Skeletal Manifestations of Infantile Scurvy

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ABSTRACT Recent investigations of human skeletal material from the historic St. Martin’s cemetery, England, found a range of abnormal lesions in six infants that are almost certainly related to scurvy. Porous and proliferative bone lesions affecting the cranial bones and scapulae were found, and this paper presents images obtained using both macroscopic and scanning electron microscope examination of the lesions. Previous work on infantile scurvy (Ortner et al., 1997–2001) relied heavily on changes at the sphenoid, which is often missing in archaeological bone, so the identification of changes attributable to scurvy on other cranial bones and the scapulae is encouraging. The ability to recognize changes related to scurvy on a range of bones will ensure an enhanced potential for recognition of this disease in future research involving archaeological bone. Research on historical documents from Birmingham dating to the eighteenth and nineteenth centuries, combined with the probable cases of scurvy identified, supports the view that the paucity of cases of infantile scurvy from the archaeological record reflects a lack of understanding and recognition of bone manifestations, rather than a lack of occurrence in this period. Changes linked to scurvy were only found in infants from the poorer sections of the community from St. Martin’s, and this is almost certainly linked to patterns of food consumption and may be related to shortages of potatoes, due to blight, experienced during this period. Am J Phys Anthropol 129:163–172, 2006. © 2005 Wiley-Liss, Inc.
In a more recent review of cases of scurvy in Thailand 93% of reported cases were in children between 1–4 years of age, although cases were reported in individuals as young as 10 months and as old as 9 years (Ratanachu-Ek et al., 2003). Clinical manifestations of scurvy are likely to develop after an infant has lacked vitamin C more or less completely for 6–10 months (Jaffe, 1972, p. 449; Resnick, 1988). However, it was suggested that the condition could appear in as little as 2–4 months of inadequate intake of vitamin C (Tamura et al., 2000). As Ortner et al. (1999) discussed, as the most rapid proportionate growth occurs in infancy and early childhood, the probability of forming defective blood vessels in this age group is greatest. Therefore, skeletal evidence of scurvy is expected to be highest during infancy and early childhood, and recent research on the dry-bone manifestations of scurvy examined juveniles aged from birth to 2.9 years and 3–6.9 years (Ortner et al., 2001).

Between 1997–2001, Ortner et al. (Ortner and Erickson 1997; Ortner et al. 1999, 2001) carried out various investigations of pathological features related to abnormal porosity of the cortex. They suggested that the pathological lesions identified are a response to chronic bleeding at the site of the porosity or hyperplasia (Ortner et al., 1999), and related such abnormal bleeding to scurvy (see also Jaffe, 1972). Using some of the criteria developed by Ortner et al. (1997–2001), and some additional observations of skeletal material, this paper presents the first cases of infantile scurvy identified from this important period in British history. In order to highlight these important changes, which are under-reported in archaeological investigations, the range of less severe skeletal changes recordable in archaeological juvenile bone is detailed. The range of changes identified and illustrated provides information on criteria that may enable diagnosis of less severe cases of infantile scurvy. Documentary evidence relating to the important historic burial ground of St. Martin’s, Birmingham, England, provides interesting information on the socio-economic status of individuals investigated and possible reasons for the development of scurvy in infants from poorer families. The ability to recognize metabolic conditions such as scurvy is important because this type of material allows important additional information on socioeconomic aspects of past societies to be determined. The purpose of this research is to identify probable cases of infantile scurvy in the skeletal collection from St. Martin’s, Birmingham, England, and to consider possible causes for the pattern of pathological changes recorded in different socioeconomic groups.

MATERIALS AND METHODS

Excavations carried out between May and November 2001 at St. Martin’s churchyard, Birmingham, England, recovered 857 individuals dating to the late Eighteenth and Nineteenth centuries. Individuals from the site came from two different types of burial; earthcut graves and vault burials. This difference in burial treatment reflects socio-economic status of the individuals with the wealthier middle class using the vaults and poorer individuals being buried in earthcut graves. To obtain information for inclusion in the site report (Brickley and Buteux, in press) all individuals removed from vaults (99) and 406 of the individuals excavated from earthcut graves were analyzed. Age determination undertaken as part of this recording demonstrated that 20 of the individuals from vaults and 133 of those from earthcut graves were classified as juveniles. As part of the present study, it was possible to undertake recording of larger numbers of individuals than had been previously possible and in total, 164 juveniles were examined in detail for evidence of metabolic bone diseases.

Following a review of the Ortner et al.’s (1997–2001), criteria for the diagnosis of scurvy, detailed investigations were undertaken on all juvenile bone. Specifically, all cranial bones, scapulae, and long bones were examined macroscopically for the presence of an increased vascular response comprising abnormal porosity penetrating the cortex. Other paleopathologically recognized lesions of scurvy include proliferative bone formation, particularly as a bone response to hemorrhage in the orbits (Roberts and Manchester, 1997, p. 173). Where present, new bone formation on the cranial bones and long bones was also recorded. In order to better understand the nature of abnormal porosity compared to the normal bone surface, small samples from the orbits and scapulae were also examined under a scanning electron microscope. It was possible to undertake destructive analyses on the human bone as the collection could not be retained for future investigation, but was to be re-buried. Reburial of the St. Martin’s collection has now taken place. Abnormal porosity in the bone surface can occur in a number of pathological conditions (see Discussion).

Therefore, the pattern of abnormal porosity occurring throughout the skeleton is important in the diagnosis of scurvy. Detailed discussion of the pattern of bones affected in the individuals studied here is presented in Results.

Six infants, aged birth to 3 years, as defined by Buikstra and Ubelaker (1994) displayed a range of manifestations of possible scurvy. In this study, it was deemed necessary to divide this wide age category. Therefore we identified five young infants, aged birth to 6 months, and one older infant aged 1–2 years with abnormal skeletal lesions (summarized in Table 1).

RESULTS

This study presents descriptions and illustrations of a range of bone lesions identified through visual analysis and investigations using a scanning electron microscope (SEM), in order to aid identification of these lesions in the future.

Maxilla

Two young infants from this collection were identified with abnormal porosity around the infraorbital foramen and surrounding region of alveolar bone. Figure 1 presents a lateral view of a maxilla from one individual. The alveolar bone in the region superior to the molar crypt and inferior to the line of articulation with the zygomatic, and extending anteriorly towards the nasal spine and superiorly towards the frontal process, is abnormally vascular, with definite channels penetrating the cortex. The vascular holes are quite substantial in size, but are considerably smaller than the infraorbital foramen seen in the center of the bone. Ortner et al. (1999, p. 327) argued that identifying abnormal porosity of the maxilla is difficult, as any assessment has to take into consideration normal porosity that typically surrounds the alveolar processes of erupting teeth. Therefore, for maxillary porosity to be defined as pathological, the area of porous involvement should extend well beyond the alveolar processes surrounding an erupting molar. Figure 1 illustrates porosity extending above the alveolar process of the molar.
crypt. The abnormal porosity is severest in the region of the cortex superior to the alveolar processes toward the frontal process. In contrast, the degree of normal, small, and localized porosity typical around the alveolar pits and infraorbital foramen of an age-matched normal infant is demonstrated in Figure 2.

Ortner and Ericksen (1997) attributed porosity of the maxilla and other cranial bones to bleeding of the gums related to minor trauma to blood vessels caused during mastication (see also Ortner et al., 2001). The two individuals identified with abnormal porosity on the maxilla in the present study were too young to have started chewing more solid food akin to those individuals studied by Ortner et al. However, movement of the facial muscles and blood vessels in sucking and swallowing actions occurring while these younger individuals were being breastfed or receiving artificial feeding may have been enough stimulus to cause bleeding and subsequent vascular changes around the maxilla.

**Hard Palate**

That the subadult hard palate displays normal porosity, distributed in a U-shaped arc adjacent to the alveolar processes of the erupting teeth, is clearly defined by Ortner et al. (1999). If scurvy is manifest in an individual, vascular changes of the alveolar processes and tooth sockets are likely to occur on the maxilla and other cranial bones. However, cortical porosity in these regions will also occur during tooth eruption, and distinguishing growth-related porosity from that which is pathological remains difficult (Ortner et al., 1999). This was recently confirmed by Melikian and Waldron (2003), who examined porosity around the alveolar margins in pathology museum specimens and clarified its common occurrence. However, Ortner et al. (1999, p. 327) described the region of normal porosity of the hard palate as becoming denser toward the anterior portion of the palate. They defined pathological porosity as existing where the denser region of porosity becomes more enhanced and extends markedly into the posterior portion of the palate, rather than remaining limited to the anterior portion.

Three young infants displayed porosity through the cortex that was considered, in light of the description of Ortner et al. (1997–2001), to be abnormal. Ridges of bone formation were identified with porosity adjacent to the alveolar processes, and these were considered normal and indicative of growth of the tooth sockets. However, toward the antero-medial portion of the palate and posterior to the incisive fissure, prominent vascular porosity was apparent, which penetrated the existing cortex. In one individual (Fig. 3), small, localized areas of minute holes in the cortex were spread throughout the palate from the alveolar pits and incisive fissure towards and along the intermaxillary suture, and this extended spread of porosity was considered abnormal.

Breast-feeding or eating solid foods could, with an underlying vitamin C deficient diet, stress the soft-tissues of the mouth and hard palate, resulting in some trauma to underlying blood vessels in the mouth. Ortner et al. (1999, p. 327) noted that the greater palatine artery provides the blood supply to the hard palate, and the vessels of this
artery participate in the vascular response to chronic hemorrhage in the alveolar processes and adjacent hard palate. It is this secondary vascular response that can stimulate increased porosity in the bony palate of scorbutic subadults (Ortner et al., 1999).

**Mandible**

Ortner et al. (1999) identified abnormal porosity affecting the medial surface of the mandibular coronoid process at the insertion of the temporalis muscle. In the present study, two young infants displayed increased vascularization of the cortex in this region. Large holes were observed penetrating the existing surface on the medial side of the coronoid process and toward the pterygoid fovea, as well as covering the region of the lingua and surrounding the mandibular foramen (Fig. 4). However, it was notable that the porosity did not continue extensively past the medial side of the mandibular foramen, and it did not reach the inferior line of the alveolar pits at the beginning of the arc of the dentition. If this porosity was growth-related, then evidence of continued vascular impressions along the inferior edge of the forming alveolar processes, where active growth would be taking place at this age, would be expected. The area of bone affected by porosity in individuals in the present study is more extensive than that observed by Ortner et al. (1999), whose examples show porosity localized to the medial coronoid process. The more extensive porosity at this location may be symptomatic of the severity of bleeding at this site, and may also indicate that the inferior alveolar nerve, vessels, and soft tissue that enter and surround the mandibular foramen were affected by trauma or suffered defective formation during the vitamin deficiency, generating a vascular response to hemorrhaging.

**Orbits**

The orbits of three young infants displayed increased vascularization of the cortex in the orbital roof and toward the lateral edge of the orbit margin (Fig. 5). A feature common to all the young individuals studied was that of bone being laid down in a layered effect toward the center of the orbit. This organization of bone is likely to reflect a normal growth mechanism of this age group, as also observed in infants who did not have scurvy (compare Fig. 5a and 5b). However, the roof of the orbit in the pathological examples displays intensely vascular regions with substantial holes clearly penetrating the existing cortex, and it is suggested that this porosity is abnormal (Fig. 5a), as comparison with a normal orbit of this age group demonstrates few localized regions of small holes in the bone surface, which do not appear to be very dense (Fig. 5b). The SEM features of the younger infants with intensely vascular orbits clearly demonstrate the mass of porous cortical bone formed as a new layer on top of the existing bone surface (Fig. 5c). Bone is not being laid down in normal layers, suggesting that it is formed in a rapid response to inflammation or trauma. In an age-matched normal infant, layers of relatively solid bone are deposited from the orbital roof toward the center of the orbit (Fig. 5d). In several examples, some porosity was apparent as holes within this cortical bone, which are likely to relate to the vascular nature of growing bone. However, this pattern of porosity is not marked and is fairly randomly dispersed throughout the bone plate. The edges of the layers of bone appear to remodel into the underlying solid cortical bone, while retaining some aspects of vascularity. Toward the center of the orbit, the typical solid cortical bone is apparent.

The appearance of scurvy in an older infant demonstrates lesions typical of a more severe or long-standing disease presence, as extensive proliferative bone changes in the orbit are clearly visible macroscopically (Fig. 5e). The new bone formation in the orbital region constitutes a discrete layer of bone spread across the orbit surface consisting of spongy and irregular bone when compared with the normal solid cortex of an age-matched infant (Fig. 5f). SEM images of the region displaying new bone formation reveal that the layer of new bone is highly vascular, with holes penetrating through the cortex (Fig. 5g). The margins of new bone are irregular and spiculated compared to the definite solid layer of cortex observed as normal. The age-matched normal individual does have some small random holes in the surface of the cortex, but these are not deep and are not indicative of an intensely vascular reaction (Fig. 5h). The severity of hemorrhaging in this individual must have stimulated osteoblastic bone
Fig. 5. Composite showing range of abnormal manifestations attributed to scurvy compared with normal counterparts. a: Orbital roof of young infant with probable scurvy, showing intensely vascular bone. b: Orbital roof of normal young infant, showing only slight porosity in orbital margin and normal layering of bone growth toward center of orbit. c: SEM image of orbital roof of young infant with probable scurvy, showing mass of extremely vascular cortical bone formed as rapid response to inflammation. d: SEM image of orbital roof of normal age-matched young infant, showing normal growth-related layers of bone and some regions of porosity limited to orbital margin that are becoming incorporated into underlying solid bone. e: Orbit of older infant with probable scurvy, showing proliferative new bone in discrete layer across orbit. f: Normal solid cortical surface of orbit of age-matched older infant. g: SEM image of irregular and spiculated new bone in orbit of older infant, with proliferative bone growth indicative of scurvy. h: SEM image of age-matched normal older infant orbit, showing solid cortex with a few random holes, in contrast to intensely vascular response identified in g.
formation similar to that observed in long bones in severe cases (Aufderheide and Rodriguez-Martí, 1998).

Ortner and Erickson (1997, p. 213) suggested that defective blood vessels formed during the vitamin deficiency result in an increased susceptibility to hemorrhage, and they proposed that normal movement is enough stimulus to cause bleeding and prompt a vascular response. They argued that normal eye movement is enough to provoke such a reaction, although they suggest that bleeding will be more evident following trauma (see also Aufderheide and Rodriguez-Martín, 1998, p. 311). As the individuals with increased orbital porosity identified here were very young, it is likely that the blood vessels developing during the last fetal months and/or after birth may well have been defective if formed during a vitamin deficiency. Therefore, it is possible that slight movements of the eye could result in hemorrhaging, thus provoking the vascular response suggested by Ortner and Erickson (1997).

Ortner et al. (2001) described the abnormal feature of scurvy manifest macroscopically to be abnormal porosity penetrating the cortex of the orbit. The results of the current investigation confirm the macroscopic presence of such irregular and abnormal vascularity. As such, this evidence supports the suggestion of Ortner et al. (2001) that these features probably represent skeletal manifestations of scurvy at an earlier or less advanced stage than the more commonly recognized proliferative bone lesions. However, microscopically, some porosity penetrating the cortical bone of the orbit is a consistent feature of normal young infants, where it occurs in the formation of layers of new bone in the orbits. It is the increased concentration of vascularity apparent within thickened layers of rapidly formed new bone in the orbital roof that can help identify scurvy in future research.

**Parietals**

The ectocranial surface of the parietals of one young infant (HB767) displayed extensive porosity, indicating a severe vascular reaction (Fig. 6). No bone proliferation was apparent. Increased vascularization, with holes of an irregular size and pattern, was present on much of the bone surface, and the porosity clearly penetrated through the cortex. It is the presence of this severe vascular response affecting the parietals, combined with the pathological changes in the orbits and on the sphenoid (see below) in this individual, that suggested the presence of an underlying vitamin C deficiency at time of death.

**Occipital**

Three young infants displayed bone changes on the endocranial surface of the occipital bone that are suggestive of scurvy. In two cases (HB767 and HB802), a localized region of increased vascularization of the cortex is apparent, with large holes penetrating the cortex (Fig. 7). SEM imaging confirmed a coarsening of the cortical bone, which appears very vascular and slightly raised from the normal bone surface. The occipital of the third young infant with these changes (HB316) did demonstrate spicules of disorganized new bone on the lesser wings and part of the body of sphenoid (Fig. 8), as well as abnormal porosity on other cranial bones. The pattern of changes observed throughout the other skeletal areas in this individual indicates that scurvy was present.

**Sphenoid**

In the research presented previously (Ortner et al., 1997–2001, great emphasis was placed on the diagnostic potential of the greater wings of the sphenoid to portray the lesions identified as probable scurvy. While it is important that such consistent features can be used, an overreliance on a single skeletal indicator should be avoided, especially when dealing with archaeological bone, where poor preservation may limit the appearance of the bone element. In the present study, no sphenoids with complete greater wings were available for analysis. However, one young infant (HB767) did demonstrate spicules of disorganized new bone on the lesser wings and part of the body of sphenoid (Fig. 8), as well as abnormal porosity on other cranial bones. The pattern of changes observed throughout the other skeletal areas in this individual indicates that scurvy was present.

**Scapulae**

Abnormal porosity on the supraspinous and infraspinous areas of the scapula was also observed by Ortner et al. (2001, p. 347), which they associated with the manifestations of scurvy similar to those apparent in the cranial bones. In post-mortem records of children affected by scurvy, Barlow (1883) identified swelling over the scapulae caused by blood clots, notably affecting the infraspinous fossa (1883, p. 228, 239, 240). It is likely that blood vessels supplying the supraspinous and infraspinous muscles and underlying the bone suffer trauma under normal muscle contractions in individuals with scurvy. This results in hemorrhaging that stimulates a vascular reaction, visible on the dry bones (Ortner et al., 2001).

The five young infants examined in the current study all displayed manifestations of porosity affecting the supraspinous fossa of the scapula. Of these infants, only one displayed additional porosity on the infraspinous area (HB612). Figure 9 shows the extent of increased vascularization on the surface of cortical bone underlying the region of supraspinous muscle. Holes of various sizes are apparent, and these are clearly visible when examined with SEM imaging and when compared to a normal solid bone surface of this region (Fig. 9). The older infant with
between 1997–2001, Ortner et al. investigated pathological lesions of abnormal porosity of the cortex sometimes, although not consistently, accompanied by new bone formation. Ortner et al. (1999) suggested that pathological lesions represent a vascular response to chronic bleeding attributable to scurvy (see also Jaffe, 1972). The exact mechanism causing the abnormal bleeding is unclear. Chronic bleeding occurs commonly at sites where blood vessels are near skin surfaces or are stressed by muscle activity (Jaffe, 1972). Contraction of muscles is enough to traumatize already defective blood vessels, and causes chronic bleeding (Ortner et al., 2001). Bleeding triggers a vascular response that includes the formation of additional defective blood vessels and increased vascular pathways through bone in situations where the stimulus occurs on or near a bone surface (Ortner et al., 1999, 2001), ultimately resulting in the holes of blood vessels penetrating the underlying cortex (Ortner et al., 1999, p. 322).

Abnormal porosity. The primary pathology of scurvy identified previously (Ortner et al., 1997–2001) is abnormal porosity caused by blood vessels penetrating the bone surface, sometimes, although not consistently, accompanied by periosteal new bone formation. Abnormal porosity was defined by Ortner et al. (1999, p. 323) as a localized condition in which fine holes, typically less than 1 mm in diameter, penetrate a compact bone surface and are visible either with or without low magnification. Vascular holes in cortical bone are a normal anatomical feature of juvenile bones. However, normal vascular holes are much fewer in number in a given area and more variable in size, often exceeding 1 mm in diameter (Ortner et al., 2001, p. 344). Ortner et al. (2001) argued that it is the pattern of porous lesions at multiple sites within a skeleton that identifies the presence of a systemic disease with, in most cases, a single underlying cause. On any isolated bone it could be difficult to distinguish changes caused by infectious inflammatory diseases, such as hematogenous osteomyelitis, with those produced in the early stages of scurvy. In all cases, when suggesting a possible diagnosis for changes seen, it is important to consider the pattern of changes across the skeleton. Abnormal porosity was identified on a number of cranial bones including the greater wing of the sphenoid, the roof and lateral margins of the orbit, the posterior maxilla, the interior surface of the zygomatic bone, the infraorbital foramen, the hard palate and alveolar process of the maxilla, and the coronoid pro-

other manifestations of scurvy did not have scapulae well enough preserved for examination.

**DISCUSSION**

**Manifestations of scurvy**

Between 1997–2001, Ortner et al. investigated pathological lesions of abnormal porosity of the cortex sometimes, although not consistently, accompanied by new bone formation. Ortner et al. (1999) suggested that pathological lesions represent a vascular response to chronic bleeding attributable to scurvy (see also Jaffe, 1972). The exact mechanism causing the abnormal bleeding is unclear. Chronic bleeding occurs commonly at sites where blood vessels are near skin surfaces or are stressed by muscle activity (Jaffe, 1972). Contraction of muscles is enough to traumatize already defective blood vessels, and causes chronic bleeding (Ortner et al., 2001). Bleeding triggers a vascular response that includes the formation of additional defective blood vessels and increased vascular pathways through bone in situations where the stimulus occurs on or near a bone surface (Ortner et al., 1999, 2001), ultimately resulting in the holes of blood vessels penetrating the underlying cortex (Ortner et al., 1999, p. 322).

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**Fig. 7.** Endocranial vascularity and plaques of new bone formation on occipitals from two young infants, with changes across the skeleton indicative of scurvy.

**Fig. 8.** Sphenoid from young infant with spicules of new bone formation on lesser wings and part of the body, which together with other cranial changes indicate scurvy.
cess of the mandible (Ortner et al., 2001). As Ortner et al. (2001) argued, the most plausible diagnostic option for such a pattern of porous lesions is scurvy. The definition for abnormal porosity as given above, and the locations in which it was identified, were adopted in the current study to identify infants with dry bone manifestations of vitamin C deficiency. These changes are almost certainly linked to bleeding resulting from trauma to blood vessels.

**New bone formation.** Aufderheide and Rodríguez-Martín (1998) presented a detailed account of the soft-tissue manifestations of scurvy, and identified the role that hemorrhage has on the periosteum in both adults and children. In infants and children, bleeding that occurs between the cortex and periosteum may result in the loosely attached periosteum becoming stripped from the underlying bone, activating bone formation (Ortner and Putschar, 1985). Individuals in the past affected by severe scurvy are likely to exhibit lesions apparent on the long bones, manifest as periosteal new bone formation (Ortner et al., 2001; Carli-Thiele, 1996). Where chronic bleeding occurs at the joints of infants, widening of the long bone metaphysis and of the costo-cartilage junctions of the ribs was identified (Jaffe, 1972, p. 449; Aufderheide and Rodríguez-Martín, 1998, p. 311).

Ortner et al. (1999, 2001) noted that abnormal porosity can be accompanied by porous new bone, although this seemingly occurs less often than abnormal porosity alone. Ortner et al. (2001) suggested that manifestations of reactive new bone formation represent a more severe expression of bleeding than abnormal porosity alone, with the probable stripping of the periosteum as the activating factor generating reactive bone formation. The identification of criteria that may enable diagnosis of the less severe and perhaps more frequent occurrences of infantile scurvy in archaeological human bone is therefore important.

**Differential diagnosis of abnormal porosity**

While the porous lesions presented in this paper are thought to be indicative of scurvy rather than of other nutritional diseases or infection, consideration of alternative diagnoses and methods of differentiating between alternative conditions need to be addressed. Because of rapid bone growth, which is normal in infants, the surface of bones can appear porous because of remodeling of the developing bones and the appearance of cutback zones. However, in porous bone related to growth in infants, irregular pits typically do not penetrate through the entire cortex, unlike the porosity resulting from a vascular stimulus.

The pattern of bone changes linked to scurvy and, in particular, their distribution across the skeleton are different from those expected as a result of infectious organisms in most areas of the skeleton. Acute infectious inflammatory diseases, such as osteomyelitis, are relatively common in children. All bones of the skeleton can be involved in such infections, but the lower limbs, particularly the distal femur and proximal tibia, are most frequently affected. Due to the bone structures and the disease process, changes tend to be manifest at the diaphysis, with new bone formation following periosteal detachment (Aufderheide and Rodríguez-Martín, 1998, p. 173). Consideration of the common features of changes linked to infection should help in suggesting a diagnosis, but it may be more difficult to distinguish infections of the scalp from scurvy, especially if cranial bones are viewed in isolation.

Scurvy often occurs with anemia (Stuart-Macadam, 1989), and the possibility that an individual may have suffered from more than one condition (co-morbidity) should always be considered. Anemia can result in thinning or destruction of the compact bone, producing porotic lesions in the cortex, due to the increasing size of the marrow (Stuart-Macadam, 1991). Small, scattered foramina in the orbits occurring in less severe anemia may be difficult to differentiate from the vascular response in the early stages of scurvy. However, more advanced stages of anemic changes, with enlarged or linked trabeculae (Stuart-Macadam, 1991), are more easily differentiated from less severe scurvy in the orbits. In addition, as Ortner et al. (1999, p. 346) argued, porous bone in scurvy does not result in marrow hyperplasia as in anemia.

Porosity occurring with small, spiculated bone formation in the orbits and on the ectocranial surface can occur in rickets (Ortner and Mays, 1998), and may also be difficult to distinguish from infantile scurvy. It remains unlikely that we can confidently identify scurvy from an orbital or a localized ectocranial lesion alone. However, it is emphasized throughout this research that it is the presence of abnormal porosity occurring throughout a number of regions of the cranium and scapulae together that suggests that scurvy was present in these individuals. The orbital lesions of scurvy, whether subtle or more severe, are unlikely to occur without other cranial lesions. As Ortner et al. (1999, p. 330) stated, “the pattern of the lesions is crucial in differential diagnosis.” Therefore, the pattern of pathological change throughout the cranial bones and post-cranial skeleton should help to distinguish scurvy from anemia and rickets. Following previous studies (Ortner and Ericksen, 1997; Ortner et al., 1999, 2001), we suggest that abnormal porosity represents a less severe manifestation of scurvy. Analysis of abnormal porosity in the
skeletal manifestations of infantile scurvy

Clinical comparisons

The identification of vitamin C deficiency scurvy from archaeological remains is difficult, as evidenced by the paucity of cases recognized from post-medieval Britain (Roberts and Cox, 2003). Ortner et al. (2001) suggested that identification of the manifestations of scurvy apparent within documented clinical cases may be of use in helping to establish the range of features that could be recognizable archaeologically. While the examination of clinical specimens is valuable, an inherent problem in the use of such cases is that the majority will be from more severe cases of the disease. Many less severe cases will have gone unrecognized and will not be present in such collections. Melikian and Waldron (2003) studied four specimens from the Royal College of Surgeons known to have had scurvy and used them as known cases against which possible archaeological examples could be compared. All the clinical examples examined in their study had significant areas of new bone formation present, and none of the archaeological material examined resembled these cases (Melikian and Waldron, 2003). However, for the reason discussed above, this was probably not a surprising discovery. It is to be expected that clinically collected cases of scurvy will be more severe, with well-established bone formation. As Ortner et al. (2001) stated, the significance of the identification of porous lesions related to scurvy is that they probably represent a less severe or earlier manifestation of the disease than more advanced proliferative bone formation. The results of Melikian and Waldron (2003) simply underline the importance of developing new diagnostic criteria to identify the disease in varying stages of severity, enabling a better understanding of its probable prevalence in the past.

Historical accounts

All changes described were recorded from well-preserved, relatively complete infants from earth-cut graves from St. Martin’s Church. The completeness of individuals and their position in the stratigraphic matrix indicate that they were probably buried during the final phase of use of the churchyard. The number of burials peaked in 1851 and 1852, when 2,900 and 3,252 burials were performed, respectively, and then dropped considerably until 1863, when use of the churchyard ceased. It is likely that most of the complete individuals excavated were buried after 1840, as intensity of use was such that earlier burials were disturbed and truncated. Research on the documentary evidence from this period indicates that a shortage of potatoes, which have a high vitamin C content, may have affected the poorer individuals buried at St. Martin’s. The potato famine that affected Ireland from 1846–1850 is widely known. However, far less attention has been paid to the impact of potato blight on crops in England. Research on newspapers printed in Birmingham during the 1840s demonstrated that potato crops in the surrounding region were destroyed by the blight. On August 22, 1846, the Birmingham Journal and Commercial Advertiser carried the following report: “we regret to be compelled to say that the result of that examination is a conviction that the potato crop is everywhere diseased, and that, in many places, it is threatened with total destruction.” Newspaper reports from subsequent years indicate that the problem continued. The importance of potatoes in preventing scurvy in the poor was recognized relatively early. For example, Cheadle (1878) noted that “poor children are often saved from scurvy by the common use of potatoes. If potatoes are excluded and only the bread and butter diet given, scurvy sooner or later is exceedingly likely to manifest itself” (cited by Barlow, 1883; see also Poynton, 1935). The impact of potato blight in England has been relatively little studied because, unlike in Ireland, there was a far wider range of grains available, providing alternative sources of energy. However, for poorer individuals, the potato was one of the few affordable sources of vitamin C. Although other forms of vitamin C such as marine fish and citrus fruits would have been available in Birmingham at this time, these foods would not have been as accessible to poorer families.

The lack of secure dating for all burials means that it is not possible to be sure that the cases of scurvy identified are definitely linked to failures in the potato crop. Dates were only available for 38 of the individuals buried in vaults, and of the dated burials, two come from years when crops were affected by potato blight. However, the lack of evidence for scurvy in vault burials indicates that the comparative affluence of these individuals gave them access to foods that would have protected them from scurvy.

CONCLUSIONS

The present study demonstrates that identification of skeletal changes linked to less severe or long-standing cases of infantile scurvy, such as abnormal porosity prominent in the various cranial sites, has an important role in studies of archaeological bone. Many of these changes, as first identified (Ortner and Ericksen, 1997; Ortner and Mays, 1998; Ortner et al., 1999, 2001), when found together in the skeleton, appear to provide good evidence for the occurrence of scurvy. The often fragmentary nature of archaeological bone means that care should be taken to avoid over-reliance on a single skeletal area such as the greater wing of the sphenoid. The findings of the present study also demonstrate that clinical specimens held in historic collections do have limitations, and that developing new diagnostic criteria to identify diseases in various stages of severity is important. Information from historical accounts highlighted the potential of conditions such as scurvy to increase our knowledge of socio-economic differences in past populations. The finding of evidence of scurvy in infants from earthcut graves, but not vaults, backs up the significant socio-economic differences that existed between these individuals.

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LITERATURE CITED


Barlow T. 1883. On cases described as “acute rickets” which are possibly a combination of rickets and scurvy, the scurvy being essential and the rickets variable. Med Chir Trans 66:159–220.


