6. HEALTH AND TRAUMA

6.1 Introduction

In order to understand the general health of a population, one needs to evaluate multiple factors that are crucial to the health status of individuals. These factors may be related to the environment, diseases, nutrition and social constraints or limitations. Usually only those factors that leave traces on the human skeleton, e.g. chronic diseases and nutrition, are available for evaluation of health of archaeological populations. Even these are not always sufficiently and accurately reflected on the human skeleton and therefore scientific assumptions and theories become inevitable.

The study of the health of prehistoric communities based on the presence or absence of diseases in skeletal populations has become increasingly important (e.g., Mensforth et al 1978; Ortner and Putschar 1981; Stuart-Macadam 1989; Ortner and Aufderheide 1991; Wood et al. 1992; Steyn 1995; Roberts and Manchester 1995; Kent and Dunn 1996). This approach has become known as the study of palaeopathology (Steinbock 1976). Palaeopathology is a broad field of study in which the evolution and progress of diseases are examined in conjunction with the evolution and progress of human populations. Description and classification of lesions on human tissue are the first steps in conducting a palaeopathological study (Ortner and Aufderheide 1991). The second step is to interpret that which has been described and classified and the ultimate step is to make a general statement on the health status of the individual or population being studied (Buikstra and Cook 1980; Ortner and Aufderheide 1991; Roberts and Manchester 1995).

It is through the study of palaeopathology that anthropologists and other researchers are familiarised with the manner in which past populations adapted, both culturally and biologically, to environmental and biological factors (Angel 1966; Steinbock 1976; Mensforth et al. 1978; Manchester and Roberts 1995). An indirect way of studying the health of archaeological populations is to study the environment within which communities lived. Studies of palaeoenvironments are a good source of information of the kinds of food resources, pathogen populations and physical constraints of a community in the past.

Numerous studies of both humans and animals have shown that it takes a long time for any disease to manifest itself in the skeleton. For the osteological manifestation of any disease to occur, such a disease has to be chronic in nature (Steinbock 1976; Ortner and Putschar 1981; Krogman and İşcan 1986; Roberts and Manchester 1995; Steyn and İşcan 2000) and the individual affected has to survive long enough for skeletal lesions to be established. The study of palaeopathology, based on human skeletal remains, therefore gives just a glimpse of the multitudes of diseases that would have affected prehistoric populations. There are countless acute and chronic soft tissue diseases that can not be accounted for in palaeopathology based on skeletal remains (Roberts and Manchester 1995). It must, therefore, be borne in mind that the absence of skeletal lesions does not, in any way, equate to the absence of diseases (Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). Wood and co-authors (1992) also bring to the forth the fact that sometimes the disease may be so severe that those affected die quickly before skeletal involvement is elicited. Therefore those who survive to show lesions may actually represent the stronger members of a community in that they did not succumb to death as a result of a disease.

Different diseases leave skeletal lesions that require different techniques for identification. Some lesions can be noted by simple macroscopic observation of dry bone, e.g., degenerative disease that cause the development of osteophytes on the vertebral column, while some lesions require the use of radiographic techniques to identify e.g. Harris lines on long bones. Chemical analysis of dry bone may also be used to identify possible changes in bone due to diseases or diet, e.g., stable isotope analysis can be used to determine the types of food that were predominant in a prehistoric community. DNA can now be extracted from archaeological remains and has proved to be very informative regarding a number of diseases (Ortner and Aufderheide 1991).

Roberts and Manchester (1995) differentiate two main sources of evidence of diseases that affected archaeological populations: human remains and art/documents. Human remains provide the primary or direct evidence of the presence of diseases and nutritional problems. Secondary evidence comes from ancient written documents and art. For instance some of the earliest evidence of Pott's disease comes from ancient drawings and text (Stuart-Macadam 1992; Roberts and Manchester 1995).

6.2 Problems and limitations

Skeletal remains have been used extensively in palaeopathological studies as the main source of information on the health status of prehistoric populations. Unfortunately, skeletons alone can not inform us of all aspects and dynamics of general health of the dead (Buikstra and Cook 1980; Wood et al. 1992). Such aspects as mental health, environmental hardships, level of exposure to life threatening conditions etc, can not be inferred from the skeleton. Not only are skeletons limited in the amount of information available, they have also been found to be a lot more difficult and complicated to interpret because a lot of factors need to be taken into account before making inferences (Wood et al. 1992).

One of the limitations emanates from the often incompleteness of archaeological remains. Depending on the aetiology and pathogenesis of any disease, skeletal lesions resulting thereof tend to 'favour' certain parts of the skeleton. The implication is, therefore, that sound results are dependent on how much of a skeleton has been found. Unfortunately, archaeological skeletons are not always complete. This is more problematic when dealing with diseases that affect the small bones of hands and feet (e.g., rheumatoid arthritis). Preservation and recovery of such small bones is limited by numerous factors and therefore such bones may not always be available for analysis (Brothwell 1981; Ubelaker 1989a; Roberts and Manchester 1995).

Another problem is that most skeletal lesions are ambiguous (Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995). Bone tissue has a limited number of ways in which it can respond to stress and hence it tends to produce ambiguous lesions (Buikstra and Cook 1980). Such lesions become difficult to attribute to a specific disease especially when the skeleton is incomplete. Despite similarities in the appearance of some lesions, many diseases tend to have a very specific characteristic that can only be identified on a specific bone or set of bones. Such a feature can be used in differential diagnosis to identify the most possible diseases responsible for lesions found in the skeleton.

Differential diagnosis itself requires knowledge and understanding of numerous diseases that affect the skeleton. It is only successful provided key features that differentiate diseases with similar skeletal lesions have been identified (Buikstra and Cook 1980). An example is that of psoriatic arthritis and rheumatoid arthritis, both of

which affect joints of the upper and lower extremities (i.e. hands and feet). The key difference between these two diseases is that while psoriatic arthritis is asymmetrical, rheumatoid arthritis affects the body symmetrically (Ariaza 1993). Ambiguous lesions can only be attributed to specific diseases on the basis of differential diagnosis, provided bones needed for the procedure are present.

Some skeletal lesions produced during developmental years, e.g. Harris' lines and porotic hyperostosis, tend to disappear or become obliterated in adulthood (Buikstra and Cook 1980; Ortner and Putschar 1981; Martin et al. 1985; Aufderheide and Rodriguez-Martin 1998). This means that the risk of exposure to stress during early years would remain unknown once an individual recovers and survives to adulthood (Wood et al. 1992). In this regard, skeletal pathology provides information on the number of individuals who died during stress exposure or during the healing process, not the total number of individuals who were exposed to stressors (Wood et al. 1992).

Diagnosis of diseases from skeletal remains relies on clinical criteria and this presents a multifaceted problem in palaeopathology (Ortner 1991; Wood et al. 1992). Firstly, observations made on clinical orthopaedic cases are superimposed on palaeopathological cases despite the fact that many palaeopathological cases have no direct correlation to clinical medicine. Just like humans, pathogens evolve through random and non-random processes (Buikstra and Cook 1980; Ortner and Aufderheide 1991). Using clinical cases to interpret palaeopathological cases implies that the evolutionary processes that pathogens have gone through are not taken into account. Knowing the evolutionary status of pathogens responsible for diseases in ancient times is important in that it can help explain any deviations in expected lesions of known diseases (Buikstra and Cook 1980).

Palaeopathology is based mainly on skeletons, which may demonstrate lesions that may not be fully known in clinical medicine (Ortner and Utermohle 1981; Ortner and Putschar 1981; Rodgers 1982; Ortner 1991; Ariaza 1993; Aufderheide and Rodriguez-Martin 1998). For instance, Ortner and Utermohle (1981) reported on a case of rheumatoid arthritis from a pre-Columbian skeleton in Alaska. Although the individual suffered from rheumatoid arthritis, the skeleton shows severe destruction of the sacroiliac joint and the lumbar vertebrae. These lesions are not clinically associated with rheumatoid arthritis and even on the basis of differential diagnosis there was no evidence

to suggest the occurrence of any other disease on this individual. They note the fact that radiological imaging may have failed to recognize the involvement of the sacroiliac joint in rheumatoid arthritis in some clinical cases. Since their discovery, several similar cases were reviewed from clinical patients and cadavers and revealed some cases of sacroiliac involvement in rheumatoid arthritis (Ortner and Utermohle 1981). The implication of such a case is that numerous palaeopathological rheumatoid arthritic lesions of the spine and the sacroiliac joint may have been misdiagnosed.

Congenital abnormalities are problematic because there is no clear differentiation between malformations resulting from simple deviation from normal development without clinical significance i.e. anatomical variants and those with clinical significance to such an extent that survivability and reproductive success may be compromised i.e. physiological variants (Turkel 1989). Quite often these are lumped together in palaeopathology and consequently, anatomical variants alter the true results of the health status of the skeletal sample under study. For instance, it has been found that posterior neural arches of the vertebral column may fuse much later than expected in some individuals and this can be misinterpreted as spina bifida occulta (Ferembach 1963; Turkel 1989; Aufderheide and Rodriguez-Martin 1998).

The traditional approach in interpreting skeletal lesions in palaeopathology has been to make estimations on the prevalence of diseases in populations. This approach has been influenced by the idea that the presence of skeletal lesions implies a healthy or an unhealthy population depending on the quantity of lesions found. However, it has been found that the occurrence of skeletal lesions has a more complex relationship with the health of a skeletal population than initially thought (Buikstra and Cook 1982; Wood et al. 1992). The absence of lesions can be attributed to low or no exposure or very high exposure to risks. High exposure can lead to death within a short time before bony response is evoked. The presence of lesions, on other hand, can be a result of moderate exposure to stress that allows time for osteological manifestation to occur. This implies that a population with less skeletal lesions than the other is not necessarily the healthier one. With no information on the levels of risks exposure, comparing the health statuses of two or more populations is complicated (Ortner and Aufderheide 1991; Wood et al. 1992). According to Wood et al. (1992), a comparison of distribution of lesions should not be equated to a comparison of health.

One of the problems within the theoretical field of palaeopathology involves understanding that which ought to be classified as disease and that which is a 'dysfunctional biomedical response' (Ortner and Aufderheide 1991). A disease would be any physical, psychological, emotional or other abnormality or dysfunction that makes the individual unhealthy and therefore increases the chances of death or reproductive failure (Buikstra and Cook 1980). A dysfunctional biomedical response, on the other hand, is similar to disease in terms of the side effect but has positive effects of increasing survivorship and reproductive possibilities. It is therefore crucial to differentiate between these when inferring the health of a past population on the basis of skeletal material because when dysfunctional biomedical responses are viewed as health hazards they give incorrect results (Wood et al. 1992). For instance, sickle cell anaemia (heterozygote) in a malaria infested area could be viewed as a dysfunctional biomedical response or adaptation to the environment (Angel 1966; Ortner and Putschar 1991; Wood et al. 1992). The totality of causes of death in a given skeletal population is never known. At any given point in time, individuals or populations are exposed to different factors that can result directly or indirectly in their death (Wood et al. 1992). This means that the total number of deaths is made up of fractions of different causes of death which, unfortunately, can not be estimated from skeletal remains only.

One of the factors that needs to be taken into account is that skeletal remains can not give any clues of individuals' resistance to diseases and stress. Individual differences in response or susceptibility to diseases and stress, also known as frailty, can be influenced by a variety of factors (Wood et al. 1992). Influential factors include, among others, genetic makeup, access to food and other resources, economic and social status.

The prevalence of malformed individuals in skeletal samples may be influenced to some extent by the cultural beliefs of the population being represented. Differential burial practices associated with 'out of the normal births' and stigmatised diseases interfere with the recovery and consequently evaluation of congenital and other stigmatised diseases in prehistoric time (Turkel 1989; Buikstra and Cook 1980). Preservation of individual bones is affected by soil conditions, depth of graves, burrowing animals, burial practices etc (Brothwell 1981; Ubelaker 1989a).

A prior knowledge of bone changes resulting from taphonomic processes is essential when studying palaeopathology (Buikstra and Cook 1980; Ortner and Putschar

1981; Brothwell 1981; Roberts and Manchester 1995). Some post-depositional processes can alter the appearance of bones in such a way that differentiating between them and pathology becomes obscured. The condition where post depositional damage resembles pathology is referred to as pseudopathology. Small bones of fetuses and newborn babies are at high risk since they have very limited resistance to taphonomic processes. Erosion of outer or inner tables of cranial bones would present limitations when examining lesions such as porotic hyperostosis on infants.

6.3 Specific diseases found in the Toutswe population

Some skeletal lesions can be associated with specific diseases. Such diseases can, for example, be congenital, degenerative, infectious or metabolic (Steinbock 1976; Ortner and Putschar 1981; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). The following sections examine the presence and prevalence of specific diseases as reflected by skeletal lesions on various individuals.

6.3.1 Spina bifida occulta

Introduction

Spina bifida is a developmental neurulation defect. Failure of the neural arches to fuse means that a vertebral arch develops abnormally, resulting in a cleft (Barnes 1994). Depending on the size of the cleft created, the neural tube may be displaced outside the vertebral column if the cleft is large, whereas if the cleft is small, a lipoma (an extra layer of fat) may develop in place of the missing bone to protect the neural tube. Sometimes the cleft is small and the neural tube remains unthreatened (Barnes 1994; Roberts and Manchester 1995). In severe cases, cranioarachischis may develop resulting in death during embryonic or early life (Barnes 1994; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998).

Diagnosis of severe spina bifida, associated with anencephaly and meningomyelocele, is very difficult in palaeopathology given the fact that individuals with this condition died prematurely (Barnes 1994; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). However, it may be easy to identify this defect in mummified rather than skeletal remains. The less severe condition usually referred to as spina bifida occulta (Ortner and Putschar 1981; Barnes 1994; Roberts and Manchester

1995), can be found in archaeological skeletons of older children and adults since the condition is not life threatening. Spina bifida occulta varies depending on the size of the cleft created. It can occur with clinical symptoms such as severe pain, impaired motor function and impaired sphincter control when the neural tube is affected (Barnes 1994; Roberts and Manchester 1995). Spina bifida occulta without neural tube defects is more common than that with neural tube defects (Barnes 1994; Aufderheide and Rodriguez-Martin 1998).

Spina bifida occulta affects individuals of both sexes equally. It is one of the most commonly reported spinal defects in archaeological human remains (Ferembach 1976; Ortner and Putschar 1981; Roberts and Manchester 1995). Within the first year of life, neural arches remain unfused (Black and Scheuere 2000) and so it is difficult to differentiate between normal unfused neural arches and pathologically unfused neural arches when dealing with skeletal material only. Although commonly found on the sacrum, spina bifida occulta also affects other types of the vertebrae.

In the early 1960s, there were concerns regarding the description of that which constitutes sacral defects. Ferembach's concern was based on the identification of incompletely developed neural arches that were not necessarily due to spina bifida occulta (Ferembach 1963). Aufderheide and Rodriguez-Martin (1998) point out that sometimes neural arches may not fuse until later than the expected age of fusion and this makes it difficult to distinguish between pathologically induced lesions and simple deviation from the normal anatomical situation. The implication here is that the prevalence of spina bifida occulta in a skeletal population tends to decrease with age, indicating that delayed fusion of the vertebrae in young individuals is being mistaken for spina bifida occulta.

Methods

Evidence for spina bifida occulta was examined, through simple visual observation, in all individuals whose vertebral arches had fused. No attempts were made to examine infants and young children since it is difficult to differentiate between normal and pathologically unfused neural arches of young infants. Unfortunately not all older children and adults could be included because of poor preservation leading to loss of vertebrae.

Results

From the Toutswe sample, three individuals have evidence for partial spina bifida occulta involving the cervical, lumbar and sacrum. On Bosutswe Burial 5, a male of 17 - 20 years old, the fifth lumbar was involved while on Bosutswe Burial 11, a 12 - 15 year old child of unknown sex, the lesion is on the first sacral vertebra. Lesion on the axis was identified on Toutswemogala Burial 6, a child aged between nine and 11 years old. In all three cases, the other vertebrae were normal and therefore delayed fusion was excluded as a cause. However, the defects appear to have been asymptomatic because the lack of contact between the neural arches left very small fissures. Therefore the lives and reproductive capabilities of those affected were not in endangered in anyway. From this result, it is apparent that spina bifida occulta was not a common problem for this population. The incidence of spina bifida occulta in the Toutswe is low and the lesions found are small fissures. Numerous individuals from Toutswe sites have fragmented or missing parts, and some even have no vertebrae present. Therefore, the anomaly could not be assessed on the whole population. Statistical analysis of the prevalence of this condition on the study sample in thus limited.

The prevalence of the defect is often reported for other archaeological populations e.g. Morris (1984) Steyn and Henneberg (1995a). On the K2/Mapungubwe sample, three adults aged between 17 and 30 years had spina bifida occulta (Steyn and Henneberg 1995a) and in all cases the anomalies were not serious. Morris (1984) identified six individuals with this defect and one of them was a 3- year old child with a more severe spina bifida. The lesion was associated with an under developed spinous process.

In both Toutswe and K2/Mapungubwe skeletons, the incidence of this defect is below five percent of the total skeletal sample. However, the incidence may be slightly higher if expressed on the basis of only those individuals with complete or nearly complete vertebrae. No cases have been reported on the Oakhurst sample (Patrick 1989).

6.3.2 Arthritic and degenerative diseases

Introduction

Arthritic disease is a general term used to refer to diseases that affect joints. They may be infectious, metabolic, genetic or degenerative (Steinbock 1976; Aufderheide and

Rodriguez-Martin 1998). Degenerative and arthritic diseases are non-inflammatory, chronic and progressive. Although the etiology differs, the most common predisposing factor has been found to be the development of lesions on cartilage that separates bones in joint and thereby exposing bone surfaces. These conditions are characterized by new bone formation, usually visible by the age of 40 years (Aufderheide and Rodriguez-Martin 1998).

Methods

Macroscopic visual observations for gross morphological alterations associated with arthritic diseases were made. Each lesion found was recorded in terms of its size and location on the bone. The assessment included vertebral segments and peripheral joints.

Results

Lesions associated with degenerative and arthritic diseases were osteophytes on the vertebrae and around articular facets of major weight bearing joints. Those affected were aged between 30 and 75 years old. One male (Bosutswe Burial 12) will be discussed in more detail since differential diagnosis produced significant results. In two cases, Taukome Burials 1 and 5, both males and both aged between 40 and 60 years old, osteophytes were identified on the vertebrae only, as long bones were either incomplete or missing. Some of the lumbar vertebral segments had fused to each other on Taukome Burial 5 (Figure 6.1). Unfortunately the hip and knee joints of this individual were not preserved. The sacrum of Taukome Burial 2 shows signs of involvement. Taukome Burial 2 had osteophytosis on the cervical region as well as bone growth on the lesser tubercle of the left humerus where the subscapularis inserts (Figure 6.2). On Kgaswe B-55 Burial 17, a 50 - 75 year old male, most of the cervical and thoracic vertebrae had been involved but unfortunately none of the long bones was present. The axis had fused to the third cervical vertebra and the lateral articular facets between the atlas and the axis are partially eroded. The lumbar vertebrae are missing but lesions on S1 suggest possible fusion to L5. On two individuals the vertebrae were present but not involved while the hip and knee joints had lesions. One individual is about 30 - 50 years old (Toutswemogala Burial 25), and one is about 40 - 60 years old (Thatswane Burial 5) and they were both females. Toutswemogala Burial 25 had lesions around the distal articular

surface of the right femur but not on the proximal articular surface of the right tibia. The left femur and tibia are both missing. Thatswane Burial 5 has lesions on the vertebrae, distal femur, patellae and some bones of the feet i.e. Toutswemogala Burial 25 and Thatswane Burial 5.

Kgaswe B-55 Burials 5(2) and 7(2), both males of 30 - 50 years old, have osteophytes surrounding the distal articular surfaces of their femora. Unfortunately, both individuals are represented by incomplete long bones only. Thus peripheral joints of the three adults (Taukome Burials 1 and 5) with vertebral osteophytosis were not preserved and the one individual (Taukome Burial 2) with other joints showed involvement of the shoulder joint. Two individuals (Kgaswe B-55 Burials 5(2) and 7(2)) both show involvement of the knee joints but their vertebrae were not preserved.

Table 6.1 shows the distribution of arthritic lesions on the sample. Bosutswe Burial 12 has been excluded from the table since it presents a more different condition. The elbow has also been excluded in the table since none of the individuals indicated involvement of this joint. Of all individuals included in the table, only Toutswemogala Burial 25 and Thatswane Burial 5 have all joints assessed. Lesions on different parts of the vertebrae are pooled because of small sample size. The most commonly affected site is the vertebra (eight cases) followed by the knee (five cases). The feet are the least affected. Thatswane Burial 5 shows the most affected joints which include the hip and feet. Osteoarthritis is the most probable cause of lesions found on the skeletons.

Vertebral osteophytosis associated with osteoarthritis are common in archaeological and historical skeletons. They have been reported at K2/Mapungubwe (Steyn 1994; Steyn and Henneberg 1995a), Oakhurst (Patrick 1989), Riet River and Kakamas (Morris 1984) and historical skeletons from South Africa (Peckmann 2002). The skeletons from 18th and 19th century Griqua, Colesberg and Wolmaransstad show relatively higher incidences of osteoarthritis by comparison to earlier ones from Toutswe, K2/Mapungubwe, Riet River and Kakamas.

Lesions found are located on the vertebrae and bones of the feet and hands. The distribution of lesions on the vertebrae is associated with the three primary curves of the vertebral column. Individuals with osteophytosis on the K2/Mapungubwe skeletons are mostly middle aged and old adults (Steyn 1994) and thereby similar to what was found at Toutswe. It is possible that degeneration of vertebrae at Oakhurst was linked to daily

activities because the individuals are mostly young whereas at K2/Mapungubwe and Toutswe individuals affected are older and therefore expected to show such lesions.

Other degenerative conditions found include a deformed mandible of Taukome Burial 2 as a result of dental wear and antemortem tooth loss and calcification of sternal ends of first ribs on Taukome Burial 5, which are normally cartilage. Kgaswe B-55 Burial 14, a 40-60 year old female has bone exostosis on the patella and calcaneus.

Table 6.1 Distribution of arthritic lesions on adults

Burial	Age	Sex	Vertebrae	Shoulder	Hip	Knee	Feet
Toutswemogala 19	40-60	M	Present	-	-	-	-
Toutswemogala 22	40-50	M	Present	-	-	Preset	-
Toutswemogala 25	30-50	F	-	-	Present	Present	-
Taukome 1	40-60	M	Present	-	-	-	-
Taukome 2	40-60	F	Present	Present	-	-	-
Taukome 5	40-60	M	Present	Present	-	-	-
Thatswane 5	40-60	F	Present	-	Present	Present	Present
Kgaswe B-55 5(2)	30-50	M	-	-	-	Present	-
Kgaswe B-55 7(2)	30-50	M	-	-	-	Present	-
Kgaswe B-55 17	50-75	M	Present	-	-	-	-
Kgaswe B-55 19	40-60	M	Present	-	-	-	-

Figure 6.1 Fused lumbar vertebrae on Taukome Burial 5

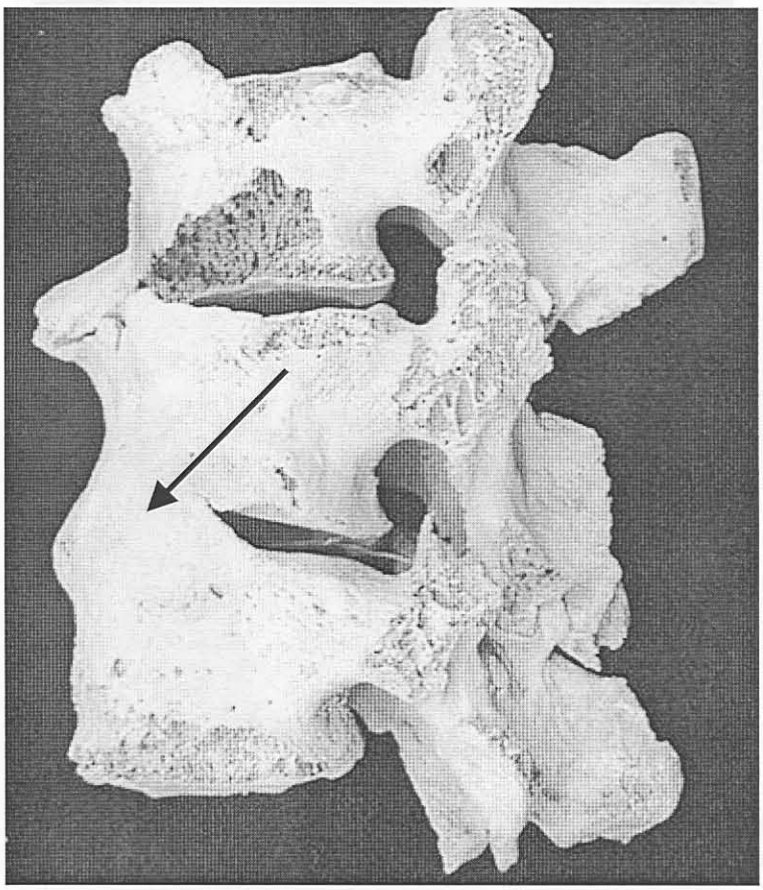
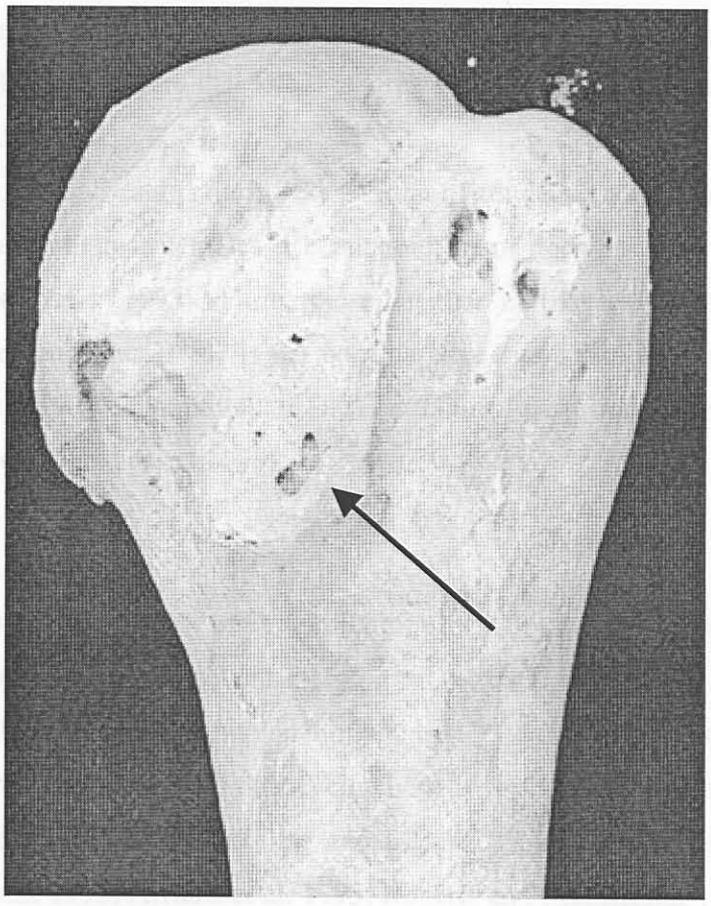


Figure 6.2 Bone growth on the left distal humerus, Taukome Burial 2



6.3.3 Possible case of Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Introduction

Reports on Diffuse Idiopathic Skeletal Hyperostosis (DISH) also known as Forestier's disease, date as far back as the 1950s (Rodgers 1982; Crubezy and Trinkaus 1992; Arriaza 1993; Arriaza et al. 1993; Roberts and Manchester 1995; Maat et al. 1995; Aufderheide and Rodriguez-Martin 1998; Reale et al. 1999; Jankauskas 2003). This is a degenerative condition occurring in both archaeological and modern populations. The etiology of this condition is not fully known (Rodgers 1982; Crubezy and Trinkaus 1992; Arriaza 1993; Arriaza et al. 1993; Roberts and Manchester 1995; Maat et al. 1995; Aufderheide and Rodriguez-Martin 1998; Reale et al. 1999; Jankauskas 2003). It is,

however, commonly associated with diabetes and obesity. In clinical cases it has been demonstrated that obese patience with type II diabetes have impaired insulin function. The insulin impairment increases levels of serum growth hormones that in turn evoke bone growth. It has been suggested that DISH is a result of metabolic disorders caused by high calorie intake (Jankauskas 2003).

The condition usually starts during the fourth or fifth decade of life and is slightly more common in males than in females. Rheumatoid factor does not play a role in the etiology of DISH (Arriaza 1993). Furthermore, neither cartilage (intervertebral) nor synovium is involved and thus DISH is not considered a true arthropathy (Aufderheide and Rodriguez-Martin 1998). Symptoms of DISH in clinical cases include back stiffness and reduced movement (Jankauskas 2003).

Skeletal manifestations of DISH

DISH causes fusion of the vertebral column, especially on the thoracic vertebrae through the development and subsequent merging of vertebral osteophytosis. However, spaces for intervertebral disks are maintained. In addition to vertebral lesions, extra spinal manifestations of this condition include development of osteophytes on major weight bearing joints and exostosis on the patellae and calcanei (Rodgers 1982; Crubezy and Trinkaus 1992; Arriaza 1993; Arriaza et al. 1993; Roberts and Manchester 1995; Maat et al. 1995; Aufderheide and Rodriguez-Martin 1998). One of the key diagnostic features of this condition is the development of osteophytes on the sternum where the first ribs articulate (Rodgers 1982; Arriaza 1993; Arriaza et al. 1993; Reale et al. 1999; Jankauskas 2003). The sacroiliac joint may be fixed by bony bridges, but not by intra-articular bony ankylosis, which is the case in ankylosing spondylitis (Aufderheide and Rodriguez-Martin 1998; Jankauskas 2003).

Results

A possible case of DISH has been found on an adult male aged between 50 and 75 years (Bosutswe Burial 12). This individual has osteophytes on the cervical (Figure 6.3), thoracic and lumbar vertebrae (Figure 6.4), and had caused partial fusion of the vertebral column at the time of death. Articular surfaces of vertebral bodies show no signs of involvement, therefore strongly suggesting lack of intervertebral disk involvement.

However, the axis and C3 had fused to each other (Figure 6.5). On the sternum, articular surfaces of the first ribs had been involved (Figure 6.6). Lesions on the mandibular condyles suggest that the temporomandibular joints were fusing (Figure 6.7). Other extra spinal lesions found include exostosis on the posterior aspect of the olecranon of the ulnae, where the triceps brachii muscles insert, the anterior surfaces of the patellae (Figure 6.8) at the insertion of the quadriceps femoris muscles, and posterior surfaces of the calcanei at the insertion of the Achilles tendon (Figure 6.9). On the same individual, the sacroiliac joint has some lesions. Bones of the hands and feet of Bosutswe Burial 12 are complete and have no lesions.

Figure 6.3 Vertebral osteophytosis on the cervical region of Bosutswe Burial 12

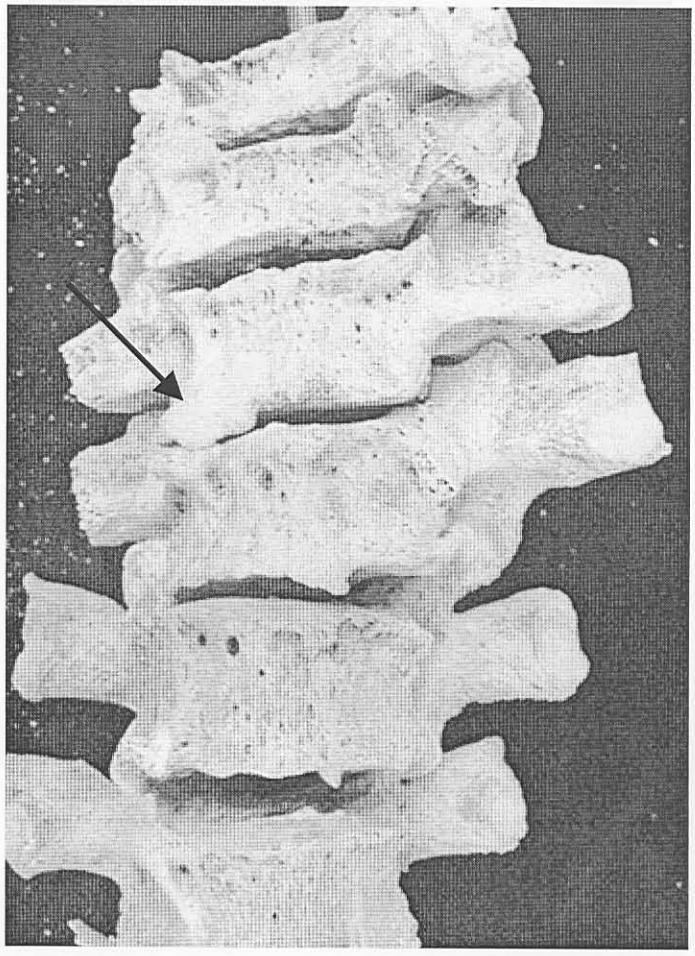


Figure 6.4 Vertebral osteophytosis on the thoracic and lumbar regions Bosutswe Burial 12.

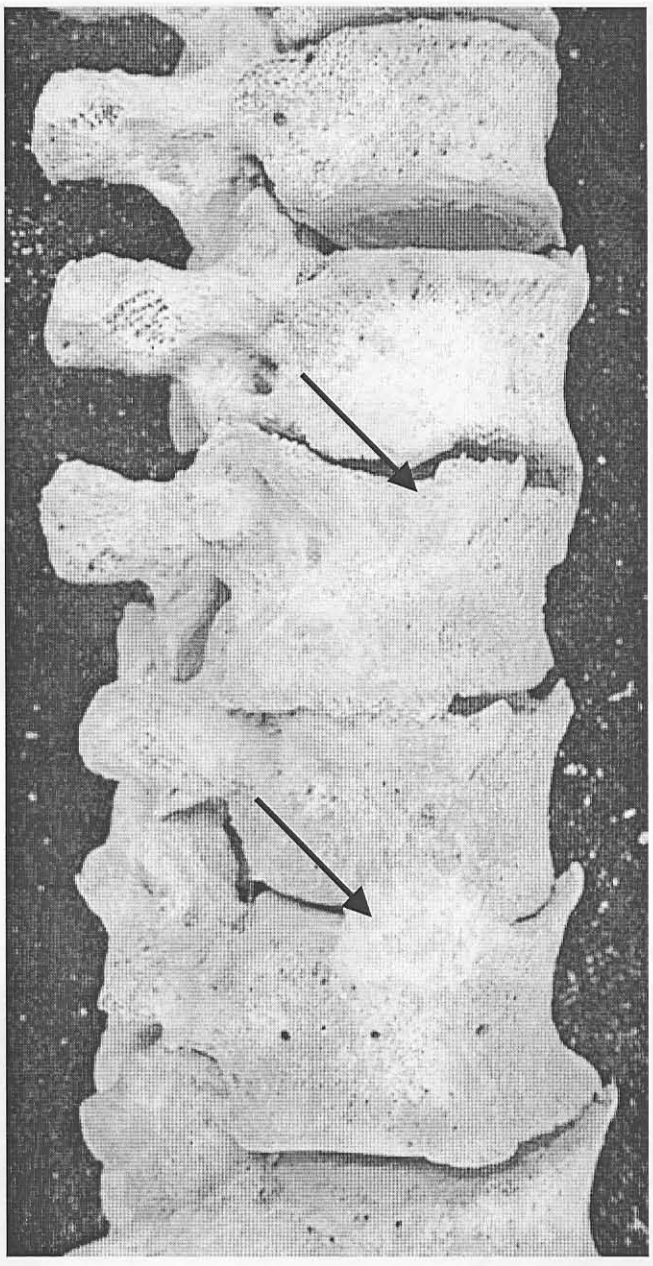


Figure 6.5 Fused axis and C3, Bosutswe Burial 12

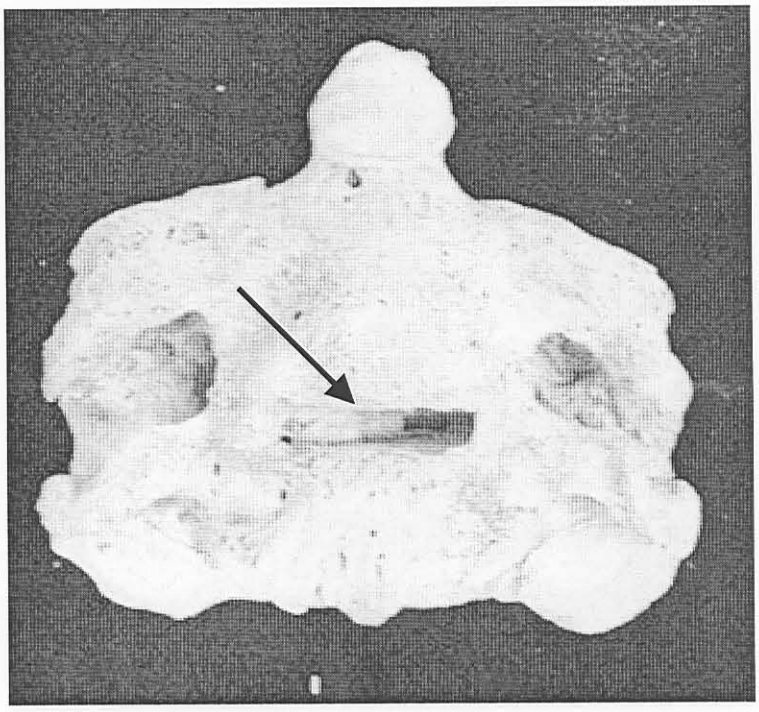


Figure 6.6 Osteophytes on the manubrium where first ribs articulate, Bosutswe Burial 12

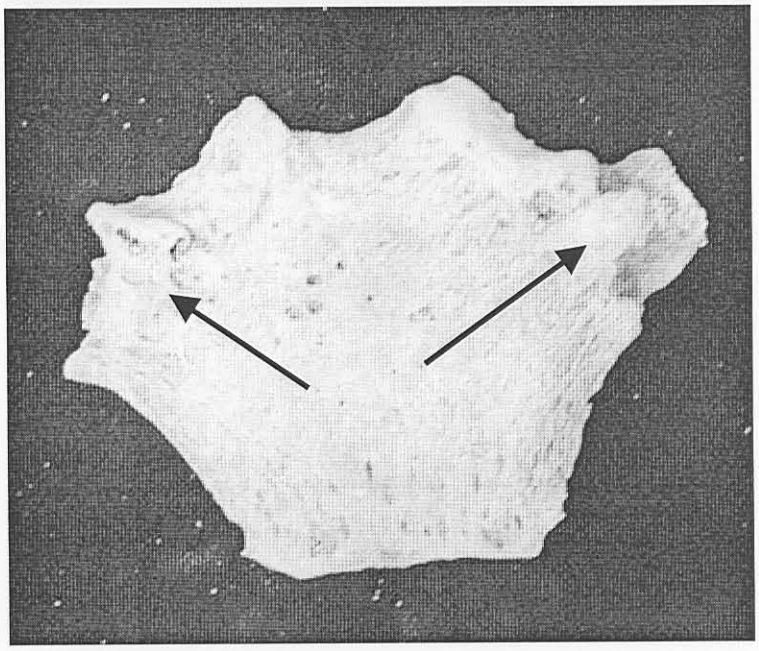


Figure 6.7 Lesion on the right mandibular condyle, Bosutswe Burial 12

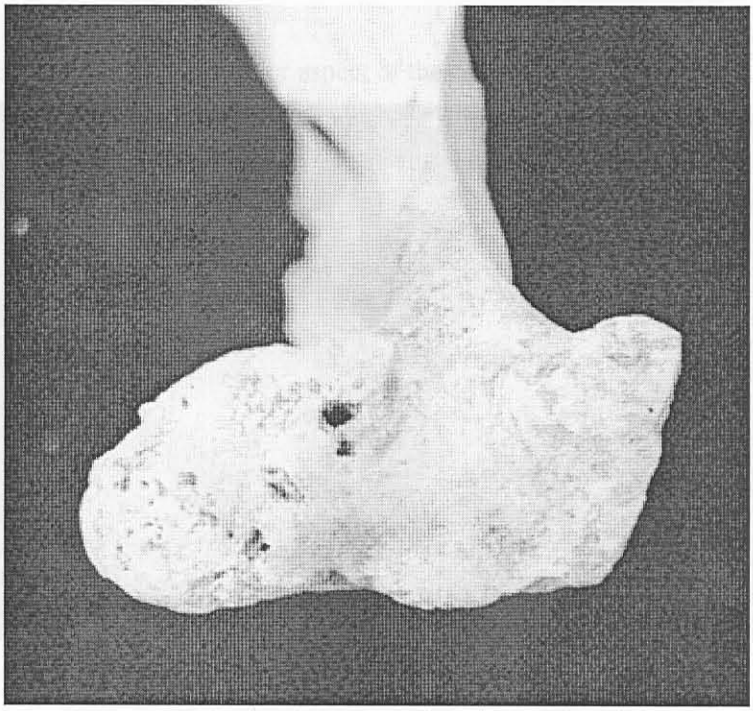


Figure 6.8 Exostosis on the patella, Bosutswe Burial 12

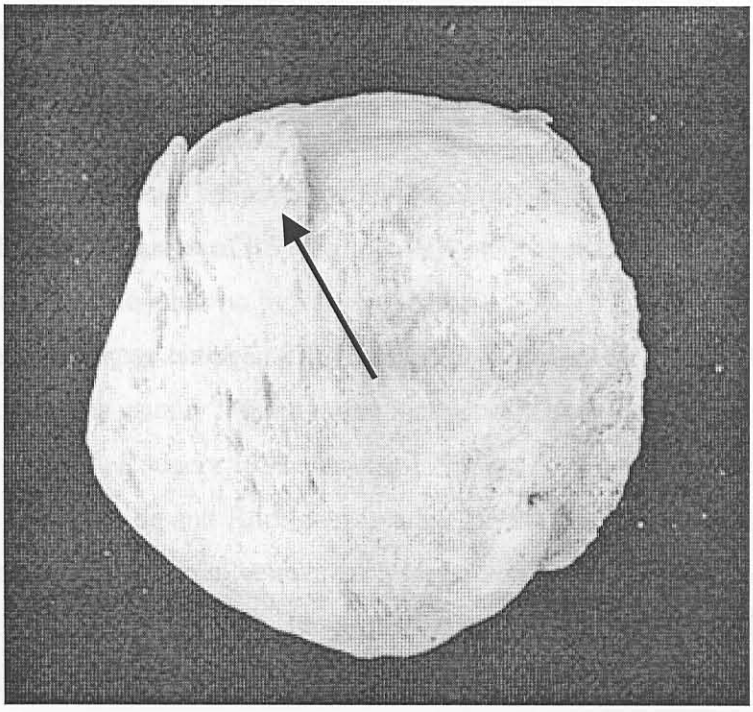
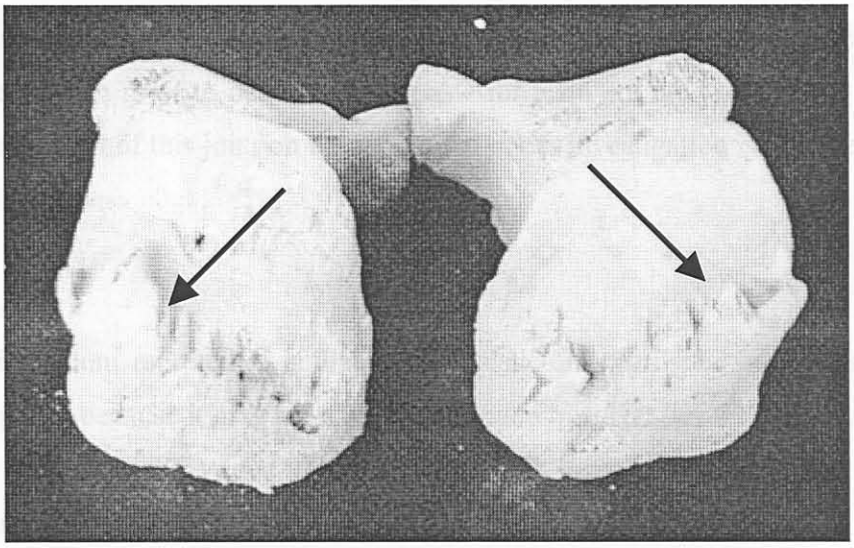


Figure 6.9 Exostosis on the posterior aspect of the calcanei, Bosutswe Burial 12



Differential diagnosis

Other diseases that may have caused the lesions found on this individual include ankylosing spondylytis (AS), rheumatoid arthritis (RA), psoriatic arthritis (PA), Reiter's syndrome (RS) and osteoarthritis (Rodgers 1982; Arriaza 1993; Arriaza et al 1993; Rodgers and Manchester 1995; Jankauskas 2003). RA and PA can probably be excluded, since in both conditions small bones of the hands and feet are the main affected areas whereas in DISH the small joints are not affected. Phalanges of the big toes are one of the main sites of involvement in cases of RS (Arriaza 1993) and as previously mentioned, the individual being discussed here has no lesions on his feet.

One of the differences between DISH and AS is that while DISH affects other parts of the skeleton, AS is usually limited to the lumbar vertebrae and the sacroiliac joint (Ortner and Pustchar 1981; Rodgers 1982; Arriaza 1993; Arriaza et al. 1993; Rodgers and Manchester 1995; Aufderheide and Rodriguez-Martin 1998; Jankauskas 2003). The case under study shows no signs of involvement of intervertebral disks or spaces and this is typical of DISH. AS would have most probably shown signs of involvement of intervertebral disks by lesions on the articular surfaces of the affected vertebral bodies. Post depositional damage makes it difficult to determine whether the sacroiliac lesions on

this individual are characteristic of DISH or AS defined by Aufderheide and Rodriguez-Martin (1998) and Jankauskas 2003. Degeneration of the temporomandibular joint is usually associated with severe dental wear, especially on posterior teeth, and excessive antemortem loss of teeth (Richards and Brown 1981; Richards 1990). Bosutswe Burial 12 has lost only one tooth prior to death and the posterior teeth are not worn down to root level. The involvement of this joint on the individual under investigation is assumed to be one of the DISH lesions.

Discussion

DISH is seldom reported in archaeological skeletons, partly because it is a rare condition and partly because it may have previously been mistaken for other diseases like AS (Rodgers 1982; Crubezy and Trinkaus 1992; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998; Jankauskas 2003). One of the earliest known cases is of a Neanderthal male aged between 35 and 40 years old from Iraq (Crubezy and Trinkaus 1992). Rogers (1982) diagnosed this condition on a 79-year old female from Europe. In Africa cases of this nature have been reported from a site associated with Meroitic Nubians at Semma South in Sudan (Arriaza 1993). From a total of 134 adults, DISH was identified in 13.4% of the sample and most of the individuals affected were males. To the best of the author's knowledge, no cases of DISH have been reported from southern African Iron Age sites yet. A recent study by Jankauskas (2003) on the skeletons from Lithuania indicates a correlation between socio-economic statuses and the prevalence of DISH. The condition was found to be most prevalent on individuals from higher social classes who had more access to foods rich in calories, which are some of the predisposing factors to DISH.

Cases reported by Rodgers (1982), Crubezy and Trinkaus (1992) and Reale and co-authors (1999) demonstrate lesions similar in morphology and distributions to those found on Bosutswe Burial 12. Vertebral osteophytes, bone spurs on the olecranon, patellae, calcanei and other lesions have been found in all cases including the individual under study. However, there are differences in the magnitude of bone involvement with previously reported cases as many of them had complete fusion of the vertebral column while Bosutswe Burial 12 did not. Bearing in mind all factors mentioned, it is proposed that individual suffered from Diffuse Idiopathic Skeletal Hyperostosis.

6.4 Trauma

Introduction

Traumatic lesions are commonly encountered on skeletal remains in archaeological samples (Ortner and Putschar 1981; Brothwell 1981; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). There are several kinds of trauma including fractures, dislocations, deformations, burning, mutilations and trephination (Ortner and Putschar 1981; Brothwell 1981; Roberts and Manchester 1995). Very often mutilations and trephination are restricted to specific cultural groups, whereas the other kinds of trauma occur across cultures. Trauma on bone can be sudden and intense or it can result from long mild stress applied continuously to bone.

Lesions associated with trauma can provide information on the occupation or lifestyles of those affected (i.e. occupational trauma) and can also indicate the social and political status of a study population. For instance, a community characterised by violence would demonstrate a high incidence of trauma, especially fractures. Size and location of trauma can help hypothesise on the cause of death particularly when there is no evidence of healing (Roberts and Manchester 1995). Fractures result from any event that leads to the partial or complete breaking of bone (Roberts and Manchester 1995). Fractures occurring shortly before death, whether they resulted in death or not, are difficult to differentiate from post depositional damage. As a result, the most commonly reported fractures in palaeopathology are those that were in the process of or completely healed at the time of death (Roberts and Manchester 1995).

Methods

Macroscopic visual observation of bone was used to identify any morphological changes associated with trauma. Where such changes were observed, they were described in terms of the bone(s) affected, location on the bone and the gross appearance of the affected area. The bones were subsequently photographed. No x-rays were used in this study.

Results

One individual, Bosutswe Burial 3, a 30 - 40 years old male, shows evidence of a fracture on a left metatarsal (Figure 6.10) and another individual, Kgaswe B-55 Burial 16,

a male aged between 20 and 30 years, had a fracture on the left fibula. In both cases the fractures had healed long before death. The metatarsal had been fractured on the middle one-third of the diaphysis whereas on the fibula the fracture was close to the distal end. Another healed traumatic lesion was found on the diaphysis of the left femur of Toutswemogala Burial 14. The individual was a child of five to seven years old.

Two juveniles, both from Toutswemogala (Burials 4 and 6), had possible traumatic lesions on their skulls. Burial 4 is a child of between six and eight years old and Burial 6 is between nine and eleven years. Each of them has a hole with sharp exterior margins and internal beveling. In both cases the holes are situated on the left parietal bones. Figure 6.11 shows the small, round lesion on the cranium of Toutswemogala Burial 4. On Toutswemogala Burial 6 the hole is oval in shape. Unfortunately there is no evidence of healing in both of them meaning that if indeed the holes were traumatic, the trauma would have occurred around the times of their deaths. In the absence of substantial evidence for trauma, the possibility of these lesions having resulted from post depositional processes cannot be ruled out. Thus skeletal changes mentioned above could be simply pseudotrauma.

The bones of an adult male aged between 40 and 60 years (Toutswemogala Burial 19) have evidence of burning. Shafts of the femora and tibiae are charred. The head of the right femur, the fragments of the pelvis as well as the distal ends of the ulnae are whitened. Several of this individual's phalanges are charred. The cranium and vertebrae, although incomplete, appear not to have been affected by the burning. The skeleton itself was mixed with animal bones, which have also been burnt. Prolonged exposure of bones to fire after burial is not ruled out as a possible cause of this burning. Unfortunately the provenance and context of the grave are not known and no sound conclusions can be made at this point.

Figure 6.10 Healed fracture on a left foot metatarsal of Bosutswe Burial 3

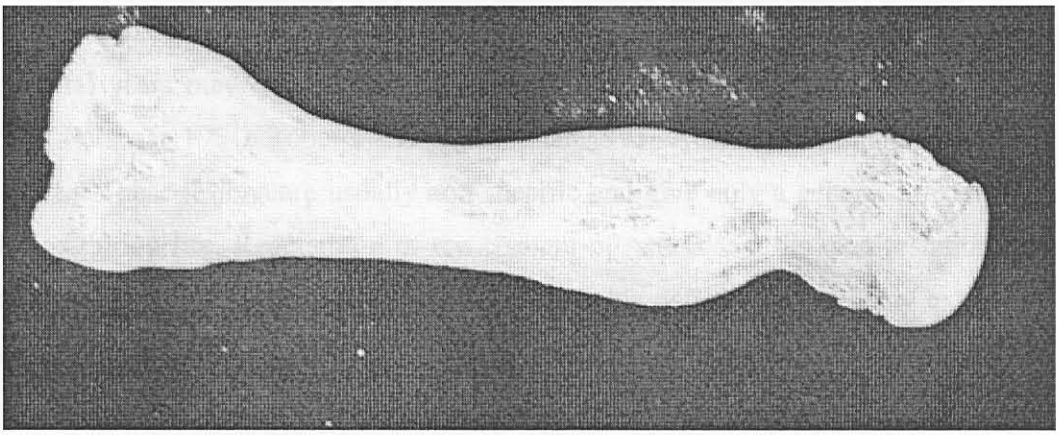
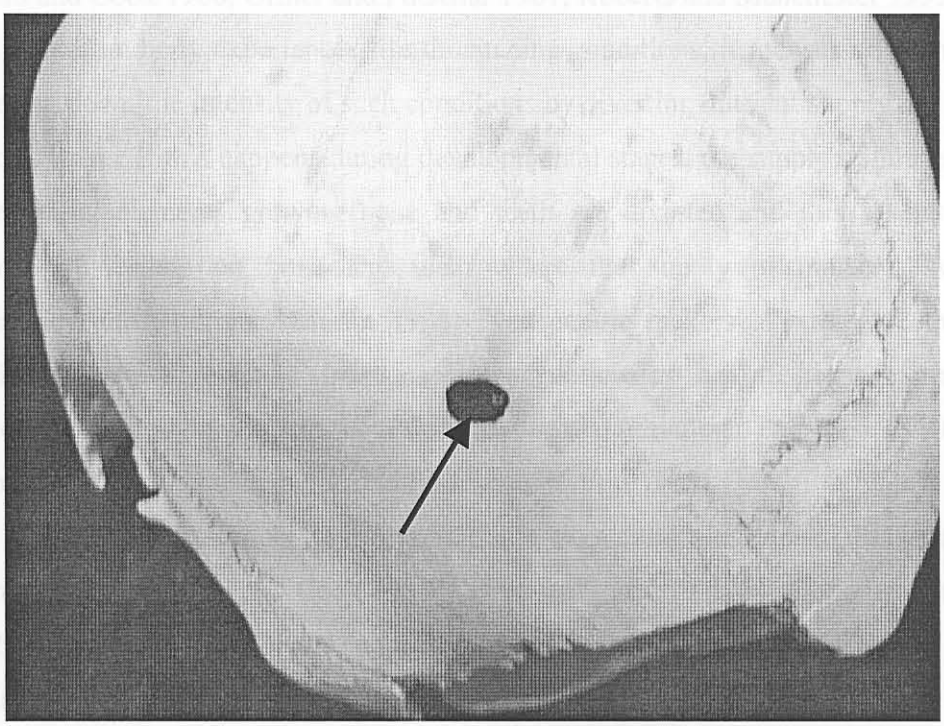


Figure 6.11 A round hole on the skull of Toutswemogala Burial 4



6.5 Nonspecific lesions

Stress due to nutritional deficiency and pathogen invasion experienced during developmental years may leave marks on bone (Selye 1973; Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). These lesions are usually non-specific and give only a general impression of hardships during life. Recognition of the concept of 'stress' and its manifestation on bones dates as far back as the 1950s (Selye 1973). Selye defines stress as any extrinsic condition or set of conditions that impact on an organism, at a magnitude that provokes that organism's biological and physiological reaction. Such extrinsic factors include, among others, severe nutritional deficiency and microbial invasion (Steinbock 1976; Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995). Stress can be acute or chronic and depending on intensity can have short-term asymptomatic lesions or can result in retarded growth with life term consequences. Nonspecific skeletal lesions do not have a one-to-one relationship with stressors (Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995).

When the body experiences life threatening conditions, it focuses on eliminating or lowering down the intensity of such conditions by diverting nutrient supply and energy to affected areas. If this happens during developmental stages, the supply of nutrients and other essential elements growing bone and teeth are diverted and this results in the formation of lesions on bone and teeth where the supply of nutrients was cut (Aufderheide and Rodriguez-Martin 1998). Nonspecific makers of stress are therefore indicative of stress resulting from nutritional deficiency or infections experienced elsewhere in the body not where the lesions are located (Goodman and Rose 1980; 1990; Goodman et al. 1980; Aufderheide and Rodriguez-Martin 1998).

Nonspecific indicators of stress fall into two categories: dental indicators and bone indicators (Buikstra and Cook 1980; Ortner and Putschar 1981; Goodman and Rose 1980; Martin et al. 1985). Dental indicators of stress are macroscopically visible enamel hypoplasias. Enamel hypoplasias are pits or grooves resulting from arrested enamel growth whereas discoloration of enamel may result from water chemicals and poor nutrition (Buikstra and Cook 1980). Nonspecific indicators of stress on bone can also be macroscopically visible (e.g. cribra orbitalia, porotic hyperostosis and periostis) or radiologically visible e.g. Harris lines. From the demographic point of view, high infant

mortality rate is seen as a nonspecific indicator of stress (Buikstra and Cook 1980; Wood et al. 1992). Frequencies of any of these nonspecific stress indicators within a population are often calculated to assess the extent to which a population survived or succumbed to environmental insults (Buikstra and Cook 1980; Ortner and Putschar 1981; Roberts and Manchester 1995; Aufderheide and Rodriguez-Martin 1998). Harris lines were not included in this study because of the difficulties with obtaining x-rays for these bones.

6.5.1 Cribra orbitalia and porotic hyperostosis

Cribra orbitalia and porotic hyperostosis are generally accepted to result from anaemia. However, this anaemia may be due to iron deficiency, but is more likely to be a reflection of the occurrence and severity of infectious diseases. By definition, anaemia refers to a condition in which there is a reduction in the concentration of haemoglobin and/or red blood cells below the quantities required for normal body function (Mensforth et al. 1978; Stuart-Macadam1989; 1991; 1992; Ascenzi et al. 1991). The body stores its own iron supply in the liver and spleen, but continuously absorbs iron from food to maintain certain levels of iron stores. This iron is needed in the formation of haemoglobin in new red blood cells developing in the bone marrow (Stuart-Macadam 1989; 1992; Ascenzi et al. 1991; Kent et al. 1994; Roberts and Manchester 1995). When there is shortage of iron for new haemoglobin, the red blood cells become pale and often die within half the time of their normal life span. There are different kinds of anaemia and therefore the etiology differs from one type of anaemia to the other (Stuart-Macadam 1989; 1992; Ascenzi et al. 1991; Kent et al. 1994; Roberts and Manchester 1995).

Anaemia can be acquired (e.g. resulting from infections, parasitic and bacterial invasion, trauma with excessive blood loss) or it can be congenital, as in sickle cell anaemia and thalassemia (Mensforth et al. 1978; Stuart-Macadam 1989; 1992; Ascenzi et al. 1991; Kent et al. 1994). Genetic anaemia, particularly sickle cell anaemia (homozygotes), is not compatible with life and therefore lack of appropriate medical intervention in prehistoric times would have resulted in premature death of those affected (Stuart-Macadam 1989; Kent et al. 1994; Roberts and Manchester 1995). Individuals with sickle cell trait (heterozygotes) may live long and successfully. An assumption is that lesions found in older children and adults in archaeological skeletons are a result of acquired iron deficiency anaemia (Stuart-Macadam 1992; Roberts and Manchester 1995).

The aetiology of iron deficiency anaemia depends on many factors. Predisposing factors include a cereal-based diet, severe blood loss, accelerated demands during pregnancy and growth (Mensforth et al. 1978; Stuart-Macadam1989; 1992; Roberts and Manchester 1995) and pathogen invasion. Red meat, legumes and fish have high quantities of iron that can be made available to the body through absorption by intestinal mucosa. On the other hand, cereals, especially maize, have compounds called phytates that inhibit the absorption of iron (Mensforth et al. 1978; Stuart-Macadam 1989; 1992; Roberts and Manchester 1995). Phytates, phosphates and carbonates bind iron to insoluble macromolecules, which the body cannot synthesize (Mensforth et al. 1978). Moreover, cereals themselves have poor iron content.

Many bacterial and viral pathogens that invade the human body require serum iron for replications but do not have their own iron supply. Such pathogens are able to compete with their host for iron to maintain their growth and reproduction. In order to prevent replication of microorganisms, the human body reduces the amount of serum iron and also reduces the absorption of iron from food. Thus the body produces a hypoferric (iron deficiency) state to deprive pathogens of essential iron supply. This physiological response is termed 'nutritional immunity' (Mensforth et al. 1978; Stuart-Macadam 1992; Kent et al. 1994). In infants and young children both mild and severe infections can result in much lowered hemoglobin levels. Adults can resist infections much better than children without producing anaemic lesions. Rather than dietary shortcomings, Stuart-Macadam proposed that chronic disease and infection are the major forces behind the etiology of acquired anaemia especially in modern populations (Stuart-Macadam 1989; 1992; Kent et al. 1994).

Morphological alterations of the skeleton, commonly known as porotic hyperostosis and cribra orbitalia, result from red marrow proliferation with anaemic stimulus as a predisposing factor (Mensforth et al. 1978). It is often not easy to identify the nature of anaemia an individual suffered from on the basis of dry bone only. However, severe haemoglobin disorders like thalassemia can be distinguished on the basis of the involvement of the postcranial skeleton (Ortner and Putschar 1981). Thalassemia can cause joint necrosis, deformation of the vertebral column and coarse trabaculation of long bones (Palkovich 1987).

The most notable skeletal feature of anaemic condition is the involvement of skull where lesions develop on the frontal bone, orbits, the parietal bones, the mastoids, diploe and on the occipital bone (Ortner and Putschar 1981; Stuart-Macadam 1992; Roberts and Manchester 1995). It is common for orbital lesions and calvarial lesions to co-exist on the same skull and when they do, they appear separate from each other (Ortner and Putschar 1981). Lesions associated with anaemia are more pronounced in young individuals and they often disappear in adulthood as a result of bone resorption and remodeling. In adulthood haemopoietic bone marrow can be doubled without altering the bone and thereby reducing chances of forming lesions (Mensforth et al. 1978; Ascenzi et al. 1991).

6.5.1.1 Cribra orbitalia

Cribra orbitalia occurs as multiple pores within a confined locality on the roof of the orbits. The outer table of the orbits is destroyed and thereby pores of cancellous bone of the diploe are exposed. The involvement of the orbits in anaemia is usually symmetrical (Nathan and Haas 1966; Mensforth et al. 1978; Stuart–Macadam 1991).

Methods

Each individual was examined for orbital lesions. Based on the knowledge that cribra orbitalia is usually symmetrical (Mensforth et al. 1978; Stuart-Macadam 1991) where only one orbit was present the scoring was taken to be reflective of the missing orbit. For instance, an individual with only the left orbit and with that orbit having no cribra orbitalia was concluded to have been unaffected. Only those individuals with one or both orbits observable were included in the analysis. Individuals whose orbits were missing or severely damaged were not included.

Mensforth et al. (1978) devised a criterion in which anaemic lesions could be classified on the basis of whether or not the disease was active or healed at the time of death. Remodeled or inactive lesions are those associated with earlier episodes of anaemia, in which case the lesions were in the process of healing at the time of death. Unmodeled lesions are those associated with active disease in which case the individual would have been suffering from anaemia around the time of his or her death (Mensforth et al. 1978). Due to the problematic nature of this classification, no attempts were made

to classify anaemic lesions from the sample being studied. In addition, the affected sample itself is too small to allow for meaningful subdivisions.

Results

Of the 84 individuals included in this study, 49 were not observable and thus only 35 individuals had one or both orbits. Out of these 35 individuals, eight had one orbit each (five with right orbits and three with left orbits) and 27 had both orbits. Twenty-nine of the 35 observed individuals had no cribra orbitalia and only six had lesions. These included five individuals from Toutswemogala Burial 2 (five to seven years), Burial 3 (seven to eleven years), Burial 9 (seven to nine years), Burial 13 (seven to nine years) and Burial 16 (10 to 12 years). One individual was from Kgaswe B-55 (Burial 15 aged between eight and 12 years old). At least five of the affected individuals had both orbits. The lesions ranged between small, shallow pores covering a very small area to large deeper pores covering a wider area on the roofs of both orbits. The sex distribution of affected individuals was not assessed since the individuals themselves were immature and no attempts had been made to determine their sex. The most severe or well-developed cribra orbitalia was noted on Toutswemogala Burial 16 (Figure 6.12), an older child aged between 10 and 12 years. The individual had large, deep pores covering a relatively large area on the roofs of both orbits.

A comparison of cribra orbitalia between Toutswe and K2/Mapungubwe samples was carried out. A total of 38 individuals from the K2/Mapungubwe had orbits (Steyn and Henneberg 1995a) while 35 individuals from Toutswe sites had orbits. Fifteen of the 38 individuals with orbits at K2/Mapungubwe were found with cribra orbitalia while on the Toutswe sample only six of the 35 individuals demonstrated these lesions.

Table 6.2 shows a summary of the age distribution of individuals affected by cribra orbitalia from the study sample. The table includes data from only two sites where individuals with cribra orbitalia were identified. None of the individuals with this defect was below five years or above 15 years. Table 6.3 summaries the comparison with other South African skeletal populations. The result indicates that there was less cribra orbitalia on the Toutswe skeletons than at K2/Mapungubwe. Thirty-eight skulls from K2/Mapungubwe were examined and 15 of them had cribra orbitalia. The number of affected individuals on the Toutswe sample is less than half the number affected at

K2/Mapungubwe and Oakhurst. In both samples, the lesions are generally mild. From Oakhurst, 11 individuals out of 18 were affected. The incidence of these lesions at Oakhurst appears to be much higher than at Toutswe and K2/Mapungubwe. It should however be kept in mind that the Oakhurst sample is small. The more recent skeletons from Riet River and Kakamas much less incidences of cribra orbitalia.

At K2/Mapungubwe, the presence of cribra orbitalia was evaluated in the following age categories; younger than two years, 2 -13 years, 13 - 20 years and older than 20 years. Each of these age categories have at least one or more individuals with cribra orbitalia (Steyn and Henneberg 1995a). Thus not only are cribra orbitalia lesions higher in the K2/Mapungubwe skeletons by comparison to Toutswe skeletons, it is also distributed across a broad spectrum of age categories at K2/Mapungwe as opposed to Toutswe. None of the Toutswe infants below two years were found with lesions but one child of below two years was found with lesions on the K2/Mapungubwe skeletons. At Oakhurst, one individual of approximately six months old had cribra orbitalia. There were also seven juveniles, two adult females and adult two males. They are aged between zero and 40 years old (Patrick 1989).

The differences in the prevalence of cribra orbitalia on the various samples are not fully understood but subsistence strategies and pathogen invasions are possible reasons. Riet River and Kakamas are pastoral groups with possibly less reliance on cereals than the Toutswe and K2/Mapungubwe farmers and herders. The Oakhurst community on the other hand is a pre-pastoral group and the sample size could be a reason for the high prevalence of cribra orbitalia seen. It thus seems that the incidence of cribra orbitalia is much lower at Toutswe, as compared to Iron Age sites at K2 and Mapungubwe, and is also only present in juveniles. This probably indicates that the stressors causing these lesions were less at the Toutswe group of sites.

Figure 6.12 Cribra orbitalia on Toutswemogala Burial 16.

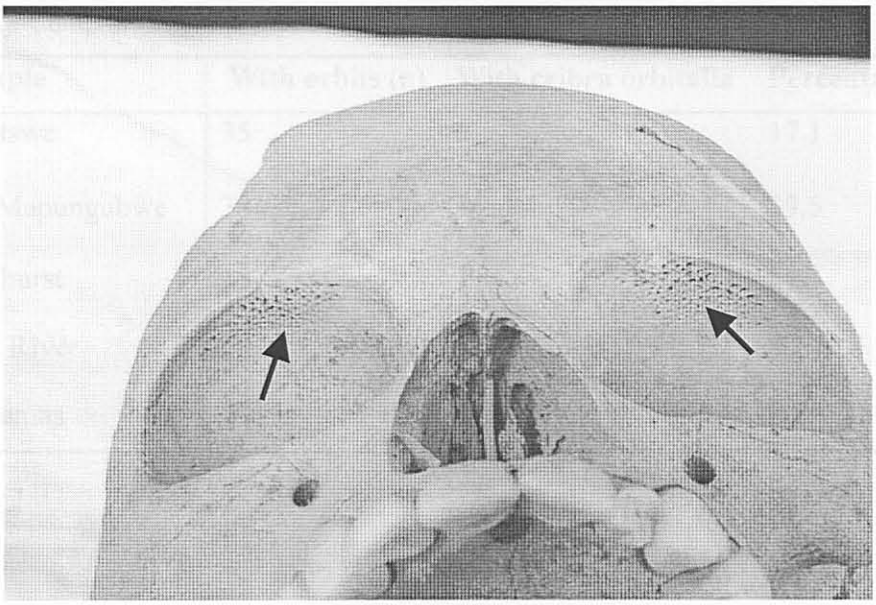


Table 6.2 Summary of age distribution of cribra orbitalia

Site	<5y	5-10y	10-15y	Total
Toutswemogala	0	4	1	5
Kgaswe B-55	0	0	1	1
Total	**0**	**4**	**2**	**6**

Table 6.3 Comparison of the prevalence of cribra orbitalia on different samples

Sample	With orbits (n)	With cribra orbitalia	Percentage
Toutswe	35	6	17.1
K2/Mapungubwe	38	15	39.5
Oakhurst	18	11	61.1
Riet River	74	7	9.5
Kakamas	53	2	3.8

6.5.1.2 Porotic hyperostosis

Porotic hyperostosis has been studied as far back as the late nineteenth century but at the time was thought to be of different causes, such as carrying water jugs on the head and geographic location (Angel 1964; 1966; Stuart-Macadam 1989). The geographic location as a predisposing factor to porotic hyperostosis was partly based on the distribution of areas infested with *falciparum* malaria (Angel 1966; Ortner and Putschar 1981). The main interest at the time was to describe the lesions and hypothesize on their etiology. Porotic hyperostosis was viewed as a direct evidence for nutritional stress and this led to a general misconception that those archaeological populations with high incidence of porotic hyperostosis were nutritionally disadvantaged (Angel 1964; 1966; Hengen 1971; Mensforth et al. 1978; Stuart-Macadam 1991; 1992; Kent et al. 1994). It was not until the late 1920s that a breakthrough in the understanding of the aetiology of porotic hyperostosis was made. Radiographic similarities between the lesions in clinical cases and archaeological cases were identified. Since the clinical cases were mostly of individuals with iron deficiency anaemia, the archaeological cases were readily interpreted as having resulted from anaemia (El-Najjar and Robertson 1976; Stuart-Macadam 1991; 1992).

Porotic hyperostosis is a generic term used to describe skeletal lesions resulting from a decrease in bone density (Ortner and Putschar 1981; Ascenzi et al. 1991). It has

the same aetiology as cribra orbitalia. Bone density decreases in response to increased haemopoietic bone marrow. The lesions appear as small pores on the cranial vault, especially on the temporal, parietal and occipital bones. They range in size and appearance between small holes on a small or localised area to large openings on a larger area (Ascenzi et al. 1991; Stuart-Macadam 1991; 1992). The lesions are frequently found around the mastoids, around the bregma and on the posterior surface of the skull. Porotic hyperostosis is less common but almost always found in association with cribra orbitalia (Mensforth et al. 1978).

Individuals with anaemia may display thicker diploe than normal (Mensforth et al. 1978; Ortner and Putschar 1981; Stuart-Macadam 1991; 1992). In thalassemia cases, the diploe expands leading to a reduction in the number of the trabeculae. The external table is eroded and new subperiosteal bone developing creates a 'hair on end' appearance (Ortner and Putschar 1981).

Methods

All cranial bones present were observed regardless of whether the skull was complete or not. Where only a few cranial fragments were present and were included. Therefore the analysis included all individuals with complete and incomplete skulls. Individuals of all ages were included in this analysis.

Results

Five individuals with porotic hyperostosis were identified on five individuals and the lesions are distributed on the temporal, frontal and occipital bones with little evidence on the parietal bones. Cribra orbitalia was found in association with porotic hyperostosis in two cases, Kgaswe B-55 Burial 15 (10-12 years) and Toutswemogala Burial 2 (five to seven years old). Two individuals had no cribra orbitalia and they are Swaneng Hill Burial 1, an adult male aged between 20 and 30 years, and Toutswemogala Burial 8, a child of five to seven years. One individual, Thatswane 3, aged between three and five years, had no orbits.

On the K2/Mapungubwe sample, only one individual had porotic hyperostosis. The individual was aged approximately eight years (Steyn and Henneberg 1995a). No cases of this condition were reported on the Oakhurst sample. Peckmann (2002) also

found a few cases. Thus the incidence of porotic hyperostosis on Toutswe exceeds K2/Mapungubwe, but cribra orbitalia shows a higher incidence K2/Mapungubwe than Toutswe. In all samples the numbers of individuals affected are too small to make statistical inferences from. It therefore appears that the Toutswe results are within ranges previously quoted for southern Africa.

6.6 Enamel hypoplasia

Enamel hypoplasias are bands or pits marking levels where enamel is thin (Goodman et al. 1980; 1984a; 1984b; 1987; Lukacs 1989; Goodman and Rose 1990; 1991; Hillson 1996; Aufderheide and Rodriguez-Martin 1998) as a result of the cessation of ameloblast activity during developmental stages of a tooth. During the secretion phase of amelogenesis, stress resulting from nutritional deficiency or other factors, e.g. major acute bacterial invasion, retards or withholds the growth of enamel. Once the stress is relieved, enamel growth starts again. When growth restarts, a band or pit of thin enamel is left marking the cessation of growth (Goodman et al. 1980; 1984a; 1984b; 1987; Goodman and Rose 1990; 1991; Aufderheide and Rodriguez-Martin 1998). Enamel hypoplasia occurs only during developmental ages and therefore any stress experienced after the completion of amelogenesis does not produce lesions on teeth. Two broad categories of stress that causes enamel defects are malnutrition and diseases (Skinner and Goodman 1992). In malnourished children, the material needed for matrix formation or mineralisation are supplied in quantities below normal requirements or are not supplied at all. A disease, on the other hand, impairs normal cellular function.

The ability of developmental defects of enamel to progress to stages where they are observable depends on two factors. First they depend on the susceptibility of an affected individual or group to environmental insults and secondly, on the susceptibility of the dental tissue to allow for a reactive response and thereby creating a record of the stress (Skinner and Goodman 1992; Wood et al. 1992). Enamel hypoplasia is only an indicator of strenuous episodes during developmental years (approximately five months in utero to about seven years) when amelogenisis of deciduous and permanent teeth is at its highest. It may also be detected at the ages of 10 to 16 years when third molar amelogenisis is occurring (Anderson et al. 1976; Goodman et al. 1980; Goodman and Rose 1990; 1991; Skinner and Goodman 1992). In many of case studies (e.g. Goodman et

al. 1980; 1984a; 1984b; 1987; Lanphear 1990; Steyn and Henneberg 1995a), it has been found that enamel hypoplasia occurs mainly between two and four years which is the period corresponding to the hype of ameloblast activity.

Unlike bone, enamel cannot remodel once formed. Therefore, lesions produced during the early years remain permanently unless other diseases or post depositional damage (Goodman and Rose 1980; Goodman et al. 1984; Aufderheide and Rodriguez-Martin 1998) destroys affected teeth. Previous studies have shown that mandibular permanent canines and maxillary central incisors are the most prone to enamel hypoplasia. These teeth have a relatively longer developmental period and therefore their ameloblast activities have more chances of being disrupted (Goodman et al. 1980). By measuring the distance between the cemento-enamel junction and enamel hypoplasia, the age at which enamel hypoplasia occurred can be estimated.

Methods

Enamel hypoplasia was recorded as present or absent where the enamel was observable. All teeth were analysed, regardless of the side from which they came. In cases where the enamel had been destroyed by either pathology or post-depositional processes, enamel hypoplasia was recorded as unobservable. All deciduous and permanent teeth present were examined. From the literature surveyed, there is no clearly defined criterion to score the severity of enamel hypoplastic lesions. Without proper guidelines, no attempts were made to differentiate the severity of lesions found. However, if a lesion appeared to be more pronounced than the others, such a case was noted. A distinction between pits and linear lesions was made.

All lesions recorded were measured from the cemento-enamel junction to the nearest millimeter using a digital sliding caliper. The distances were used to estimate ages at which enamel hypolasitic lesions occurred and only permanent teeth were included. Regression equations for estimating age at which enamel hypoplastic lesions formed on permanent teeth is detailed in a publication by Goodman and Rose (1991). The distances measures were fit into relevant regression equations to estimates ages of formation. A total of 50 lesions from 44 teeth were included in the estimation of age at which lesions formed.

Results

Due to either pathological and/or post-depositional damage, 20 permanent teeth were excluded from the analysis. None of the deciduous teeth were excluded. A total of 53 out of 784 teeth from 16 individuals had been affected by this developmental defect. The teeth affected include eight central incisors, five lateral incisors, twenty-two canines, five first premolars, two second premolars, two first molars, five second molars (all permanent) and three deciduous canines. Table 6.4a shows the distribution of permanent teeth affected by enamel hypoplasia and Table 6.4b summarises data on deciduous teeth. The number of each tooth type affected is expressed as a percentage of the total number of teeth affected. Table 6.3a shows that the most commonly affected permanent tooth is the canine (26.83%).

A comparison between permanent maxillary and mandibular teeth indicates that mandibular teeth were more affected than maxillary teeth. Of the 50 permanent teeth affected, 40 are mandibular and 10 maxillary. In both the maxilla and mandible, the most commonly affected tooth is the canine, followed by the lateral incisor of the maxilla and central incisor of the mandible.

Out of 46 individuals examined, 14 had enamel hypoplasia i.e. 30.4% of the individuals. The number of teeth involved per individual ranged between one tooth (e.g. Bosutswe Burials 5 and 6) and nine teeth (Bosutswe Burials 9 and 13). Of the 53 teeth involved, 11 had pitting enamel hypoplasias and the remaining 42 had horizontal linear enamel hypoplasias. Only one individual had both types of lesions (Toutswemogala Burial 6), but on different teeth. None of the teeth displayed multiple pitting enamel hypoplastic lesions but multiple horizontal linear lesions per tooth were noted on four individuals. Most of the recurring lesions occurred on mandibular canines.

The ages at which enamel hypoplastic lesions were formed is presented in Table 6.5. The ages were estimated on permanent teeth only. The data used in the table was pooled from mandibular and maxillary teeth, because a small number of lesions were identified. The earliest age at which enamel hypoplastic lesions were formed was at 0.8 years. This age was recorded on a mandibular central incisor of Toutswemogala Burial 6. At time of death this individual was approximately 9-11 years old. The oldest age of lesion formation was at six and a half years, estimated on a mandibular second molar of Kgaswe B-55 Burial 2. The individual had survived to be 30-50 years old. On the same

tooth an earlier episode of stress had occurred at approximately four and a half years. In Toutswemogala Burial 29 (6-10 years) the episodes were experienced at one and half, two and a half and four years on the right mandibular canine. A left mandibular canine of Bosutswe Burial 5 (17-20 years) had three lesions formed at two and half, three and nearly five years respectively. The average age of occurrence of hypoplastic lesions was 3.7 years.

Table 6.4a Distribution of enamel hypoplastic lesions on permanent teeth.

Tooth	n	Affected	Percentage
I1	62	8	12.9
I2	68	6	8.82
C	82	22	26.83
PM1	78	5	6.41
PM2	75	2	2.67
M1	104	2	1.92
M2	76	5	6.58
M3	46	0	0
Total	591	50	8.46

Table 6.4b Distribution of enamel hypoplastic lesions on deciduous teeth

Tooth	n	Affected	Percentage
i1	27	0	0
i2	30	0	0
c	35	3	8.57
m1	50	0	0
m2	51	0	0
Total	**193**	**3**	**8.57**

Table 6.5 Ages at which enamel hypoplastic lesions were formed on permanent teeth

Age (years)	I1	I2	C	PM1	PM2	M1	M2	Total
0-0.5								0
0.5-1.0	2							2
1.0-1.5			1					1
1.5-2.0								0
2.0-2.5	2	2	1	2		1		8
2.5-3.0	4	2	3			1		10
3.0-3.5		2	1					3
3.5-4.0			1	1				2
4.0-4.5			8	1				9
4.5-5.0			5		1		1	7
5.0-5.5			2	1	1		2	6
5.5-6.0							1	1
6.0-6.5							1	1
Total	**8**	**6**	**22**	**5**	**2**	**2**	**5**	**50**

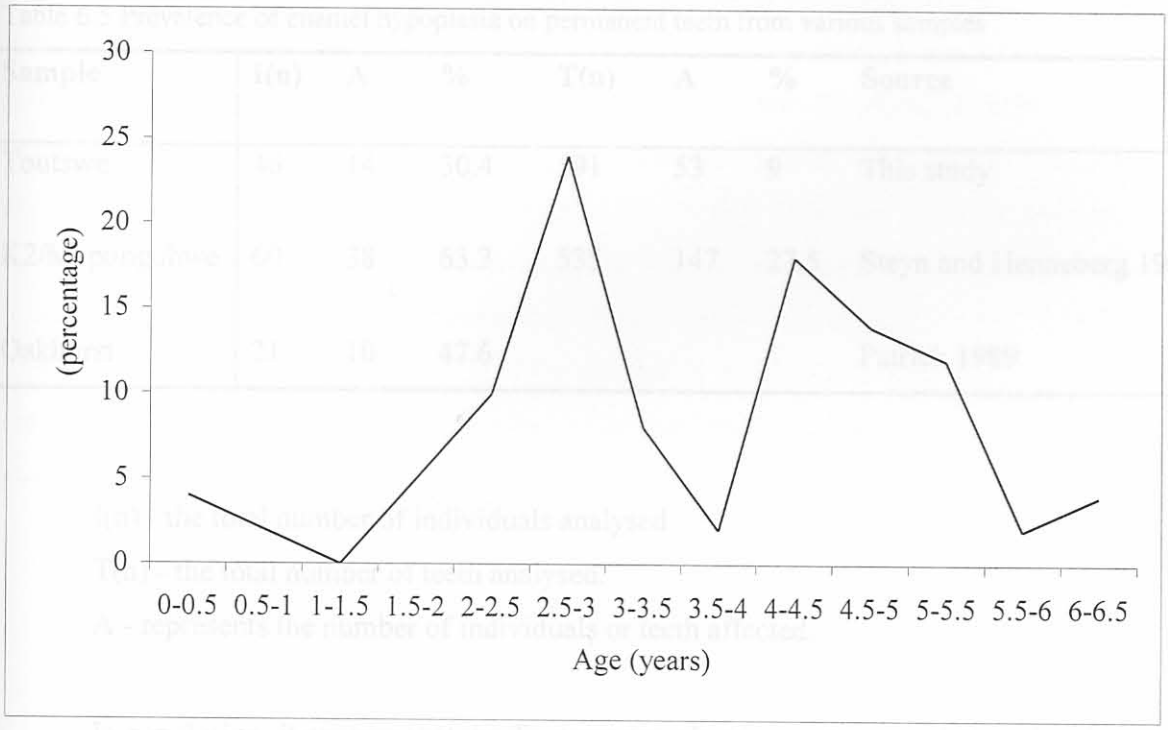
The chronology of formation of enamel hypoplasia on permanent teeth (Figure 6.13) shows a bimodal curve indicating that there were generally two phases during which children were exposed to stress. The figure shows that stress was minimal during the first year of life where less than five percent of the total hypoplastic lesions were formed. After one and half years, there is a rapid increase in the formation of lesions until two and a half to three years and the period that follows is characterised by a decline in the formation of enamel hypoplasia. The age of around three to four and half years is marked by the second phase of accelerated formation of hypoplastic lesions. The age at which the most number of lesions was recorded is between two and half and three years where approximately 25% of the total number of lesions was formed.

It is difficult to make sound comparisons of the distribution of teeth with enamel hypoplastic lesions between the Toutswe and K2/Mapungubwe samples. The K2/Mapungubwe sample is more than twice the Toutswe sample with 147 lesions (Steyn 1994; Steyn and Henneberg 1995a) while the Toutswe sample has only 53 lesions. However some similarities can be identified despite the sample size differences. For instance, the most commonly affected mandibular tooth in both samples is the canine. On the K2/Mapungubwe sample, the age of lesion formation raged between 1.1 to 6.5 years with an average occurrence of 3.7 years. The Toutswe sample shows an age range of lesion formation of 0.8-6.5 years with a mean of 3.7 years.

The distribution of ages at which lesions formed on the Toutswe population differs from that recorded on a modern sample from Chicago (Goodman and Rose 1990). The Chicago sample has a single peak during which most enamel hypoplastic lesions were formed. This peak was at approximately the end of the first year of life where more than 70% of the lesions were recorded. No lesions were recorded after the age of four on the Chicago sample whereas at Toutswe the last lesions were formed at about the age of six and a half years. The chronology of formation of hypoplastic lesions on an archaeological sample at Dickson Mounds distributed between the ages of six months to six and a half years similar to Toutswe. However the Dickson Mounds curve is unimodal with the peak occurring at approximately three years. About 30% of the total lesions were formed at three years (Goodman Rose 1990). The samples from America were selected as outliers whose subsistence and health are well understood. The bimodal curve displayed by the Toutswe sample may be a function of sample size. Other possibilities include

pathogen invasion at the time of weaning as well malnutrition when breast milk is stopped at the time of weaning.

Figure 6.13 Chronology of formation of enamel hypoplasia on the Toutswe population



The prevalence of enamel hypoplasia of three archaeological populations including the current sample is presented in Table 6.5. The table compares two important variables, the total number of individuals observed and the percentage of those affected and the total number of teeth analysed and the percentage of those affected. The data included comprises of pooled maxillary and mandibular teeth from all individuals. The K2 and Mapungubwe results have also been pooled.

The percentage of affected individuals at K2/Mapungubwe is more than twice the percentage affected at Toutswe. The Oakhurst sample is much smaller than the other two, but shows that nearly 50% of the individuals analysed were affected. The total number of permanent teeth analysed on the Toutswe sample is 591 while on K2/Mapungubwe it is

535. In contrast the number of teeth with lesions on the K2/Mapungubwe sample exceeds the number of teeth with lesions on the Toutswe sample by 94 (i.e. 147 versus 53).

Table 6.5 Prevalence of enamel hypoplasia on permanent teeth from various samples

Sample	I(n)	A	%	T(n)	A	%	Source
Toutswe	46	14	30.4	591	53	9	This study
K2/Mapungubwe	60	38	63.3	535	147	27.5	Steyn and Henneberg 1995a
Oakhurst	21	10	47.6				Patrick 1989

I(n) - the total number of individuals analysed

T(n) - the total number of teeth analysed.

A - represents the number of individuals or teeth affected.

In conclusion, it appears that the Toutswe sample represents a population showing relatively low levels of stress. The incidences of nonspecific makers of stress are low compared to K2/Mapungubwe. However, more recent individuals from historical sites (Morris 1984; Peckmann 2002) seem to have been healthier than the Toutswe people and other earlier populations. It therefore seems as if there is trend towards better health from earlier to modern groups.