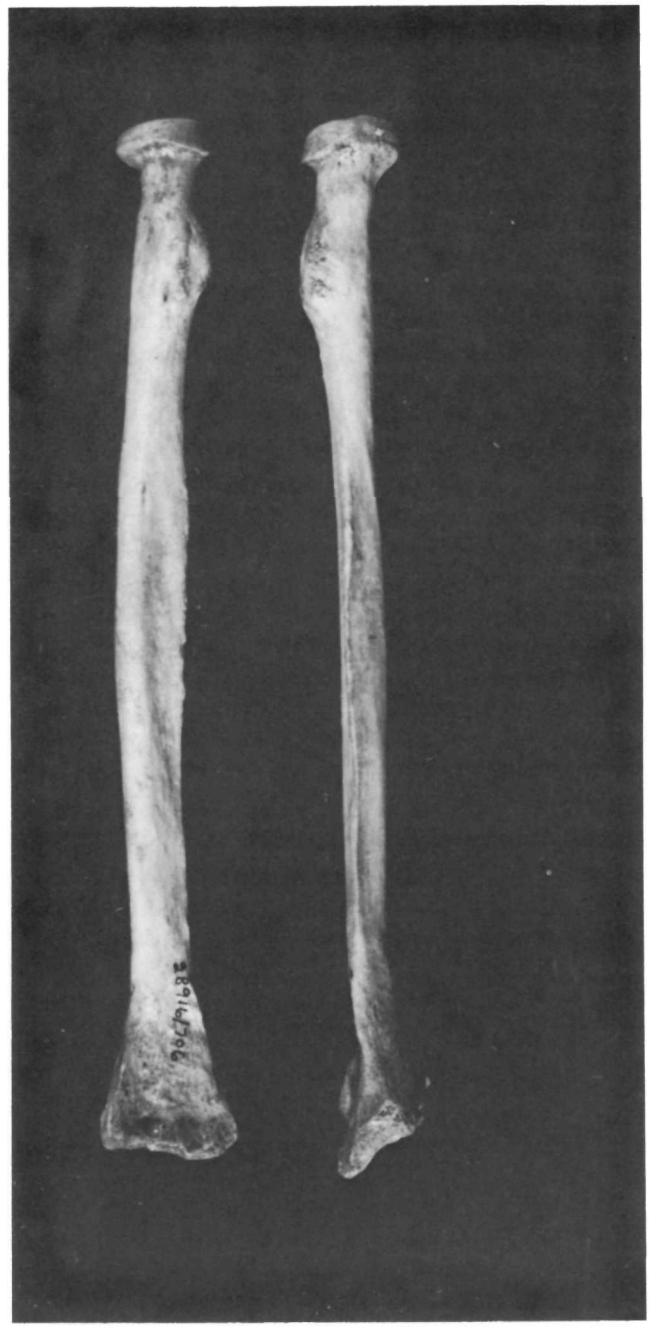


Figure 25.—Healed fracture of the radius (28916/706).

Other non-vertebral fractures are shown in figures 25 and 26. Figure 25 depicts two views of a healed fracture of the radius. The deformity of the distal end was caused by dorsal angulation and radial deviation. The density of the bone was roentgenographically normal. The fracture of the distal end of the radius involved the radio-carpal joint and resulted in malunion with residual shortening and dorsal angulation of the distal articular surface of the radius. It would probably be called a Colles' fracture, but the usual dorsal displacement associated with this type of fracture was not evident. Dorsal displacement may have been present immediately after the injury, and treatment may have resulted in its reduction without correcting the dorsal angulation. This radius presented normal bone density, and the functional result of the fracture treatment was probably quite satisfactory. In another individual (13432/702), there was a fracture of the radial head with evidence of malunion, and in all probability, this elbow would have had limited function of pronation and supination.

A fracture of the tibia is demonstrated in figure 26a, b, and c. The fracture, in the proximal tibial metaphysis, was well healed. There was excellent remodeling of the callus, indicating that the fracture was probably a year old at the

time of the individual's death. The obliquity of this fracture and the presence of an intact fibula indicate that the fracture was the result of a rotational force. Such a fracture would have had great inherent stability, and would have needed no reduction and only minimal treatment. It would have healed solidly enough to permit weight bearing in 6 to 8 weeks in this 18 to 20 year old individual.

Two other evident fractures were of a phalanx of a hand, 23687/703, and a metatarsal, 28910/706.

Multiple fractures were seen in three different individuals. A previously cited infant from Mug House (2886/175) with three rib fractures raises the specter of the "battered child," the syndrome of parental abuse that is seen so frequently today. The injury may have been fatal for this infant since there was little evidence of healing of the fractures. The three rib fractures in this infant could have been sustained as the result of a fall or an accident. On the other hand, the three rib fractures might have been incidental to another disease. The infant could have fractured the ribs as a part of "whooping cough", asthmatic seizure, or even violent vomiting. Whatever the cause of the fractures, the infant did not live long afterwards. One individual (23687/703) had two probably unrelated fractures, one of a vertebral body, and the other, of a phalanx. One hardy individual (24033/703) had factures of a clavicle, a rib, and a vertebra. These fractures probably were not incurred at the same time since the rib and the vertebral fractures were well healed, while the fracture of the clavicle was only partially healed.

The fracture of the tibia and the rotational fractures of the lumbar vertebrae were the only ones which probably resulted from major external force. All of the vertebral fractures, including the rotational fracture-dislocation one, could have been treated by simple bed rest or limitation of activity. The sternal fracture, and the clavicular, rib, radial head, and metatarsal fractures could have all been treated by simple limitation of activity. Probably no serious fracture deformity resulted from the injury in any of these cases, and none of these fractures required reduction. The fracture of the phalanx would have presented minimal deformity and would have required no reduction. It could have been immobilized by simple splinting or by binding the involved finger to one of the neighboring intact fingers. Of all the fractures seen, only the fracture of the distal radial shaft probably had significant fracture deformity and required reduction. The tibial fracture might have had a significant rotational deformity, and could have been reduced easily by straightening the leg out, and simple immobilization with external splints would have been satisfactory treatment. Such immobilization would have been of short duration since the adjacent fibula was intact in this individual.

It is surprising that only one fracture of a major long bone of the lower extremity was seen in this study (36766/711). One would expect numerous leg fractures in a population that lived under such primitive conditions. The one fracture of the tibia noted was in a male 18 to 20 years of age. It was well healed with minimal deformity and had occurred probably a year or two prior to the death of the individual. Treatment had apparently been very adequate, and the fracture itself probably did not contribute to the demise of the person.

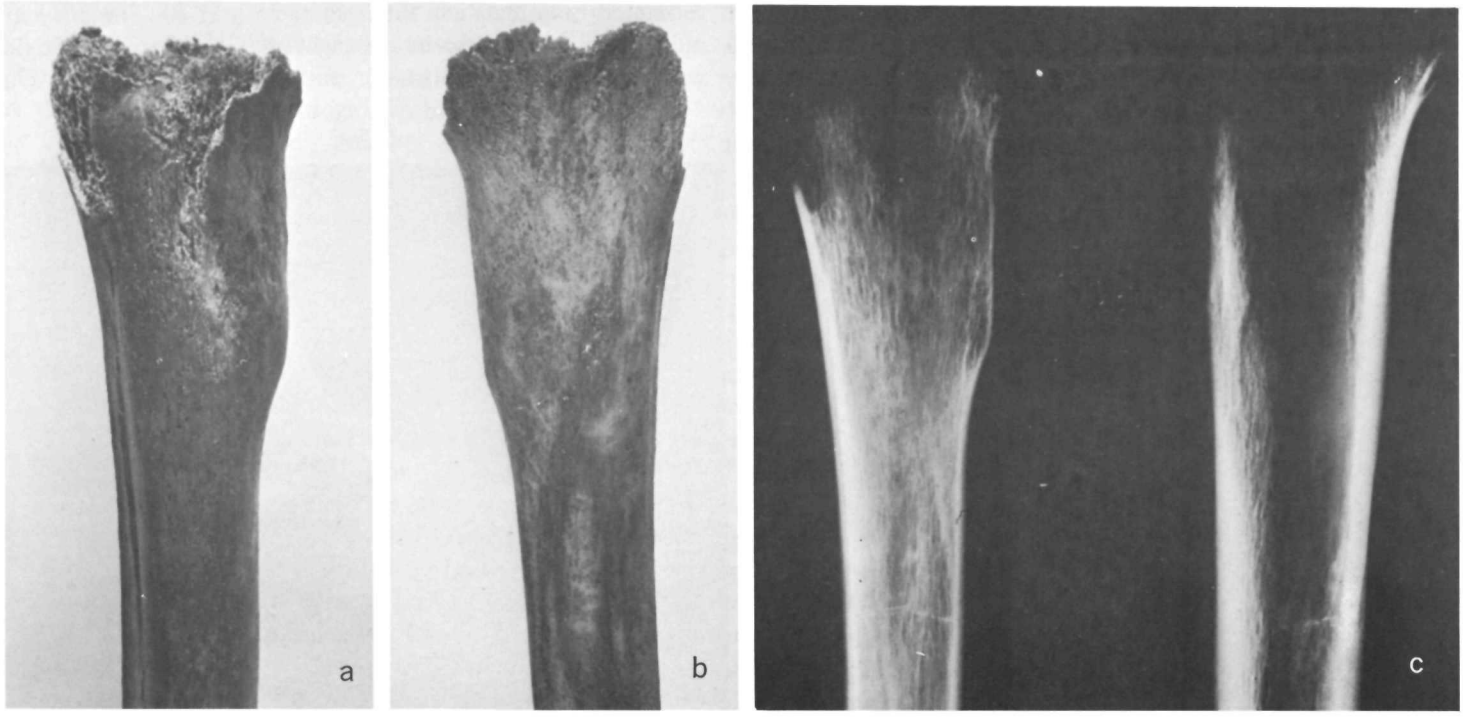


Figure 26.—*a*, Healed fracture of the proximal end of the tibia, anterior-posterior view (32766/711); *b*, minimal fracture deformity posteriorly is seen, lateral view; *c*, restoration of the bone's internal architecture is shown by roentgenograms of figure 26a and b.

It is possible, of course, that the infants and children may have experienced a number of fractures of the major bones of the extremities. Since these fractures usually are not lethal, they would heal without complication. Residual fracture deformities in infants and very young children may well have been corrected by subsequent growth and remodelling of the bone—a remarkable capability in children. The fractures would thus have become invisible both grossly and roentgenographically. However, frequent trauma of the extremities would have also been anticipated in active adults and adolescents with the evidence of such fractures clearly visible.

The absence of fractures usually associated with old age is significant, i. e., femoral neck fractures, intertrochanteric fractures, Colles' fractures, and the minimal but multiple compression fractures of the midthoracic spine. Only one fracture of the distal end of the radius was found, and it probably represented a Colles' fracture. It may well have been treated so successfully that the residual dorsal displacement deformity was removed and only the residual augulation remained. The dreaded hip fractures so common in today's population were not seen at Mesa Verde since the life expectancy there was far short of the usual age for such injuries. Compression fractures of the thoracic spine in contemporary postmenopausal and senile osteoporotic individuals were certainly not common in the persons of this study. Again, it is probable that the relatively short life expectancy of the majority of the Mesa Verde population precluded the occurrence of these injuries.

It is also significant that none of the fractures appeared to be the result of warfare. There were no depressed skull fractures, and no arrowheads or other foreign bodies imbedded in bone.

Vascular Lesions

Direct evidence of vascular diseases such as laceration, occlusion, or arteriosclerosis would have disappeared with the disintegration of the soft tissues. However, indirect evidence would remain in the bones. Bone is a very active tissue metabolically, and is very dependent upon the blood supply for its survival. Although bone is extremely vascular (each bone cell must be within one-tenth of a millimeter of a functioning capillary), its blood supply is very precarious. Thus, if an artery or arteriole to a bone or a part of a bone is occluded, the bone dies. The consequences of this necrosis of bone are so clear and characteristic that, if such changes are seen either grossly or radiographically, we may infer with more than reasonable certainty that the vessel had been damaged.

Clinically, such blood vessels are most frequently occluded by disease or trauma. If this trauma has produced a fracture fragment, the piece of bone may remain unreabsorbed and unchanged for years. In children and adolescents, the vessels to an epiphyseal ossification center may be occluded. In this case, the ossification center dies and the replacement of the dead bone by living bone proceeds. Replacement may be so perfect that no evidence of the previous necrosis may be detected. More commonly, however, some typical deformity results, and the presence of the prior ossification center necrosis can be identified.

Only two lesions of avascular necrosis were found in this study. Both were in males and were osteochondritis dissecans of the femoral condyles. One male was estimated to be about 30 years of age, and the other (41053/714) was estimated to be between the ages of 35 and 40. The 30 year old male had a crater in the right medial femoral condyle which was 1 cm. in diameter and 4.4 mm. deep (fig. 27a

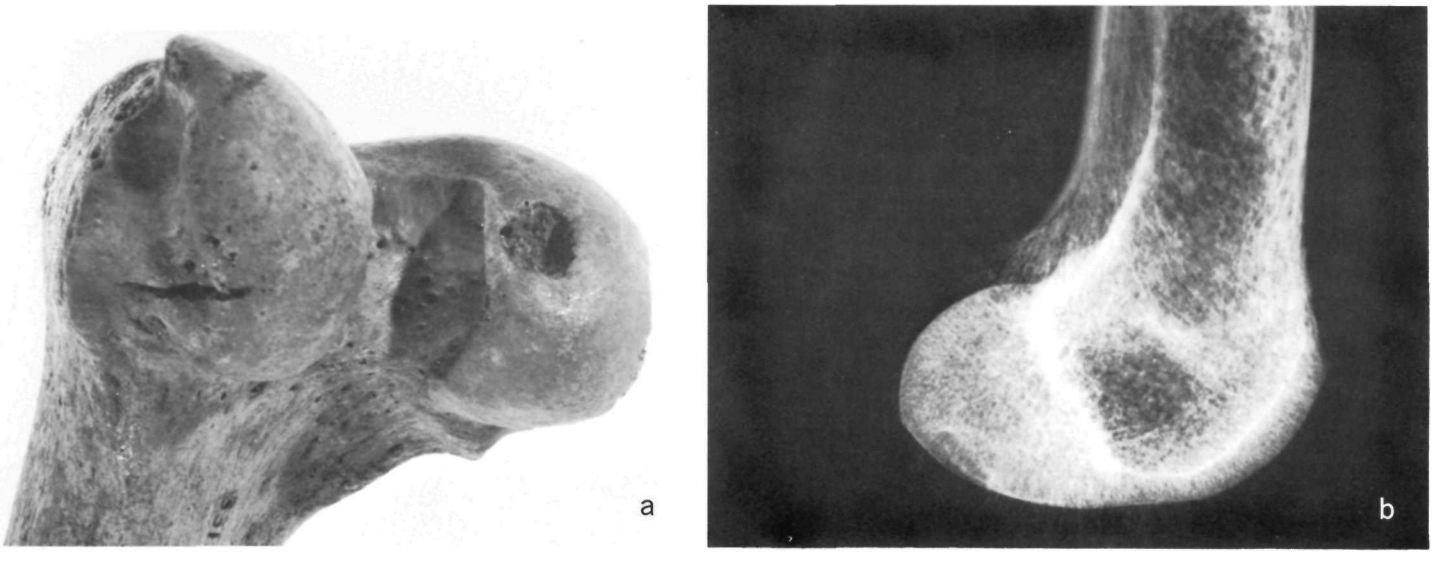


Figure 27.—*a*, Osteochondritis dissecans of the right femur (33179/708); *b*, roentgenogram of same specimen showing the defect in the femoral condyle with its sclerotic border.

and b). It exhibited minimal degenerative arthritic changes about the crater. The 35 year old male presented a much more shallow crater with evidence of healing. No signs of degenerative arthritis were present about the lesion.

It is interesting that these vascular lesions were seen in males in this age bracket. The lesion usually begins in the second decade as the result of a vascular interruption to the bone of the area. The necrotic bone is spontaneously and slowly replaced by living bone. However, the lesion fails to heal in some individuals, and the avascular necrotic bone may thus remain in the femur for a long period of time. If the area is traumatized, the necrotic fragment may be displaced from the crater, and may persist in the knee joint as a loose body producing severe degenerative arthritic changes. It is probable that these two males had only minimal symptoms in their knee joints, because the bones of the lower extremities presented normal density and showed no evidence of atrophy from disuse. These individuals may well have experienced symptoms of pain or locking of the knee joints, but these symptoms must have been of a minimal degree, since there was no evidence of a great amount of arthritis.

Neurologic Problems

Disintegration of soft tissues (the brain, spinal cord, and peripheral nerves) made direct detection of neurologic disease impossible. Indirect evidence would only come from such secondary changes as arthritis or osteoporosis. However, these diagnoses would be very difficult to establish since the bone changes are non-specific. No examples of neurologic defects of either prenatal or acquired disease origin were evident in the remains studied.

Two prosthetic devices were found which might have been used in treating neurologic problems. Illustrated in figure 28a is a pair of child's crutches measuring 38⅜ in. and 39¼ in. The axillary loops are well padded with yucca fiber covered with sewn leather (fig. 28b). They were believed to have been found at a site in the Mesa Verde area by Richard Wetherill. Their apparently long usage rules out the treatment of a simple traumatic injury. Instead, it is quite certain that the child must have had a chronic crippling disease.

The second probable prosthetic device is an aspen bark corset (fig. 29). The corset has lacing in the front and was probably used to support the spine. Recovered at Mug House, it is quite similar to the one described by Freeman (1918, p. 449) that was probably found on Chapin Mesa. It should be added that some people believe that the two aspen bark objects are not corsets, but rather are portions of infant cradle boards. In any case, they do present something of a mystery. If the device in figure 29 is indeed a back support, it probably would have been used to treat the common backache. It might have been used in the treatment of a spinal injury, a spinal deformity of neurologic origin, or in the management of an individual with a slipped disc (technically referred to as a lumbar nerve root compression from herniation of a lumbar nucleus pulposus of a lumbar intervertebral disc).

Neoplasia

Neoplasms are frequently called cancer. The term is loosely applied. A cancer, or more correctly, a carcinoma, is a malignancy developing in an ectodermal tissue. A malignancy arising in a mesodermal tissue is a sarcoma. Neoplasia may be either benign or malignant depending upon its behavior. The very malignant neoplasias are lethal. The benign neoplasias may undergo transformation to malignant neoplasia with time or with irritation.

The malignant neoplasias may spread to bone in the process of metastasis. These bony metastases may be destructive of the bone (osteolytic), or they may stimulate the bone to react (osteoblastic). In either instance, the presence of the precipitating malignancy may be inferred since the bone changes are quite typical. Metastatic lesions are frequently multiple. Benign or malignant neoplasia of bone very often produces a characteristic gross and radiographic appearance, and the diagnosis could have been made with relative ease in these skeletal remains.

Three examples of neoplasia were encountered in the skeletal remains. Two were simple osteocartilaginous exostoses while the third was a cyst of the calcaneus. One of the exostoses, measuring only 11 by 3 by 4 mm., involved the distal end of the radius in a 43 old man (24033/704). It was in such a position on the volar surface of the radius that it would have interfered with flexor tendon function about the wrist. The second exostosis, measuring 18 by 30 mm., was on the posterior surface of the proximal end of the tibia in a 27 year old male (fig. 30a). It was probably asymptomatic. Although the cartilaginous caps of these two lesions had disappeared, the roentgenographic appearances were so typical (fig. 30b) that the diagnosis could be made with assurance.

The cyst of the calcaneus (fig. 31a) appeared as a fusiform swelling of the body of the left calcaneus immediately inferior to the posterior facet of that bone. It was difficult to estimate the size of the cyst from the external surface, but it would appear the lesion produced about a 3 mm. enlargement of the mediolateral dimension of the calcaneus. The roentgenogram (fig. 31b) is quite typical of the disease process called a cyst, a xanthoma, or a giant cell tumor of the calcaneus. The precise etiology of the lesion is not known, but it is so typical roentgenographically that the diagnosis could be made with assurance. Such a lesion is typically present in the calcaneus and is found very infrequently in other bones. It apparently had not increased sufficiently to produce a pathologic fracture.

Whether or not the three examples noted above actually represent neoplasia is debatable. That they represent benign

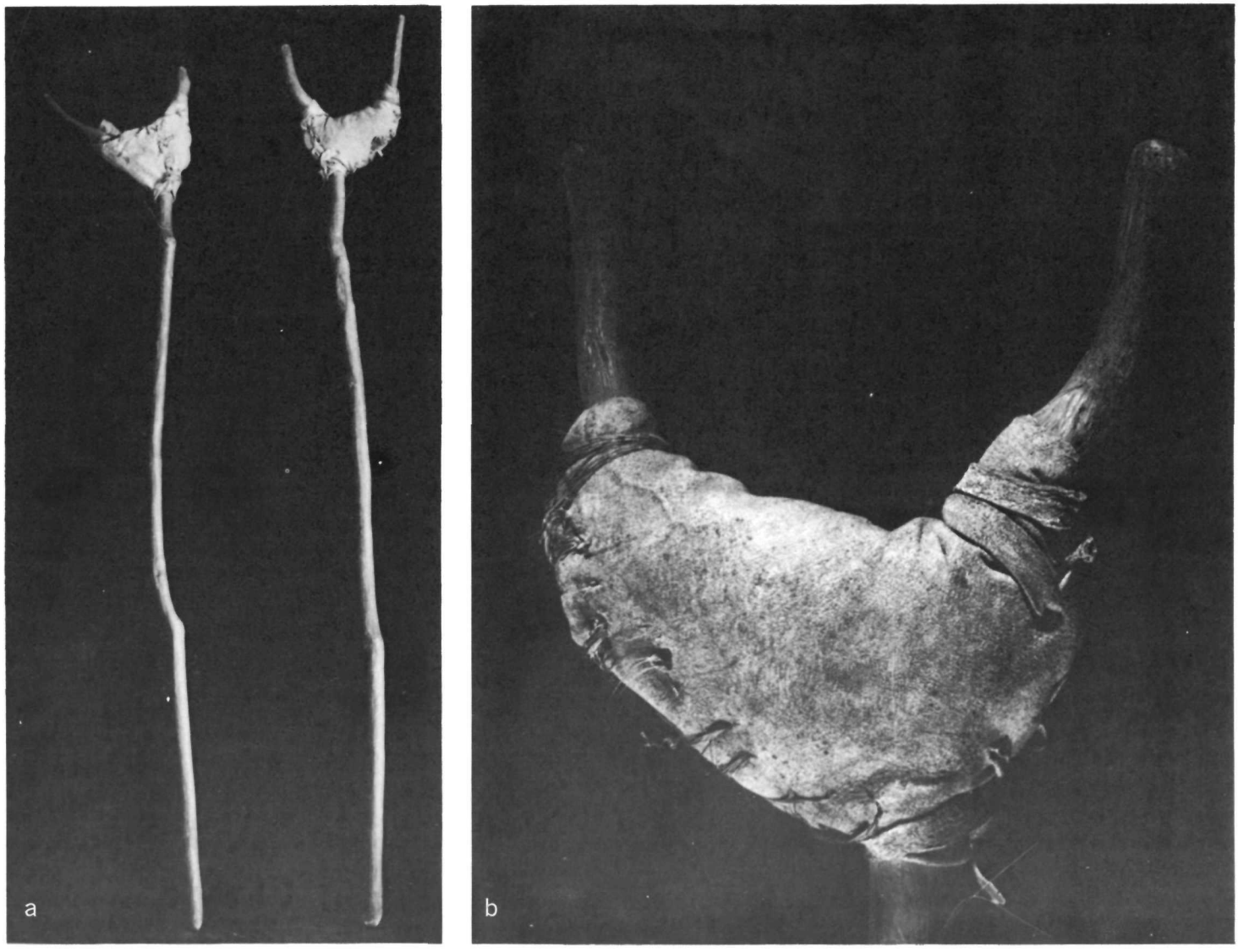


Figure 28.—*a*, A child's crutches from Mesa Verde; *b*, detail of axillary portion of the crutches.

processes cannot be questioned. However, some physicians believe osteocartilaginous exostoses are caused by the abnormal growth of remnants of displaced epiphyseal cartilage. Since some of these exostoses undergo secondary malignant transformation, the tendency has been to include them among examples of benign neoplasia. These lesions may be solitary or multiple exostoses. Persons with such lesions tend to have limited function of nearby joints and deformity of the long bones.

The true nature of the lesion of the calcaneus again is a mystery from an etiologic standpoint, although most physicians would include it among examples of neoplasia. It is possible, however, that the cyst may result from a lipoid storage disease, a vasular disturbance, or an infection.

Diseases of Uncertain Origin

This category includes those diseases that remain uncertain or confusing etiologically, the diseases for which two or more etiologies may be equally probable, and the diseases that are a complete mystery. This uncertain category is not simply a wastebasket or miscellaneous category.

The confusing or uncertain diseases of the first group of this category noted above are diseases that have been identified or described rather completely in the past. They are usually quite typical in appearance, in age groups, in deformities, and in radiologic presentation. They have been subjected to many clinical research projects, but the evidence obtained is primarily negative. They are usually named for the physician who first presented the clear description or picture. If priority of description is in doubt, two or more names may be applied, as in Legg-Perthes-Calve's Disease, an osteochondrosis of the capital femoral epiphyseal ossification center (necrosis of bone and cartilage of the center). Often national pride or ignorance of foreign literature will dictate that different names be applied in different countries. Thus in the previously cited example, the disease would be Legg's Dis-

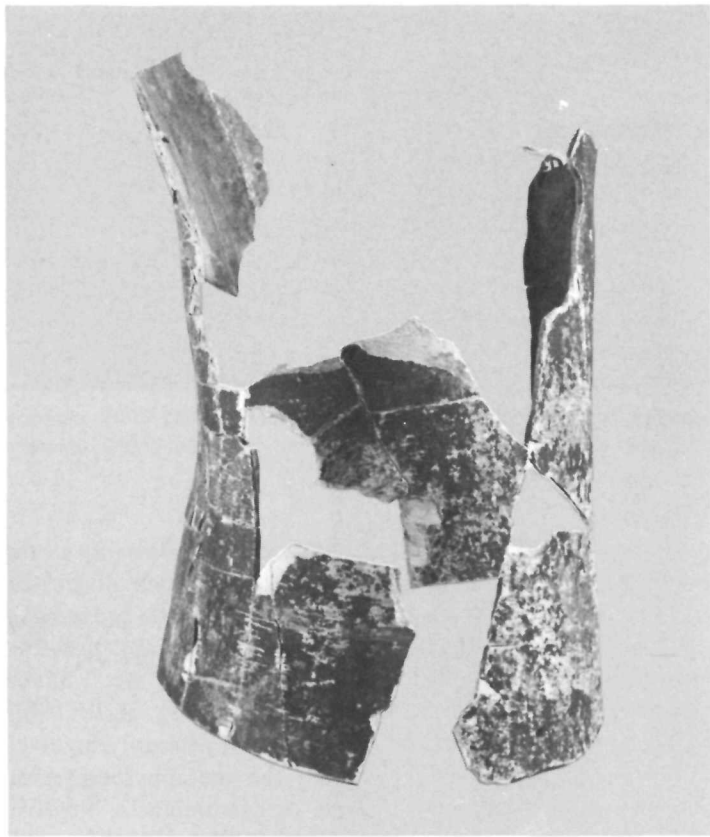


Figure 29.—A molded aspen bark corset.

ease in the United States, Perthes' Disease in Germany, and Calve's Disease in France. The first probable discription of the disease was by Waldenstrom of Sweden, but apparently that literature was not as widely read as that of the other three countries. Also, Waldenstrom's description may not have been as precise as those of the other three physicians who, writing independently and simultaneously in their own countries, provided us with accepted descriptions of the disease. Interestingly, during the sixty or more years since the three simultaneous descriptions, no specific etiologic agent has been proven, despite many studies in bacterial cultures, many genetic studies, and many assays for such biochemical causes as vitamin deficiencies or hormonal imbalances.

In the field of orthopedics, a widely known disease is Paget's Disease. The gross, the radiologic picture, and the prognosis are all easily recognized. However, the etiologic agent is unknown. It therefore remains in the uncertain category until such time as definitive research is performed. It can then be placed in its proper category.

An example of a disease of dual probabilities is Xanthoma of bone. It could be a lipoid storage disease (of lipoid metabolism) or a benign neoplasia. In addition, osteoporosis is multifactorial, and demonstrates the final common reaction of bone to many influences.

No examples of the common, currently recognized diseases of uncertain origin, such as Paget's disease and fibrous dysplasia, were discovered in this skeletal material. However, three isolated abnormalities of mysterious origin were found.

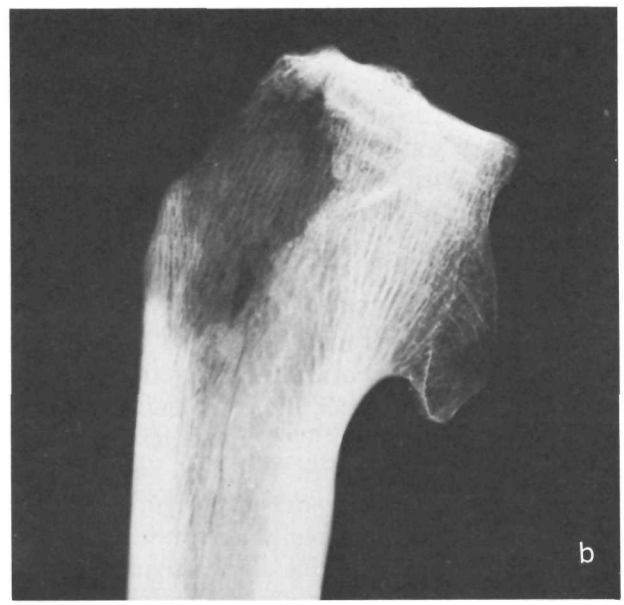
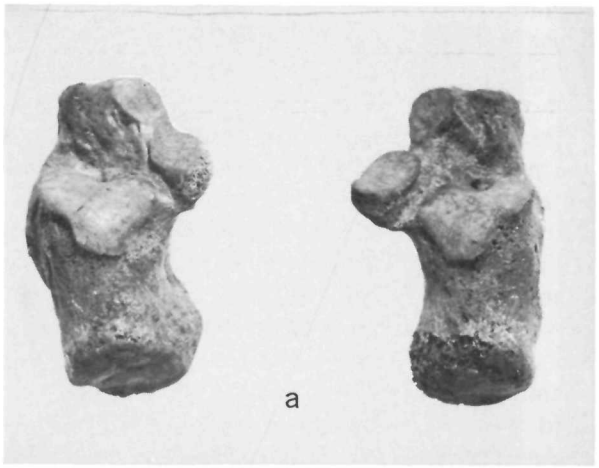


Figure 30.—*a*, Osteocartilaginous exostosis of the tibia (28707/706); *b*, roentgenogram the same exostosis.



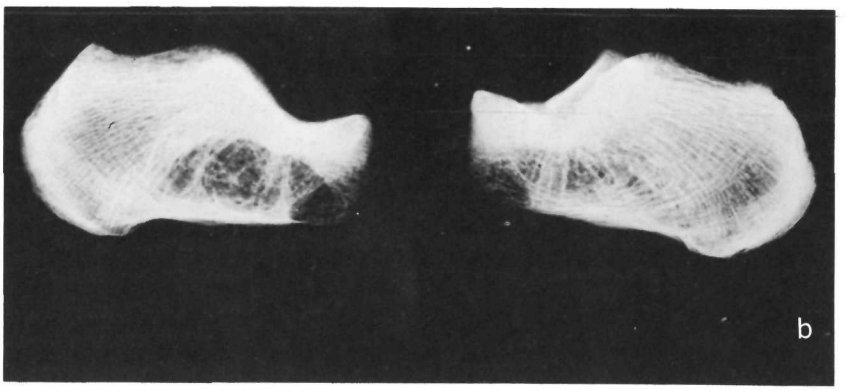


Figure 31.—*a*, Photograph of left and right calcanei (13651/700) showing enlargement laterally of the left calcaneus; *b*, lateral roentgenograms of same bones showing cyst of the left calcaneous.

The exact nature of these processes could not be determined from physical examination or from the roentgenograms of the bones. Therefore, these three abnormalities are simply listed as "mysteries."

The first mystery was in an adult female found by the survey (fig. 32a). The right radius presented symmetrical thickening of the distal diaphysis and metaphysis. The bone surface presented a minimal smooth fusiform enlargement without pitting or erosion. The roentgenograms (fig. 32b) revealed an undisturbed medullary canal, no destruction of the endosteal surface of the cortex, and simple thickening of the diaphyseal bone as the result of periosteal new bone formation applied to a normal cortex. The new formation was quite mature and blended with the cortex. None of the other bones of this skeleton presented any abnormalities. The diagnosis was simply periostitis possibly caused by infection, vascluar stasis, trauma, or simple hypertrophy of the bone.

The second mystery was found in a 24 year old male from Mug House. He presented bilateral symmetrical enlargement and very slight bowing of both tibiae. Figure 33a provides a lateral view of the tibiae in which the bowing and enlargement of the two diaphyses and metaphyses can be seen. The smooth surface of the bones is evident. The linear fracture of the distal shaft of the right tibia is probably post-mortem trauma. Figure 33b, a roentgenogram, is an anterior-posterior view of the same tibiae. It demonstrates the enlargement of the shafts by periosteal bone. The trabecular bone and medullary contents are undisturbed. A lateral view of these tibiae is provided in the roentgenogram of figure 33c. The thickening, bowing, and normal architecture of the trabecular bone can be seen. The fracture of the distal metaphysis of the left tibia is post-mortem. The cancellous bone was normal, and the articular surfaces of the knee and ankle joints were normal. Microscopic sections were made of the tibiae, but the interpretation of the sections was not of diagnostic significance.

The lesions did not appear to be the result of the medullary contents, nor of the bone itself. The external configuration of the bones suggested Paget's disease, syphilis, pyogenic infection, or fibrous dysplasia. However, the normality of the roentgen appearance of the bone excluded such diagnoses. The estimated age of the person (24 years) made a diagnosis of Paget's disease most unlikely since this disease most frequently occurs in the fifth and sixth decades of life. Vitamin D deficiency was considered, but the bowing of the tibia was in the anterior-posterior direction and primarily involved the diaphyseal region rather than the metaphyseal region that is usually involved in rickets or osteomalacia. Involvement of both tibiae in a symmetrical fashion would be most unusual for pyogenic infection. Syphilitic infection must be considered, but the smooth appearance of the periosteal surfaces and the absence of involvement of other bones do not support such a diagnosis.

Both of these mysteries, therefore, probably represent some form of unusual infection or simple periostitis. A diagnosis of syphilis could be readily applied to each of these mysteries, thereby adding more presumptive evidence to the theory of the New World origin of syphilis. However, the available evidence, namely the gross and roentgenographic examination of the involved bones and the remaining portions of the skeleton, is insufficient for scientific proof. The roentgeonograms are quite inconclusive, and experienced, competent radiologists who have examined the films have included a number of diseases in their differential diagnosis. Syphilis is known to the clinician as the great imitator, because its symptoms are similar to those of many other diseases. It therefore follows that other diseases may also appear much like syphilis. In an extensive study of pre-Columbian bones, Williams (1932, p. 978) came to the conclusion that there was overwhelming presumptive evidence for the theory of the New World origin of syphilis, but that scientific proof could not be achieved. Most physicians would agree with the latter part of the conclusion, but most must still remain skeptical about the "overwhelming presumptive evidence." Hrdlicka (1908, p. 191) stated that signs of syphilis were absent in the bones of pre-Columbian Southwest Indians. The scientific proof demands new methods, and hopefully, they will be available in the future. Until

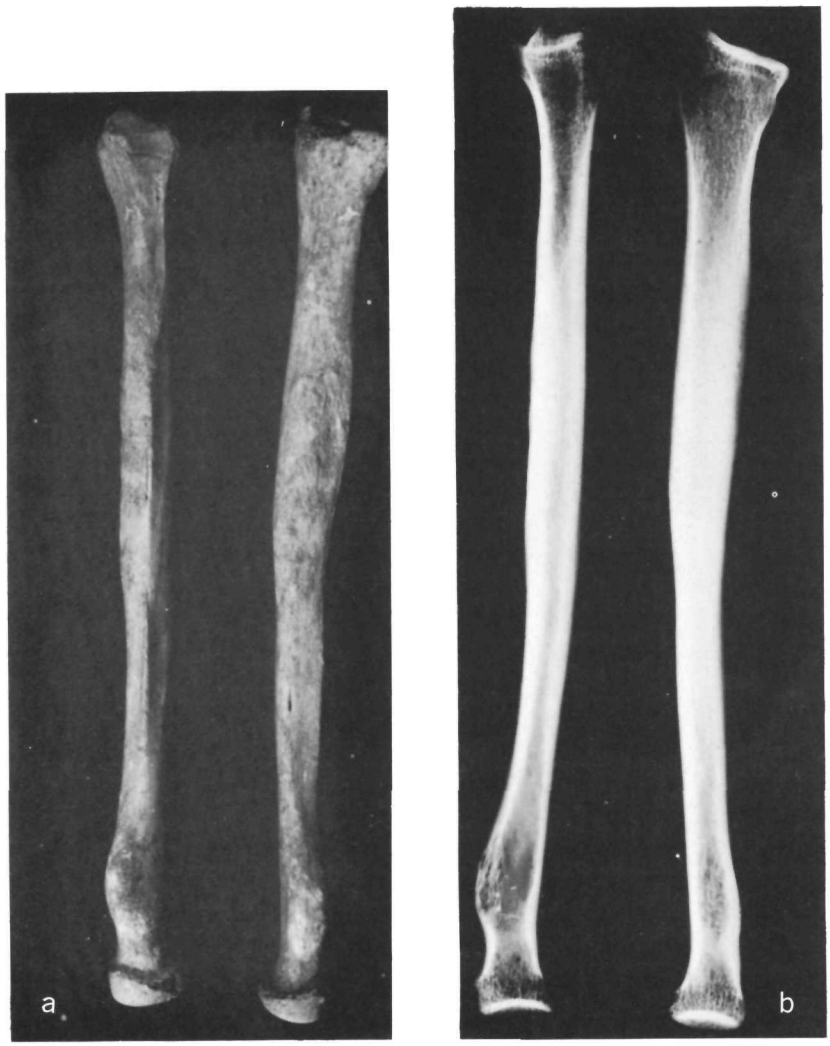


Figure 32.—*a*, Two views of periostitis of the left radius (17170/702); *b*, roentgenograms of the same radius.

that time, the theory remains an interesting speculation. Theories must evolve from exact data and probabilities, not from possibilities!

Another example of an uncertain process was neural arch defects found in three persons of this study. The term spondylolysis is used to designate simple neural arch defects either of a unilateral or bilateral nature. The defect is usually found in the pars interarticularis between the lamina and the superior articular facet process. The term spondylolisthesis is applied if the spondylolysis is associated with displacement of vertebral bodies. This superior vertebral body with all of the superimposed vertebral column is usually displaced anteriorly on the lower vertebral body. The neural arch defect is filled with fibrous or fibrocartilaginous tissue, and the area appears to be elongated. The lamina, the spinous process, and inferior articular facets of the listhetic vertebra remain normally articulated with the superior articular facets of the inferior vertebra. Thus, the vertebral column appears essentially bisected throughout the neural arch defect with the entire vertebral column and a portion of the listhetic vertebra displaced anteriorly on the lower vertebral body and the remaining laminar fragment of the listhetic vertebra.

There is also damage to the intervertebral disc between the involved vertebrae. The nucleus puplosus and annulus fibrossus are grossly damaged with fibrous tissue invasion and scar tissue formation replacing both elements of the disc. Such intervertebral disc changes would not have been detected in the Mesa Verde skeletal remains because of the disintegration of the discs along with other soft tissue. Clinically, spondylolysis and spondylolisthesis are most frequently found at the interspace between the fifth lumbar and the first sacral vertebrae. However, the defects have been identified at other levels of the vertebral column with the order of frequency being greatest in the lumbar vertebrae.

The degree of spondylolisthesis is measured by the amount of displacement of the superior vertebra on the inferior vertebrae as seen in the lateral view or roentgenogram. A

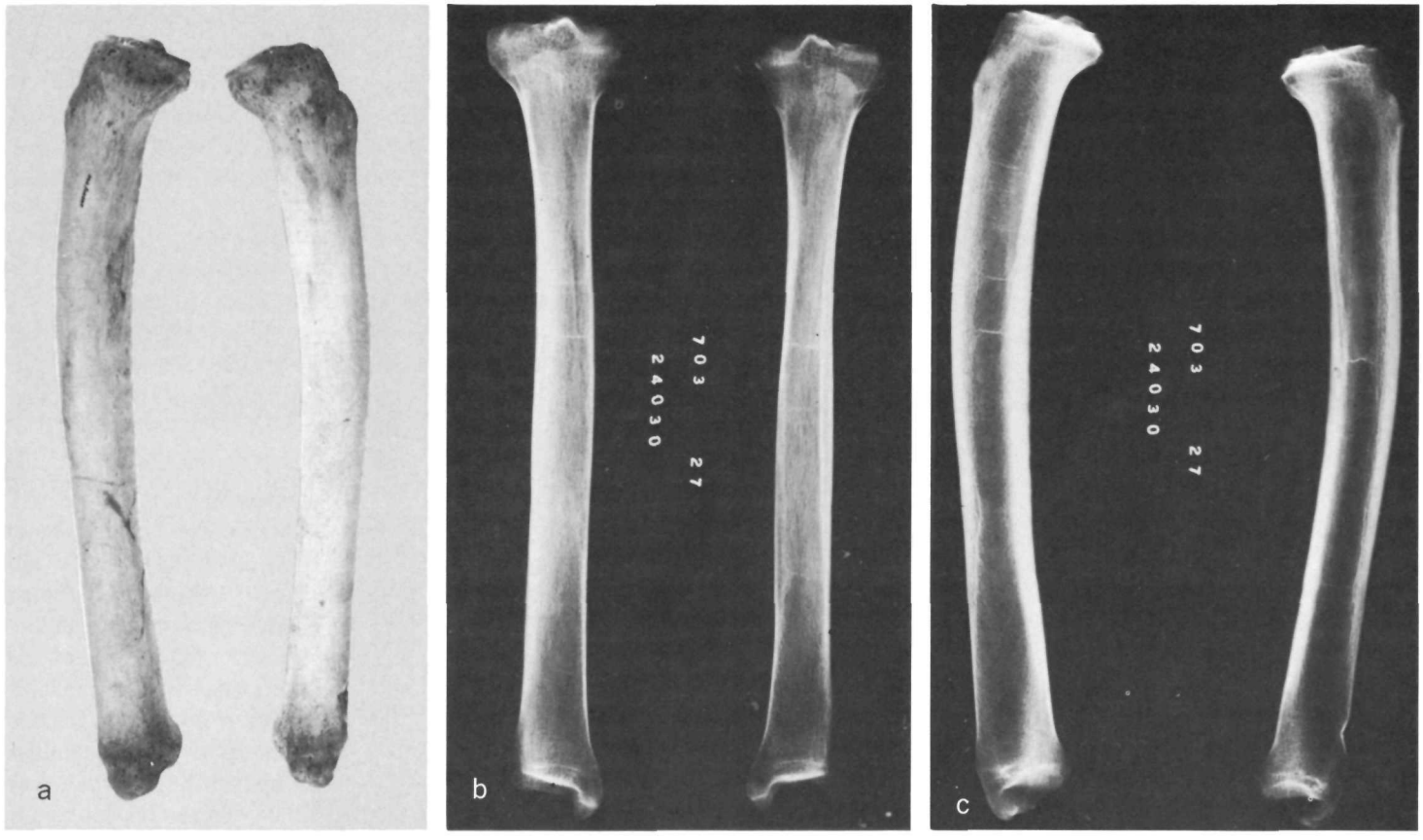


Figure 33.—*a*, Lateral view of uncertain disease of the two tibiae (24030/703); *b*, roentgenogram of the same tibiae, anterior-posterior view; *c*, roentgenogram of the same tibiae, lateral view.

displacement anteriorly of 0 to 33 percent of the width of the inferior vertebral body is termed first degree. A displacement of 34 to 67 per cent is referred to as second degree, and over 67 percent is third degree. Complete (100 percent) displacement of the superior vertebral body is termed fourth degree.

Extreme vertebral displacement (third or fourth degree) is associated with great deformity of the neural canal posteriorly. If the spondylolisthesis is above the second lumbar vertebra, the spinal cord would be the neural element at risk. Between the second and third lumbar vertebrae, potential nerve damage would involve the conus. Below the third lumbar vertebra, the neural elements potentially damaged by extreme displacement would be the nerve roots of the cauda equina. It is possible that extreme displacement could produce complete paralysis (paraplegia) below the level of displacement. Such paralysis would be evidenced by osteoporosis in the lower extremities of skeletal remains.

Burial M46 (fig. 34a, b, and c), a 30 year old male, demonstrated bilateral spondylolysis with spondylolisthesis of L5 and S1. The spondylolisthesis was of the first degree. There was degenerative arthritis involving the fifth lumbar and first sacral vertebral bodies and equally advanced arthritis of the facet joints. The facet joints of the loose laminar fragment of the fifth lumbar vertebra and the superior articular facets of the first sacral vertebra also demonstrated advanced degenerative arthritis changes. The superior articular processes of the fifth lumbar vertebra were markedly hypertrophic. The two margins of the neural arch defect in the isthmus of the fifth lumbar vertebra presented serrated and attenuated borders suggestive of symmetrical absorption of the bone rather than a destructive process. There were numerous small vascular foramina perforating the remnant on the isthmus of both sides of the arch defect. This individual also had osteochondritis dissecans of the distal right femur, probably an entirely unrelated process.

Burial 11 of Big Juniper House (fig. 35) was a 35 year old male who also demonstrated bilateral spondylolysis of the fifth lumbar vertebra. Again, the bodies of the fifth lumbar and first sacral vertebrae showed advanced degenerative arthritis, but post-mortem damage prevented an estimation of the degree of spondylolisthesis. There were no pedicles, superior articular processes, and facets due to post-mortem erosion. The lamina on the right demonstrated multiple vascular foramina. The isthmus was thin and attenuated, and it ended in a thin pointed margin.

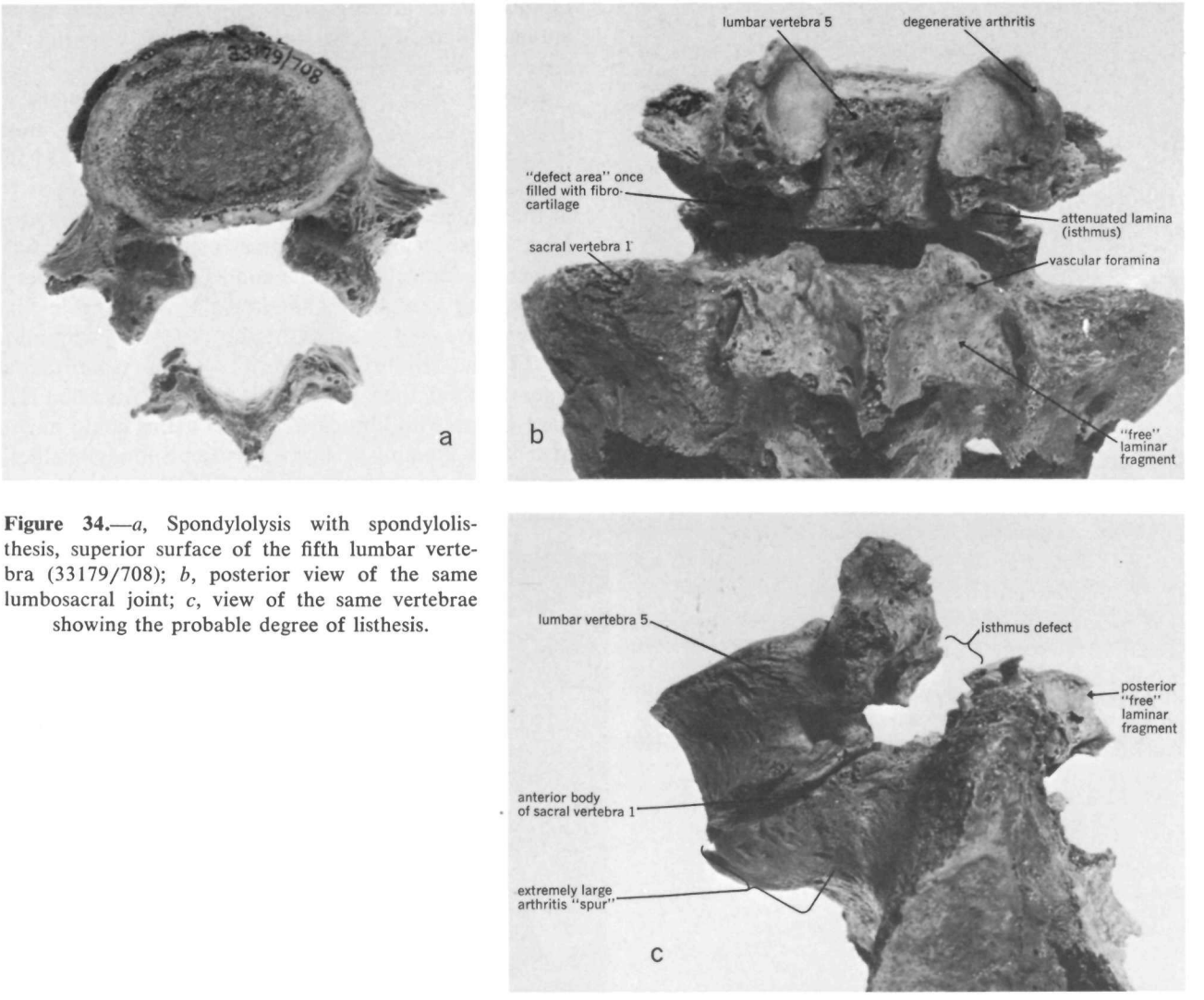


Figure 34.—*a*, Spondylolysis with spondylolisthesis, superior surface of the fifth lumbar vertebra (33179/708); *b*, posterior view of the same lumbosacral joint; *c*, view of the same vertebrae showing the probable degree of listhesis.

The third individual with a neural arch defect was Burial M5 of Mug House (fig. 36a), a 29 year old female. The neural arch demonstrated spondylolysis of the right isthmus with hypertrophy of both the left isthmus and the left inferior articular process. Spondylolisthesis of the first degree was present. Degenerative arthritis of the left lumbosacral facet joints was quite advanced with the left being more advanced than the right. The defect in the right isthmus presented a serrated border suggestive of the connection of several rounded perforations of the isthmus. Figure 36b offers a closed view of the articulated fifth lumbar vertebra and sacrum. The subluxation of the facet joint is evident, particularly on the right. The possibility that more subluxation was present in the living individual is evident in the shallow depression distal to the right inferior articular process of the fifth vertebra. On the left, the response of the sacrum was an osteophyte formation. The discoloration of the left isthmus is the result of hyperemia and could be the result of healing from an earlier spondylolytic defect. The roentgenogram (fig. 36c) shows the union of the spondylolysis area. In the left isthmus, the pseudoarthrosis "healed" with hypertrophy of the isthmus.

Since unilateral spondylolysis with spondylolisthesis is so rare, some explanation is needed. It would appear that this individual probably had bilateral spondylolysis which may have been due to trauma to an area already weakened by the perforations of the vasular foramina. Healing of the neural arch defects on the two sides was either very slow or delayed. During this delay, the spondylolisthesis occurred. Subsequently, the neural arch defect on the left healed with resultant hypertrophy of this isthmus and the articular process. Failure of healing persisted in the right neural arch.

The etiology of spondylolysis and spondylolisthesis is quite unknown. The older medical literature considers this abnormality to be a failure of the ossification centers of the vertebral elements posteriorly to fuse. Careful study indicates, however, that the vertebrae ossify from a number of centers, but that these centers do not include one above and below the isthmus area, the pars interarticularis. Multiple or anomalous ossification centers would have to be postulated, but such have never been seen in the fetus, in

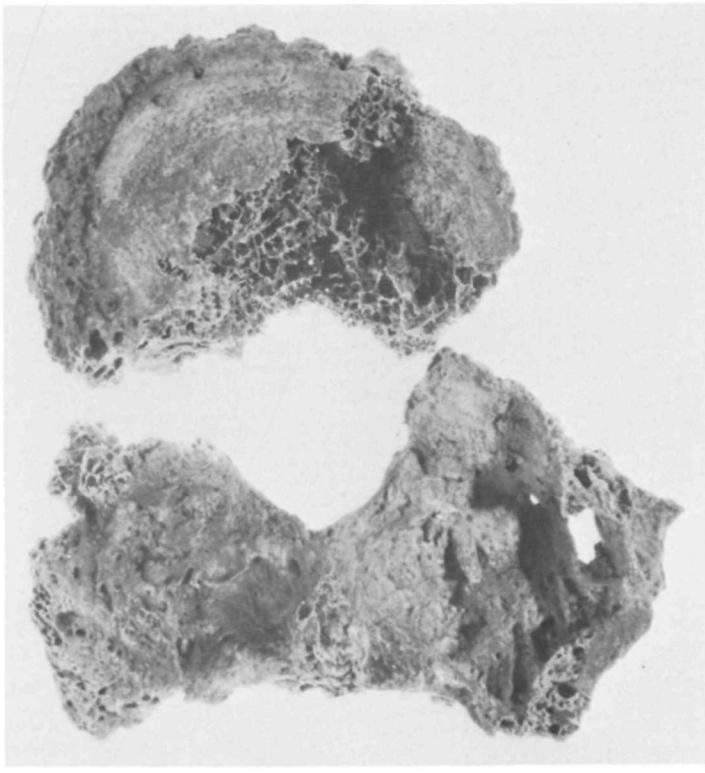
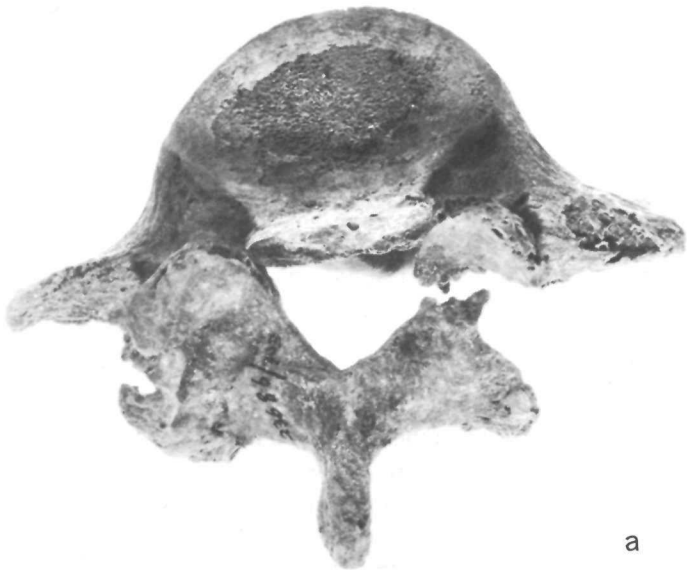


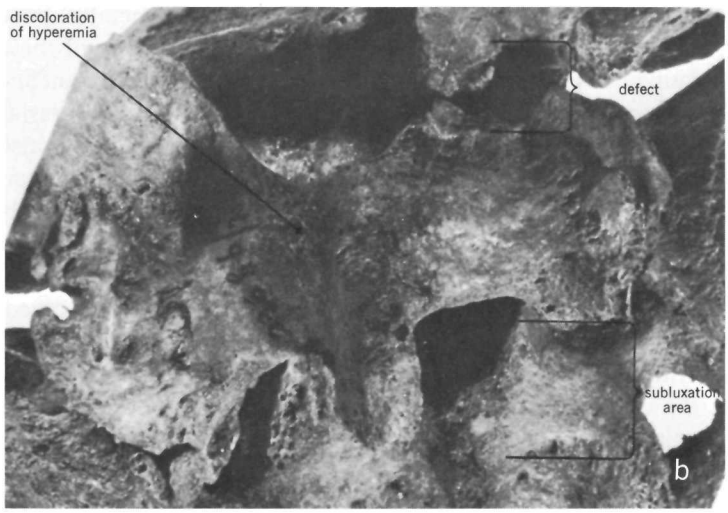
Figure 35.—Spondylolysis of the fifth lumbar vertebra (32895/710).

infants, or in growing children. Thus, failure of union of anomalous ossification centers of the posterior vertebral elements seems an unlikely possibility.

Careful study of these Mesa Verde specimens indicates that another contributing factor might be the presence of large vasular foramina in the isthmus area. All of the specimens described above demonstrated large vascular foramina in the lamina and isthmus areas. In particular, Burial M5 of Mug House demonstrated a serrated border on the isthmus defect, an indication of the connection of a number of these perforating vascular foramina. It is conceivable that a significant size and number of these vascular foramina could exist in the isthmus to weaken the area. Abnormal stress or trauma could then produce pathologic fracture. Healing of the fracture with fibrocartilaginous callus could have resulted in a large amount of fibrous tissue. Spondylolisthesis could result if the healing process were slow or incomplete.

Some specimens, M27 from Mug House (fig. 37a) and Burial R5 (36761/711) and Burial R9 (36770/711) from Two Raven House, indicated that vascular formaina exist in the isthmus area. The three cited vertebral bodies demonstrated similar and abnormally large vascular foramina. All of these foramina are quite evident with all of the soft tissue removed, but they could not be well demonstated in the roentgenogram (fig. 37c). The feeling therefore persists that these vascular foramina constituted a weakening of the neural arch that permitted fracture to occur through the area with less than the stress needed to produce fracture in a normal, non-attenuated pars interarticularis.





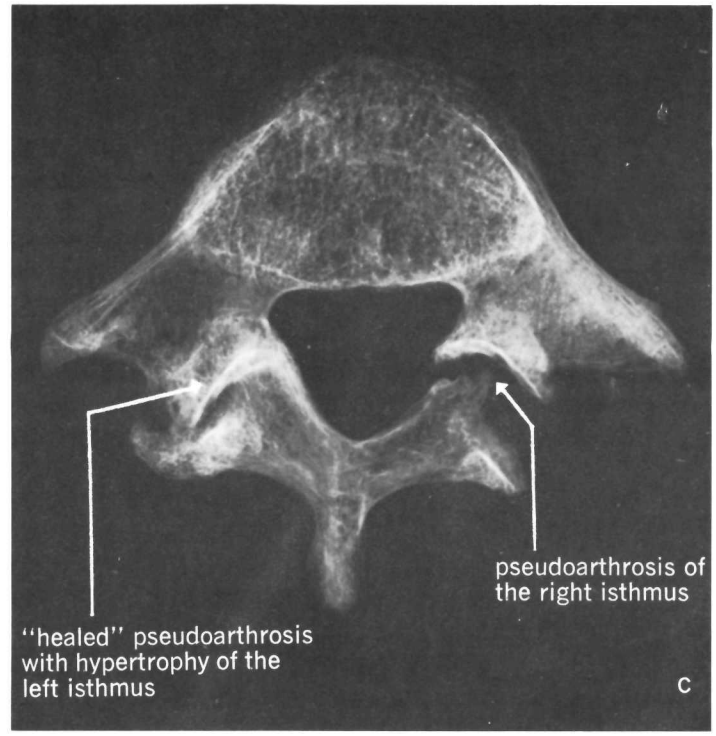
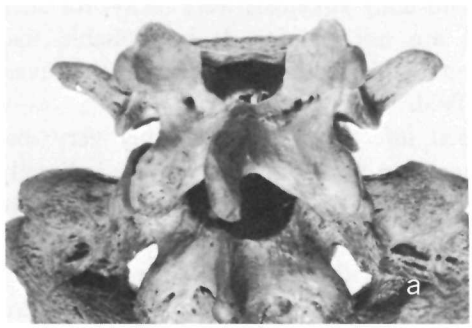


Figure 36.—*a*, Unilateral spondylolysis with spondylolisthesis of the fifth lumbar vertebra (23686/703); *b*, detail of the fifth lumbar vertebra and sacrum; *c*, roentgenogram of the same area shown in fig. 36a.



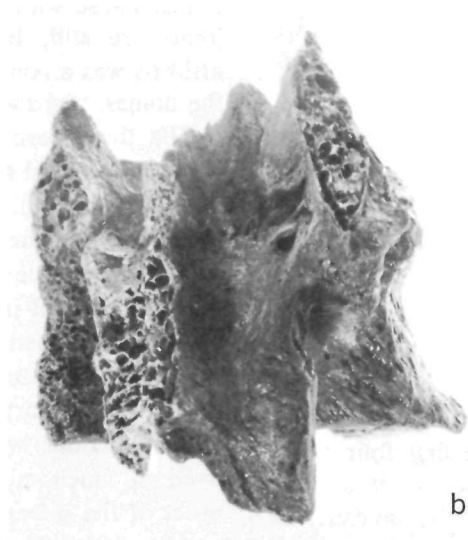


Figure 37.—*a*, Vascular foramina in the isthmus; *b*, foramina shown in the spinous process, posterior elements and a fragment of lumbar vertebra; *c*, roentgenogram in which the foramina are invisible.

3 SUMMARY

The identification and categorization of the various osseous lesions of these individuals may provide some personal satisfaction for those archeologists and physicians who obtained the specimens and examined them. Physicians, and particularly orthopedists, reading this report may also find some pleasure in the evidence of the antiquity of many diseases and processes. However, the original objective of this extensive study was to define more clearly the life and the problems of these ancient peoples. The mere identification of specific diseases may or may not provide this information depending upon the knowledge and the imagination of the reader. The interpretation of the effect of the various diseases and processes upon the "health" of these ancient individuals and of their communities is probably best provided by persons who are familiar with the entire project. Briefly stated, the disease itself may be one thing; the disability individually and collectively produced by the disease may be another.

Disability produced by a disease is quite largely a subjective reaction to a problem. One person with a disease of a particular severity may ignore it. Another person, with identical disease and severity, may become a complete invalid. To some degree, this is an individual characteristic or personality trait, but it may also be a matter of the attitude of the society or the community. The word "spartan" has become synonymous with individual or collective courage, determination, or fortitude. Thus, the ability of an individual to overcome the effects of a disease or process may be a matter of individual courage, or it may be the result of family or community pressure. We may, therefore, be able to make some general statements upon the probable effects of all of the identified osseous disease processes, but we must be cautious in expressing the opinion that this individual was disabled.

In addition, we must recognize that the spectrum of diseases we have identified in these osseous remains does not in any manner define the totality of diseases that probably plagued these peoples. For example, the bones we have examined give us no evidences of the diseases which are the major causes of death in present populations: the infectious diseases, heart diseases, vascular diseases (stroke), and cancer. We may speculate from archeological data. The average life expectancy was probably about 30 to 35 years for these ancient peoples. There was a very high infant and child mortality. If the person survived the first four years of life, it is probable that he could expect to live to a "ripe old age" in the forties or fities. Almost certainly, the cause of the high infant mortality was the high incidence of infectious disease. Such infections as pneumonia, influenza, nephritis, diarrhea, and gastro-intestinal processes were probably lethal to a far greater degree than they are in populations of today. We may also speculate that many of these persons did not live long enough to become victims of our present diseases of old-age such as arteriosclerosis, myocardial infarction, cerebral thrombosis, and cancer.

As one reflects upon the spectrum of diseases and processes that have been identified in the osseous remains, it is surprising that so few of them would have produced any symptoms or disability. Probably none of the disturbances of prenatal origin, except for the erythroblastic anemias, produced any symptoms. The erythroblastic anemias were probably fatal because of the increased susceptibility of these individuals to infections. However, some of these anemic persons could have died because of the blood dyscrasias which mark the process. The growth problems were also probably asymptomatic. The one person with osteoporosis noted in the section on metabolic problems was probably asymptomatic from the standpoint of his porosis. The two vascular lesions, osteochondritis dissecans, probably produced little if any pain in the knees of the persons involved. The three examples of neoplasia also were probably without symptoms, although it has been noted that the person with the exostosis of the radius might have had some aches and pains about the wrist as a result of the exostosis pressing upon or displacing tendons and nerves.

It is probable that the degenerative arthritis was productive of symptoms. Aches, pains, and limitations of motion surely were present in many of the persons with involved joints. Those with solid bony ankylosis were lucky, for such joints are stiff, but are not painful. It is probable that arthritis was a common subject of conversation in the kivas, the homes, and the fields.

The three described infections were probably very disabling, and could have been fatal either directly or indirectly through inanition. Certainly, the three persons were more than aware of the fact that they had a serious disease. In fact, the available osseous evidences could have been just a part of the total infectious process that the Indians experienced. The infections could have also involved more vital organs such as the lungs, or the kidneys, or the liver. The three persons survived long enough to indicate that their bone infections were in a chronic phase. They probably lived in much misery for six to twelve months after the onset of the infections.

All of the fractures probably produced temporary disability. It has already been noted that treatment for all of

the identified fractures would have been relatively simple. Indeed, lacking roentgenograms, the medicine men would probably have diagnosed many of the injuries as simple contusions, sprains, and strains. Many of the vertebral fractures would have been interpreted as being an acute exacerbation of arthritis. Certainly, none of the fractures noted would have made invalids of the persons, nor would they have been lethal.

The uncertain diseases and mysteries are more difficult to interpret. The two mysteries (an adult female and a 24 year old made) probably produced pain in the bones involved. Since we do not know the disease processes, we can not even guess as to what other symptoms might have been present elsewhere.

The neural arch defects, spondylolysis and spondylolisthesis, probably produced back pain of varying degrees of severity. Again, such would have been attributed to arthritis, and in truth, many of the symptoms were probably due to the secondary degenerative arthritis.

Our osseous evidence, therefore, indicates a surprisingly healthy population, one that "wore" poorly, but one that was capable of great physical independence.

APPENDICES

APPENDIX 1. Burials and miscellaneous bones by provenience and cultural stage, Wetherill Mesa

Site	Cultural Stage	*Distinct Burials*							Incomplete Burials
		Numbers	Adult			Sub-Adult			
			M	F	U*	M	F	U*	
Site 1676 (714)**	PI	9	5	1	3	0	0	0	12
Site 1291 (718)	PI	1	0	0	0	0	0	1	5
Two Raven House (711)	Early PI	13	6	6	0	0	0	1	27
Site 1205 (704)	Early PII	1	0	1	0	0	0	0	18
Big Juniper House (710)	PII & PIII	23	5	4	1	3	3	7	45
Badger House (706)	PII & PIII	33	12	7	2	0	0	12	57
Survey (702)	PII & PIII	5	3	1	0	1	0	0	13
Step House (709)	Early PIII	3	0	0	0	0	0	3	21
Site 1230 (715)	Early PIII	1	0	0	0	0	0	1	0
Site 1801 (719)	Early PIII	1	0	0	0	0	0	1	1
Long House (700)	PIII	41	7	5	5	0	1	23	111
Mug House (703)	PIII	39	9	5	1	0	0	24	55
Adobe Cave (708)	PIII	7	2	0	0	0	0	5	1
Site 1253 (716)	PIII	1	1	0	0	0	0	0	0
Site 1576 (717)	Late PIII	1	0	1	0	0	0	0	0
Totals		179	50	31	12	4	4	78	366

* Sex unknown.
** The numbers in parentheses refer to the site of provenience.

APPENDIX 2. Skeletal abnormalities by provenience and cultural stage, Wetherill Mesa

Site	Cultural Age	Bur. Cat.	Sex	Age	Bone	Abnormalities	Etiologic Category*
Site 1676 (714)	P I	B2 41049	M	32	Pelvis	Acetabular arthritis.	D
					Scapula	Glenoid arthritis.	D
					Vertebrae	Pyogenic spondylitis, lumbar 5 and sacral 1.	I
					Patella, L	Accessory ossification center, "Bipartite patella."	P
	P I	B5 41053	M	37	Sacrum	Facet arthritis.	D
					Femurs	Osteochondritis dissecans, R; anteversion, femoral necks, R. 33°; L 25°.	V G
					Tibiae	External torsion, R. 33°; L 25°.	G
	P I	B6	M	40	Clavicle Scapula Pelvis Vertebrae	Degenerative arthritis of all joints, vertebral bodies and facets.	D
		B8 41050 (1216/1676)	M	37	Pelvis Sacrum Vertebrae	Degenerative arthritis, acetabular, facet, vertebral body.	D
					Tibiae	*External torsion, 30° bilaterally.*	G
					Metatarsals Phalanges	Hallux valgus bilaterally, "bunions".	D
Site 1291 (718)	P I	Misc. 41051	Child		Skull	Erythroblastic anemia.	P
					Vertebrae	Congenital fusion, C-2 to C-3.	P
Two Raven House (711)	Early P II	R2 36859	F	20's	Humerus	Perforation of olecranon fossa.	P
	Early P II	R3 36760	M	18	Vertebra	Lateral bridge, C-1 vertebra.	P
	Early P II	R5 36761	F	45	Vertebrae	Degenerative arthritis, thoracic and lumbar.	D
					Vertebra	L-5, vascular foramina, isthmus.	P
					Scapula	Degenerative arthritis, glenoid.	D
	Early P II	R6 36766	M	18	Tibia	Healed fracture, metaphysis.	T
	Early P II	R7 36767	F	20	Femora	Anteversion, R 32°; L 15°.	G
	Early P II	R8 36768	M	20	Vertebrae	Degenerative arthritis, facets 2°, bodies 3°.	D
					Talus	Degenerative arthritis, sub-talar.	D
	Early P II	R9 36770	M	30	Vertebrae	Degenerative arthritis, facets 2°, bodies 1°.	D
					Vertebra	L-5, fused to sacrum, R "sacralization".	P
					Pelvis	Degenerative arthritis, acetabular.	D
					Femora	Anteversion, R 18°; L 28°.	G
					Vertebra	Vascular foramina, isthmus; Anterior wedging compression fracture.	P T
	Early II	R10 36771	M	40	Femora	Anteversion, R 38°; L 22°.	G
						Degenerative arthritis, condyles, 1°.	D
					Metatarsal I	Hallux valgus, degenerative arthritis "bunion".	D
					Pelvis	Degenerative arthritis, acetabular.	D
					Vertebrae	Degenerative arthritis, 2° and 3°.	D
					Sacrum	Degenerative arthritis.	D
						Sacral spina bifida.	P
	Early P II	R11 36772	F	30	Vertebrae	Degenerative arthritis, sacral, facets.	D
	Early P II	R12 36773	F	18	Vertebrae	Degenerative arthritis, C-1 and C2, lumbar.	D
	Early P II	Misc. 36663	U	Adult	Humerus	Degenerative arthritis, shoulder 2°, elbow 1°.	D
	Early P II	Misc. 36661	U	Adult	Vertebrae	Degenerative arthritis, facets 4°, bodies 3° and 4°.	D
					Pelvis	Degenerative arthritis, acetabular, 1°.	D
Site 1205 (704)	Early P II	1 23665	F	40	Vertebrae	Degenerative arthritis, cervical, 1°.	D
Big Juniper House (710)	Early P II	B3 32883	F	Teenager	Femora	Anteversion, 50° bilaterally.	G
	P II	B8 32890	M	32	Scapula	Perforation of blade above spine.	P
					Vertebrae	Degenerative arthritis, lumbar, 2°.	D
					Talus	Degenerative arthritis, ankle, 1°.	D
					Clavicle	Fracture, healed, medial end.	T

APPENDIX 2. Skeletal abnormalities by provenience and cultural stage, Wetherill Mesa—(Continued)

Site	Cultural Age	Bur. Cat.	Sex	Age	Bone	Abnormalities	Etiologic Category*
Big Juniper House (710) (cont.)	P II	B9 32897	M	37	Vertebrae	Degenerative arthritis, 4°, lumbar.	D
						Fracture-dislocation with rotational deformity residual, lumbar.	T
	Early P II	B11 32895	M	25	Vertebrae	Degenerative arthritis, facets and bodies 2°.	D
						L-5 vertebra, vascular foramina, isthmus.	P
						Spondylolysis and spondylolisthesis, L-5 on S-1.	U
						Thoracic vertebra, lower, lateral wedge fracture.	T
	P II	B12 32899	U	Infant	Skull	Erythroblastic anemia.	P
	P II	B13 33539	U	Infant	Skull	Erythroblastic anemia.	P
	P II	B17 33547	M	32	Scapula	Degenerative arthritis, glenoid.	D
					Ulna	Degenerative arthritis, elbow.	D
					Vertebrae	Degenerative arthritis, facets and bodies.	D
					Tibia	External torsion 30°.	G
	Late P II	B22 33555	F	Adult	Pelvis	Degenerative arthritis, acetabular, 1°.	D
	P II	B24 37282	F	Over 40	Pelvis	Degenerative arthritis, acetabular, 1°.	D
	P II	Misc. 31767	U	Adult	Vertebrae	Degenerative arthritis, bodies.	D
	P III	Misc. 31775	U	Adult	Patella	Degenerative arthritis.	D
	P II or P III	Misc. 33554	U	Child	Skull	Erythroblastic anemia.	P
Badger House (706)	Late P II	B2 27262	M	35	Vertebrae	Degenerative arthritis, thoracic, 1°.	D
					Humerus	Degenerative arthritis, shoulder, 1°.	D
	P II or P III	B4 28062	M	45	Vertebrae	Degenerative arthritis, thoracic bodies, 1°; lumbar bodies, 4°.	D
						Compression fracture, thoracic.	T
					Mandible	Edentulous.	D
	P II or P III	B5 28063	M	Adult	Pelvis	Degenerative arthritis, acetabular, 1°.	D
					Vertebrae	Degenerative arthritis, L-4 and L-5, facets.	D
	P II	B8 28903	M	45	Femora	Degenerative arthritis, knee, 1°.	D
					Tibiae	Degenerative arthritis, knee and ankle, 1°.	D
					Pelvis	Degenerative arthritis, acetabular, 1°.	D
					Vertebrae	Degenerative arthritis, facets, 1°, bodies, 2°.	D
					Talus	Degenerative arthritis, ankle and sub-talar.	D
					Patellae	Degenerative arthritis, 1°.	D
	P II or P III	B11 28906	M	37	Pelvis	Protrusio-acetabuli, left.	I
					Vertebrae	Degenerative arthritis with fusion of two thoracic laminae.	D
					Sternum	Fracture healed, malunion.	T
	P II or P III	B12 28907	M	27	Femur	Degenerative arthritis, knee, 1°.	D
					Tibia	Osteocartilaginous exostosis, R.	Np
					Clavicle	Fracture, healed.	T
					Mandible	Degenerative arthritis, T-M joint.	D
	P II or P III	B13 28908	F	48	Femora	Degenerative arthritis, knees, 2°.	D
					Vertebrae	Degenerative arthritis, cervical,	D
	Late P III	B15 28910	M	22	Tibiae	with fusion of C-4 to C-5.	
						External torsion, 45°, bilaterally.	G
					Femora	Anteversion, necks, 25°, bilaterally.	G
					Metatarsal 1	Fracture, shaft, healed, shortened.	T
	P II or P III	B20 28915	F	30	Vertebrae	Degenerative arthritis, sacral facets, 1°.	D
	Late P II	B21 28916	F	42	Radius	Fracture, healed, distal end; Colles' fracture, dorsal angulation deformity.	T
	P II	B24 28919	M	30	Vertebrae	Degenerative arthritis, odontoid-C-2.	D
						Prenatal fusion of C-2 to C-3.	P
	Early P II	B25 28920	U	13	Sacrum	Fusion anomaly: S-1 transverse process, L not fused to S-2 transverse process.	P
	Late P II	B27 28923	M	28	Vertebra	Lateral bridge, C-1 vertebra.	P

APPENDIX 2. Skeletal abnormalities by provenience and cultural stage, Wetherill Mesa—(Continued)

Site	Cultural Age	Bur. Cat.	Sex	Age	Bone	Abnormalities	Etiologic Category*
Badger House (706) (cont.)	Early P II	B29 28925	F	25	Rib	Fracture, healed.	T
	Late P II	B33 35143	U	9	Vertebra	Odontoid; accessory ossification center.	P
					Femora	Anteversion; R. 30°; L, 25°.	G
		Misc. 27628	U	Adult	Vertebrae	Degenerative arthritis; thoracic and lumbar bodies, 1° and 2°; lumbosacral facets.	D
		Misc. 27717	U	Adult	Vertebrae	Degenerative arthritis, lumbar, 1°.	D
		Misc. 29515	U	Adult	Vertebrae	Degenerative arthritis, lumbar, 1°.	D
		Misc. 29012	U	Adult	Vertebrae	Degenerative arthritis, lumbar, 1°.	D
		Misc. 27723	U	Adult	Vertebra	Fracture, healed, thoracic, wedging.	T
		Misc. 27436	U	Adult	Vertebra	Degenerative arthritis, odontoid, 1°.	D
		Misc. 28628	U	Adult	Talus	Degenerative arthritis, sub-talar joint, 1°.	D
Survey (702)		Misc. 27516	U	Adult	Metatarsal I	Degenerative arthritis, hallux valgus "bunion".	D
	Late P III	Misc. 17187	M	40	Tibiae	External torsion; R 45°, L 50°.	G
						Degenerative arthritis, knee, 1°.	D
					Femora	Degenerative arthritis, knee.	D
					Vertebrae	Degenerative arthritis, all vertebrae, 3°.	D
					Clavicle	Degenerative arthritis, sterno-calvicular, 1°.	D
					Patella	Accessory ossification center, "Bipartite patella".	D
	Late P II	Misc. 17170	F	Adult	Navicular (tarsal)	Degenerative arthritis.	D
					Radius	Periostitis.	U
		Misc. 13447	M	Adult	Vertebrae	Degenerative arthritis; bodies, 1°; facets, 2°.	D
		Misc. 13432	U	Adult	Radius	Fracture, healed, radial head, malunion.	T
	P III	1 33654	M	37	Femora	Anteversion; R 34°; L 32°.	G
					Vertebrae	Degenerative arthritis; thoracic.	D
Step House (709)	Early P III	Misc. 31880	U	Adult	Vertebrae	Degenerative arthritis; lumbar bodies, 3°.	D
	Early P III	Misc. 31896	U	Adult	Vertebrae	Degenerative arthritis; thoracic.	D
	Early P III	Misc. 31874	U	Adult	Vertebrae	Degenerative arthritis; lumbar bodies, 3°.	D
	Early P III	Misc. 31892	U	Adult	Vertebrae	Degenerative arthritis; thoracic bodies, 2°.	D
	Early P III	Misc. 34344	U	Adult	Vertebrae	Degenerative arthritis; lumbar bodies, 3°.	D
	Early P III	Misc. 31894	U	Adult	Vertebrae	Degenerative arthritis; lumbar bodies.	D
1801 (719)	Early P III	1 41052	U	5	Rib	Bifid rib.	P
Long House (700)	P III	L4 13639	F	30	Femora	Anteversion, 35°, bilaterally.	G
	P III	L5 13640	M	40	Calcaneus	Degenerative arthritis.	D
					Patella	Degenerative arthritis.	D
	P III	L7 13642	F	40	Vertebrae	Degenerative arthritis, cervical, C-1 to C-2.	D
	P III	L8 13643	M	Old	Femur	Anteversion, R 30°.	G
					Tibiae	Degenerative arthritis, 1°.	D
					Femora	Degenerative arthritis, knees, 3°.	D
					Patellae	Degenerative arthritis, 2°.	D
					Vertebrae	Degenerative arthritis, bodies, thoracic and lumbar.	D
					Scapulae	Degenerative arthritis, 1°.	D

APPENDIX 2. Skeletal abnormalities by provenience and cultural stage, Wetherill Mesa—(Continued)

Site	Cultural Age	Bur. Cat.	Sex	Age	Bone	Abnormalities	Etiologic Category*
Long House (700) (cont.)	P III	L11 13646	M	30	Vertebrae	Degenerative arthritis, bodies and facets, cervical and sacral, 1°.	D
	P III	L12 13647	U	Adult	Vertebrae	Degenerative arthritis, facets 2°, with facet subluxation.	D
	P III	L15 13650	F	Adult	Scapula	Degenerative arthritis, glenoid, 2°.	D
	P III	L16 13651	M	Adult	Femora	Anteversion, 35° bilaterally.	G
					Calcaneus	"Cyst" of calcaneus.	Np
	P III	L18 13653	F	30	Femora	Anteversion, R 30°; L 27°.	G
	P III	L24 13695	M	Old	Sacrum	Degenerative arthritis, facets, 1°.	D
					Ribs	Fusion, prenatal, ribs 1 and 2.	P
					Vertebrae	Degenerative arthritis.	D
						Fracture, compression, T-12 or L-1.	T
						Osteoporosis.	M
					Femora	Degenerative arthritis, knees, 1°.	D
					Tibiae	Degenerative arthritis, knees, 2°.	D
					Talus	Degenerative arthritis, ankle, neck of talus.	D
					Humeri	Degenerative arthritis, shoulders, 2°.	D
	P III	L29 13665	U	10	Scapula	Omohyoid bone.	P
	P III	Misc. 14248	U	Adult	Patella	Degenerative arthritis, 1°.	D
					Metatarsal	Degenerative arthritis, head of metatarsal I,	D
	P III	Misc. 27478	U	Adult		hallux valgus, "bunion", mild.	
	P III	Misc. 20069	U	Child	Femur	Osteomyelitis, pyogenic.	I
	P III	Misc. 19494	U	Adult	Tibiae	External torsion, 45° bilaterally.	G
					Talus	Neck-body angle greater than 38°.	G
	P III	Misc. 19489	U	Adult	Vertebrae	Degenerative arthritis, lumbar.	D
Mug House (703)	P III	M1 2866/172	M	Adult	Vertebrae	Lateral bridge, C-1 vertebra.	P
					Vertebrae	Degenerative arthritis, bodies and facets, thoracic and lumbar, subluxation of L-4 on L-5.	D
					Tibiae	External torsion, R 32°; L 25°.	G
					Patellae	Accessory ossification center, "Bipartite patellae".	P
					Metatarsal	Degenerative arthritis, head of metatarsal, hallux valgus, "bunion".	D
					Metacarpal	Degenerative arthritis, base of metacarpal of thumb.	D
	P III	M3 2884/175	F	Adult	Tibiae	External torsion, 30° bilaterally.	G
	P III	M4 2886/175	U	Infant	Ribs	*Fractures, healing three ribs.*	T
	P III	M5 23686	F	29	Skull	Degenerative arthritis, temporo-mandibular	D
					Mandible	joints, destruction of condyles of mandible, subluxation of joints.	I
					Vertebrae	Spondylolysis, L-5, unilateral, with spondylolisthesis.	U
						Degenerative arthritis, bodies, 3°; facets 4°.	D
					Metatarsal	Degenerative arthritis, head of metatarsal, hallux valgus, "bunion".	D
	P III	M6 23687	M	33	Phalanges	Prenatal fusion of 3 phalanges, finger 5.	P
						Fracture, phalanx with residual angulation of condyles, malunion.	T
					Vertebrae	Degenerative arthritis, lumbar, facets 1°; bodies 2°.	D
						Degenerative arthritis, C-1 and C-2, and thoracic.	T
					Vertebrae	Compression fracture, L3.	
					Pelvis	Degenerative arthritis, acetabular.	P
	P III	M12 23694	U	11	Vertebra	Cervical vertebra, division of vertebral artery foramina, C-7.	G
	P III	M15 24013	F	43	Tibiae	External torsion, 35° bilaterally.	D
					Vertebrae	Degenerative arthritis, facets, 1°.	D
	P III	M16 24019	M	40	Humerus	Degenerative arthritis, shoulder, 2°.	D
					Scapula	Degenerative arthritis, glenoid, 1°.	D
					Vertebrae	Degenerative arthritis, bodies, 1°.	D

APPENDIX 2. Skeletal abnormalities by provenience and cultural stage, Wetherill Mesa—(Continued)

Site	Cultural Age	Bur. Cat.	Sex	Age	Bone	Abnormalities	Etiologic Category*
Mug House (703) (cont.)	P II	M21 24024	F	21	Vertebra Mandible	Compression fracture, minimal, T-12. Degenerative arthritis, temporo-mandibular.	T D
	P III	M22 24025	F	36	Ulnae Vertebrae	Degenerative arthritis, elbows. Degenerative arthritis, facets, 4°.	D D
	P III	M23 24026	U	Infant	Skull	Erythroblastic anemia.	P
	P III	M25 24028	M	36	Vertebrae	Degenerative arthritis, lumbar, 1°.	D
	P III	M27 24030	M	24	Tibiae Vertebrae	Bowed, thickened shafts. Lumbar rib, L. Vascular foramina, isthmus.	U P P
	P III	M30 24033	M	43	Ulnae Radius Scapula Clavicle Rib Vertebrae	Degenerative arthritis, elbows. Osteocartilaginous exostosis, distal. Degenerative arthritis, glenoid, 2°. Fracture, healing, medial end. Fracture, healed. Degenerative arthritis, thoracic, facets 2°, fusion of bodies. Compression fractures, thoracic, 2 vertebrae. Lateral bridge, C-1 vertebra.	D Np D T T D T P
	P III	M38 25436	U	Infant	Femora	Anteversion, femoral necks.	P
	P III	Misc. 23689	U	Adult	Vertebra	Lateral bridge, C-1 vertebra.	P
	P III	Misc. 18352	U	U	Skull	Erythroblastic anemia.	P
	P III	Misc. 19122	U	Adult	Clavicle	Fracture healed.	T
Adobe Cave (708)	Late P III	M41 27247	U	2½	Skull Vertebrae	Partial closure, left coronal suture. Six lumbar vertebrae.	P P
	Late P III	M43 33176	M	35	Ribs Humerus Patellae Scapula Pelvis Vertebrae	Degenerative arthritis with fusion of first ribs to sternum, bilaterally. Degenerative arthritis, shoulders, 1°. Accessory ossification centers, bilaterally, "Bipartite patellae". Degenerative arthritis, glenoid, 1°. Degenerative arthritis, acetabulum. Degenerative arthritis, thoracic, lumbar and sacral, bodies 2° and 3°, facets 4° with fusion of T-8 through T-11.	D D P D D D
	Late P III	M46 33179	M	30	Vertebrae Scapula Femur Cuboid	Spondylolisthesis, L-5 on S-1. Degenerative arthritis, thoracic and lumbar. Degenerative arthritis, glenoid, 1°. Degenerative arthritis, knee. Osteochondritis dissecans. Accessory ossification center.	U D D D V P
Site 1253	P III	1 FS9/1253	M	32	Vertebrae Femur	Degenerative arthritis. Degenerative arthritis.	D D

* Etiologic Categories: P—prenatal; G—growth; M—metabolic; D—degenerative; I—infecton; T—trauma; Ng—neurogenic; Np—neoplastic; V—vascular; U—uncertain.

GLOSSARY

Achondroplasia—A dominantly inherited (autosomal) disturbance of chondroblastic growth and maturation resulting in dwarfism with a normal sized head, slight shortening of the trunk, and marked shortening of the extremities.

Ankylosis—The spontaneous osseous fusion of a joint produced by a disease process. Ankylosis is to be compared with arthrodesis which is a surgically produced osseous fusion of a joint.

Annulus fibrosus—The tough, fibrous outer ring of the intervertebral disc which surrounds the gelatinous nucleus pulposus. The annulus is firmly attached to the adjacent cortices of the vertebral bodies superiorly and inferiorly.

Anteversion (of the femoral neck)—The relationship of the long axis of the femoral neck to the transverse axis of the two condyles of the distal femur. The femoral neck axis is normally directed anteriorly to the axis of the condyles. In the adult, this anterior direction of the femoral neck axis is 18° to 20°. In the infant, it is up to 30°.

Arthrokatadysis—A medial depression or migration of the floor of the acetabulum with concomittant medial migration of the femoral head. The deformity produces limitation of motion in the hip joint. The term is synonymous with protrusio-acetabuli. Many processes or diseases may produce the deformity with infection, osteomalacia, rickets, and arthritis being most common. Infrequently, it is bilateral and is the result of a genetic defect. Trauma (injury) may also produce the deformity. In this instance, the individual must receive a major force acting on the lateral aspect of the hip. The femoral head is thus driven medially through the floor of the acetabulum where a stellate fracture is produced. The fracture of the floor of the acetabulum will heal in the deformed position.

Ballooning of the discs—The enlargement of the central portion of the intervertebral disc in the superior to inferior direction. Expansion of the disc is usually caused by increased water content of the nucleus pulposus and the pressure produced by such results in the indentation and deformity of the adjacent vertebral cortices. The cortices of the vertebra appear to be concavely depressed into the vertebral bodies.

Biconcavity (of the vertebral bodies)—The seeming depression of adjacent vertebral cortices into the vertebral bodies on the two sides (superiorly and inferiorly) of an intervertebral disc. This usually results from osteoporosis of the vertebral bodies and ballooning of the disc.

Bicondylar axis (of the femur)—The transverse axis of the two condyles of the distal femur.

Bipartite patella—The development of the patella from two ossification centers in the cartilaginous patella instead of a single ossification center, and the failure of these two ossification centers to unite with maturation of the individual. The patella is one of the bones which develops in a cartilaginous model, the cartilage being replaced in the process of enchrondral ossification which develops in an ossification center within the cartilage model.

Compression fracture—A fracture which results from a compressive force along the long axis of a bone. The term is frequently applied to a fracture of a vertebral body which results from a force applied along the long axis of the vertebral column (body).

Coxa vara—A deformity of the proximal femur in which the angle formed by the longitudinal axis of the femoral neck and the longitudinal axis of the femoral shaft is reduced to less than the normal 135°.

Degenerative arthritis—A diseased state of a joint produced by degeneration of the articular cartilage. The cartilage is replaced by bone, producing sclerosis (increased density in the roentgenogram) of the articular cortex and narrowing of the articular cartilage space in the roentgenogram. This new bone formation produces spurs of bone about the periphery of the joint. Joint function is limited. The process is considered to be "primary" if no known cause is apparent. The process is "secondary" if the diseased state or degeneration follows a known cause, such as infection of the joint, or fracture of the joint surfaces.

Dwarfism—The abnormal decrease of the vertical height of a person. This must be related to the expected height of an individual of that race and culture. It may be a simple, symmetrical, proportionate shortening of both trunk and extremities (a man in the miniature); or, more commonly, it may result from marked shortening of the extremities with proportionately less shortening of the trunk, as in the achondroplastic dwarf.

Dysplasia (of the hip)—An abnormal or faulty growth of the hip joint without dislocation (luxation) of the joint producing an incongruity of the articular cortices of the femoral head and the acetabulum. Usually, there is a degree of partial dislocation (subluxation) of the joint. The process is multifactorial with genetic, hormonal, and environmental influences being implicated.

Erythroblastic anemia—A decrease in the number of blood cells produced by a failure of the maturation process of the erythrocytes. Normally formed in the marrow of the bones, the mature erythrocyte develops from a nucleated erythroblast which loses its nucleus in the process of maturation. Most of the erythroblastic anemias are now assumed to be autosomal recessive diseases. The thalassemias are one example.

External torsion (of the tibia)—An excessive outward rotation of the distal end of the tibia on the long axis of the tibia. Specifically, this is measured by relating the transverse axis of the distal tibia to the transverse axis of the proximal tibia. Excessive outward rotation (torsion) of the tibia would result in the turning outward of the ankle joint and foot, the opposite of "pigeon toed" position.

Facet arthritis—Degenerative arthritis of the facet joints between vertebrae. The facet joints are the articulation of the superior articular facet process of one vertebra with the inferior articular facet process of the vertebra superior to it. The facet processes are a part of the posterior elements of the vertebra which surround the spinal canal and spinal cord. The posterior elements arise from the posterior surface of the vertebral body and include (in order) two pedicles, two superior articular facet processes, two transverse processes, two laminae (which fuse posterior to the spinal cord in the mid-line), two inferior articular facet processes, and one spinous process.

Genu valga—An angular deformity of the lower extremities beginning at the knee joint in which the knee joints are abnormally close together and the ankle joints excessively wide apart (knock knees).

Genu vara—An angular deformity of the lower extremities beginning at the knee joint in which the knee joints are abnormally sepa-

rated and the ankle joints are abnormally close together (bow legs).

Involucrum—An envelope or sheath of new, living, reactive bone laid down by the periosteum, and covering a mass of dead bone, the sequestrum.

Listhetic—A displaced or slipped bone. Usually the term is used in relation to the vertebrae (spondylo). The superior vertebra is usually slipped anteriorly to the inferior vertebra. (See spondylolisthesis.)

Mal-union—The union of a fracture with residual fracture deformity of angulation, displacement, over-riding, rotation, distraction or impaction. Usually as a result of the residual fracture deformity, there is decreased function of the part.

Metatarsus adductus—A deformity of the foot in which the fore part of the foot is deviated medially toward the mid-line of the body. This deformity is very frequently a part of a clubfoot deformity (talipes equinovarus), and sometimes is called "one-third of a clubfoot."

Non-union—The failure of union of a fracture with osseous tissue. The fracture may unite with fibrous or cartelaginous tissue which is invisible roentgenographically.

Nucleus pulposus—The central part of the intervertebral disc. This is a gelatinous mass surrounded by the annulus fibrosus. The nucleus is the remnant of the embryonic notochord. The nucleus is frequently thought to be a "shock absorber" between two vertebral bodies.

Osteoarthritis—Degenerative arthritis. Osteoarthritis is a clinical term while degenerative arthritis is a pathologic term. The two terms are synonymous. The lay term is simply "arthritis."

Osteoarthrosis—Degenerative arthritis or osteoarthritis. Osteoarthrosis is frequently used clinically to denote a very mild or benign form of arthritis or osteoarthritis of non-inflammatory character. Some physicians attempt to distinguish between an arthritis which they consider to be inflammatory in nature, and an arthrosis which they consider to be non-inflammatory. The distinction is vague and not generally accepted.

Osteocartilaginous exostosis—A tumorous growth on a bone producing a projection of bone and cartilage from the surface of the host bone. The growth may appear as a single bulge or "blow-out" of the medullary cavity and is then termed "sessile." The growth may have only a thin stalk within a large bulbous end covered with cartilage. If this is the case, it is called "pedunculated."

Osteochondritis dissecans—A dissection or separation of a fragment of bone and articular cartilage from the host bone's articular surface. The fragment becomes necrotic as a result of its removal from the vascular supply. The fragment may be incompletely separated from the host bone. It may be completely separated but remain in the cavity of the host bone. Or, it may be extruded into the joint and float freely about in the joint cavity as a "loose body."

Osteogenesis imperfecta—A dominantly inherited (autosomal) condition in which the bones are abnormally brittle and subject to fracture. These persons may have literally hundreds of fractures which may result from the most trivial injury. The bones are imperfectly formed with very thin cortices and very enlarged medullary cavities. The process results from a primary defect of collagen formation which produces a very limited amount of protein matrix of bone. Mineralization of the matrix is probably also defective. Another feature of the process is "blue sclerae", an abnormal blueness of the eyeball due to imperfect formation of the coats of the eyeball.

Osteomalacia—A condition marked by abnormal or deficient mineralization of the protein matrix of bone usually resulting from a deficiency of vitamin D. In infants and children, the syndrome is called rickets. The process produces marked weakness of the bones, and they are subject to multiple fracture. The fractures usually heal, but since they are so frequent, fracture deformities occur and remain uncorrected. The result is a very deformed bone. Osteomalacia may result from calcium deficiency or from excessive calcium loss. It is more common in females who are pregnant and transferring calcium to the fetus, or who are lactating. This process produces a change in the *quality* of bone.

Osteone—The basic unit of organization of compact (cortical) bone. It consists of central vessels and concentrically arranged lamellae of bone, of which there may be 4 to 20, each 3 to 7 microns thick. The individual osteone may be 250 to 300 microns in diameter and up to 4 centimeters in length. The osteones are oriented along the long axis of the bone, and usually pass obliquely from periosteal surface to endosteal surface of the cortex. The osteones are frequently called "Haversian" systems since they were first described by Clopton Havers, an English physician and anatomist.

Osteopetrosis—A dominantly inherited (autosomal) condition of bone in which there is markedly excessive bone formation filling the medullary canal of the bone with randomly formed bone. Radiographically, there is a loss of distinction between the cortex of the bone and the medullary cavity, and the whole bone seems to be a simple dense object. The bone is very fragile. Also called "marble bones" disease, and Albers-Schonberg disease.

Osteoporosis—A condition of bone in which there is a decreased amount of bone, but that bone which is present is of normal quality. There is, therefore, a decrease in the *quantity* of bone, but there is no change in the *quality* of bone formed. This is to be distinguished from osteomalacia in which there is a decrease in the *quality* of bone, due to a defect in mineralization of bone. Osteoporotic bones are more porous to the x-ray, are fragile, and are subject to fracture. The process is multifactorial with decreased usage and stress on bone being the most frequent cause. It may also be hormonal, and is frequently seen in the post-menopausal female.

Osteophyte—A spur of bone adjacent to an articular surface, seen most commonly in degenerative arthritis.

Paget's Disease—A disease of bone of unknown etiology originally described by Sir James Paget. The process most frequently involves the pelvis, the femur, the tibia, and the skull. Other bones are less frequently involved. The disease produces thickening of the cortex of bone, enlargement of the bones, and very frequently, bowing of the bone. In the tibia, the bone is frequently bowed anteriorly, producing a "saber shin". The skull may become grossly enlarged. Radiographically, there may be incomplete fractures which seem to progress transversely through only one cortex of the bone.

Pars interarticularis (of the vertebra)—A zone of the posterior elements of the vertebra between the superior articular facet process and the lamina. (See also facet arthritis, spondylolysis, and spondylolisthesis.)

Pronation—Rotation of the forearm at the elbow joint so that the palm of the hand is turned downward.

Protrusio acetabuli—See arthrokatadysis.

Pyogenic—An inflammatory process or infection which produces pus.

Pyogenic osteomyelitis—An infection (itis) of bone (osteo) and the medullary contents (myel). The infection may result from an open wound which extends into the bone from an open (compound) fracture, and in this instance, is termed exogenous osteomyelitis. The infection may also result by spread of bacteria via the blood stream from another focus of infection, such as a boil, or tonsillitis, and in this instance, the term hematogenous osteomyelitis is applied. The infection in bone may produce death (necrosis) of a portion of bone (sequestrum). The sequestrum may become separated from the bone and may be extruded to the outside through a sinus.

Radiating trabeculae—Spicules or trabeculae of cancellous (spongy) bone that radiate from a focus. Usually they are a reaction to the disease process of the focus and are produced by the periosteum of the bone.

Rheumatoid ankylosing spondylitis—A peculiar form of rheumatoid arthritis usually limited to the vertebral column. It is distinguished from rheumatoid arthritis immunologically. The process produces ankylosis of the vertebral column by bone formation around the intervertebral discs and across the facet joints. It may produce complete ankylosis of the vertebral column from the first cervical vertebra to the sacrum. Frequently involved also are the sacro-iliac joints. It occurs most frequently in males. The process is also called "poker spine", "bamboo spine" (because of its radiologic appearance), and Marie-Strumpell's disease.

Rheumatoid arthritis—An inflammatory process of joints (rheumatism) which usually has many excerbations and remissions. The joints are painful, and the person may become severely disabled through the actual destruction of the joints, including the articular cortices and cartilages. Since the joints are painful, there is decreased usage of the extremities, and the bones may become osteoporotic (atrophic). The disease most frequently involves females, and most frequently, the peripheral joints of the hands and feet. However, it may also involve other joints of the extremities, with multiple joint involvement being most common. It is distinguished immunologically from rheumatoid ankylosing spondylitis.

Rickets—A condition in infants and children marked by abnormal or deficient mineralization of the protein matrix of bone, usually caused by deficiency of vitamin D. It is usually due to dietary deficiency, but there is a form of the disease which is hereditary and is called vitamin D resistant rickets. (See osteomalacia.)

Sacral spina bifida—A defect in the posterior elements of a sacral vertebra in which the laminae fail to unite in the mid-line posteriorly. Additionally, the spinous process is usually absent. The primary failure is in the embryonic development of the neural tube which begins as a ridge and then folds into a tube. The posterior elements of the vertebra originate in cartilaginous masses on either side of the neural tube. The cartilage masses then unite posteriorly to the neural tube. In the maturation process, ossification centers begin laterally in the cartiiaginous vertebra and enlarge to replace the cartilage. Failure of osseous fusion of these two lateral osseous masses results in spina bifida. The process may involve only an osseous failure, or there may be other extremely complex tissue defects, including the neural tube, the nerves, the meninges about the neural tube, the subcutaneous tissues, and even the skin itself. If neural elements are involved, there may be paralysis of the parts distal to the defects with partial or complete paraplegia (myelodysplasia). Most commonly, simple spina bifida involves the first sacral vertebra, but the process may be so extensive as to involve the entire lumbar and sacral spine.

Sclerosis—An increase in the amount of bone, usually in a localized area. This bone is usually formed as a reaction to a disease process in the bone. The bone is more dense radiographically. The entire skeleton may be sclerotic as in osteopetrosis.

Still's Disease—Rheumatoid arthritis in an infant, child, or adolescent.

Sequestrum—A piece of dead (necrotic) bone that has become separated from the sound bone during the process of necrosis. The necrosis may be produced by fracture, or more commonly, by disease or infection. The sequestrum may be extruded to the outside through a sinus.

Spondylolisthesis—A slipping or displacement of one vertebra on another. Usually the superior vertebra is displaced forward on the inferior vertebra. Small degrees of displacement may be found with no defect in the vertebra. More pronounced displacement is usually secondary to a defect of bone formation in the pars interarticularis of the posterior elements of the vertebra superior. The defect in the pars interarticularis is termed spondylolysis.

Spondylolysis—A defect in bone formation in the pars interarticularis of the posterior elements of a vertebra. This defect was originally thought to be congenital, a failure of fusion of two ossification centers. Most recent studies indicate that the defect is probably a fracture of the pars interarticularis with failure of union of the fracture by osseous tissue.

Supination—Rotation of the forearm at the elbow joint so that the palm of the hand is turned upward.

Talipes equinovarus—A congenital deformity of the foot and ankle of unknown genetic control. Usually there are three components of the deformity, but in some instances one or another may be lacking. The three deformities are: plantar flexion of the foot at the ankle joint (equinus position), turning or rolling inward of the heel or hind foot (inversion), and medial deviation (adduction) of the forefoot on the hind foot. Also called a "clubfoot".

Trabecula—A spicule of cancellous bone; a part of spongy bone; a part of the spongiosa of bone. Usually found in the medullary canal of a bone. To be distinguished from cortical bone which is the heavy, dense bone formed of osteones around the medullary canal of a bone.

Transverse processes (of a vertebra)—One of the posterior elements of a vertebra. Each vertebra has two transverse processes which project laterally from the pedicles of the vertebra. In the sacrum, the transverse processes are much enlarged and fuse together to form the sacral portion of the sacro-iliac joint. Occasionally, the sacral transverse processes fail to fuse together to form the sacral portion of the sacro-iliac joint, and the sacral vertebrae are then termed "lumbarized" since they resemble a lumbar vertebra.

Unilateral sacralization—A partial or complete fusion of the fifth lumbar transverse process with the sacral transverse processes. The process may be present on only one side of the vertebra (unilateral), or it may be present on both sides of the vertebra (bilateral).

Varus and Valgus deformities—These are angular deformities of a bone or joint. In varus deformity, the part distal to a point of reference is deviated toward the mid-line of the body. In valgus deformity, the part distal to the point of reference is deviated away from the mid-line of the body. Genu (knee, the point of reference) vara deformity indicates that the leg is deviated medially toward the mid-line of the body. Hallux (great toe) valgus deformity indicates that the phalanges of the great toe are deviated away from the mid-line of the body producing the very common bunion.

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