Fetal paleopathology: an impossible discipline?

Chapter 5
Fetal Paleopathology: An Impossible Discipline?

Mary E. Lewis

This chapter will introduce the concept of fetal paleopathology in archaeological material, highlighting the limitations and potential of such research to inform us about the lives of mothers and their babies in the past. Problems with terminology, aging methods, preservation and recognizing lesions in skeletal remains are discussed, before potential new sources of research are highlighted.

Defining the fetus

In archaeology, "fetus" is used rarely, and describes tiny skeletal remains located within the abdominal area of a female skeleton, or of remains aged under 36 weeks gestation as estimated by the length of their limb bones. Substantiating that a child was buried while still within their mother's womb, however, is problematic in archaeological contexts as this relies on careful observation of the position of the fetal bones under the pelvis of the adult female during excavation (Lewis 2007). The location of the bones is necessary to differentiate a true fetus from a baby placed on the mother's stomach or chest after birth. In bioarchaeology, we more commonly use the term "perinate" which is both useful and necessary as it defines a child in and around the time of birth, generally considered to be between 36-40 gestational weeks in past populations, when they are most likely to have survived naturally (Lewis 2007:2). Although modern medical intervention allows babies to survive at 26 or even 24 weeks, it is impossible to tell whether a child aged under 36 weeks was born alive but prematurely, or if they were stillborn from the skeletal remains alone. Deciding whether a child was stillborn or a victim of infanticide immediately at birth is also problematic. The neonatal line, a defect in the tooth enamel that is laid down just after birth, is often used in forensic cases to suggest a baby was originally live-born. However, it actually forms 3-7 days after birth when the enamel forming cells have had time to recover from the shock (Whittaker and MacDonald 1989).

The perinatal period tends to cover children aged up to 41 weeks thereafter child skeletons tend to be categorized under the 'birth to one-year' age category. Children estimated to be aged up to 44 gestational weeks, by the length of their limb bones or development of the cranium, theoretically dying up to one month after birth, are the focus of this study. This rather generous cut-off point allows consideration of neonatal and post-neonatal factors that may provide indirect information on a child's health. The use of the term perinate underlines the challenges we face with accurate age estimation. Although the dentition has begun to form by the time a child is born, these tiny, fragile dental fragments are rarely recovered archaeologically (Clement and Kósa 1992). Perinatal aging relies on estimating a chronological age from physiological size, based on length measurements of the surviving long bones and other elements (Fazekas and Kósa 1978; Scheuer et al. 1980) and comparing these to standards derived from modern perinates of known gestational age, who we hope share similar growth patterns to the child under study. Understanding where these standards come from, and selecting data based on children of similar ancestry is crucial, but data on perinatal lengths taken from skeletonised remains (dry bone measurements) are

limited. Most measurements are taken from radiographs (X rays) of living children, which may add an element of measurement error due to distortions of the X ray image. Controversies surround which type of statistic bioarchaeologists should use to correlate length to gestational age (Gowland and Chamberlain 2002; Mays 2003), the accuracy of comparing standards based on ultrasound with dry bone length measurements (Christie et al. 1950; Adaline et al. 2001), post-mortem shrinkage (Huxley and Kósa 1999; Warren 1999), the unknown sex and weight of the perinate, and maternal nutritional status (Hauspie et al. 1994; Lampl and Jeanty 2003).

Even if we can be confident we have selected the right methods and standards, if a child was full-term, but born small-for-gestational-age (SGA), then our methods would underage them. While SGA babies have been shown to demonstrate shorter trunk and limb dimensions in comparison to the rest of their body proportions (Brooke et al. 1984), we are unable to measure the trunk in skeletonized material. The degree of error for ages based on long bone lengths can be several weeks either side of an estimated mean age. This, coupled with the limited amount of dental information is a particular frustration when attempting to carry out a paleopathological study. Sherwood et al. (2000) outlined several conditions that could be identified by discrepancies in dental and skeletal age. They studied 160 fetuses between 15 and 43 gestational weeks. Seventy-two were spontaneous abortions with no evidence for pathology, while the rest died with congenital conditions (i.e. trisomy 18 or 21, Turner's syndrome, chromosomal abnormalities, anencephaly, spina bifida cystic), or renal and vascular deformities. Using data from the diaphyseal lengths, spina bifida fetuses were overaged by up to three weeks while, in contrast, those with renal and vascular anomalies were underaged by 3-4 weeks. Prematurity was more common in children with syphilis, pneumonia, heart disease and tuberculosis (Griffith 1919), and while those under 38 weeks were likely to die and be recovered from the archaeological record, a two-week error in their age estimation would hide the cause of their death.

Preservation of perinates provides an additional challenge. Shallow graves or clustering within a cemetery mean perinatal bones are more likely to be disturbed or destroyed, or remain unexcavated. Their extremely fragile nature means that when they are recovered they are often highly fragmented or incomplete. Even when excavated they often end up in boxes of disarticulated material that are rarely studied, or are mistaken for other small mammals (Buckberry 2005). However, some funerary practices such as burying perinates in pots, allow for wonderful preservation (Baker et al. 2005), and Owsley and Jantz (1985) managed a sample of 489 perinates for their fetal growth study. Even the youngest fetuses can be recovered, as witnessed by the tiny radius of a 23-week old found with a young female from Bishopstone in Kent (Schoss and Lewis 2010). Sieving on-site and preserving the soil around the perinate can result in a much greater recovery of bones in the laboratory (Figure 1).

Today, it is estimated that four million babies die worldwide within the first month of life (neonatal period), with the first day being the most critical period, and two thirds dying within a week (Lawn et al. 2005). The most common causes of death are prematurity, sepsis and pneumonia, asphyxia, tetanus and diarrhea. None of these conditions are traditionally recognized in skeletal remains, and of the fourteen percent who died of 'other' causes, only seven percent were from congenital conditions.

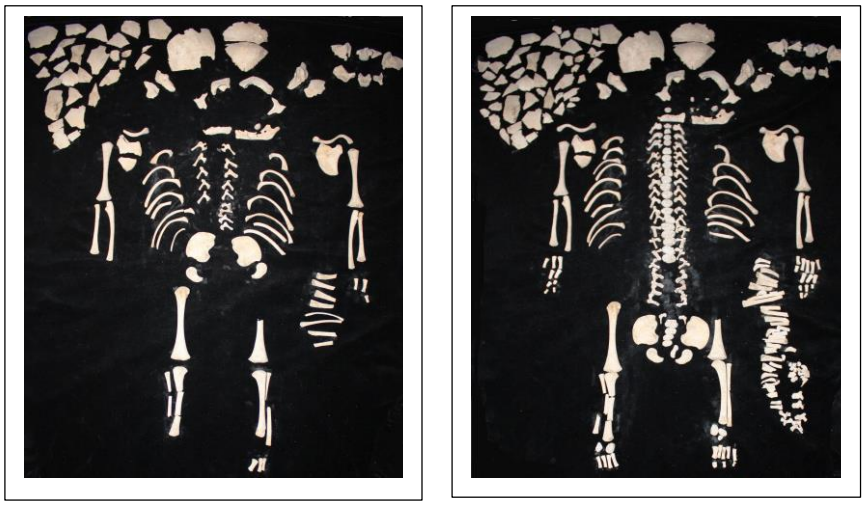

Figure 1: Archaeological perinatal bones from a Romano-British site in England displayed in anatomical position. The figures show bone recovery before (left) and after (right) the sieving of the surrounding soil by an osteologist. The sieved remains are far more numerous and there is greater recovery of the rib and spinal bones, as well as the hand and foot bones that are crucial for the identification of perinatal pathology. (Photograph: Lewis).

These figures relate to death rates in industrialized countries with clear social and economic divides, with children from poor backgrounds most at risk (Lawn et al. 2005). Other factors influencing neonatal mortality include low birth weight, complications during delivery, limited access to specialist care, maternal anemia, syphilis and fever (Black et al. 2010). But we should be cautious about directly transferring these data onto societies of different cultures, levels of development and economies, especially in the past. Clinical studies have consistently identified a higher neonatal mortality rate for males than females, suggesting that females are more buffered against environmental insults in the womb and cope better with the stress of childbirth (Bekedam et al. 2002). While the precise mechanisms behind this phenomenon are still unknown, differential treatment where boys are favored can reverse this survivability pattern (McMillen 1979; Lawn et al. 2005). Application of neonatal and post-neonatal death ratios may be utilized to explore whether children were dying from endogenous or exogenous causes (Lewis and Gowland 2007), but our imprecise age divisions make it a crude way of assessing perinatal causes of death.

Training in non-adult osteology has improved over the last decade, improving an area of research that was previously under-developed. However, the identification of fetal remains and familiarity with their normal anatomy is still limited and may result in many subtle pathological lesions going unnoticed. Given all of these issues it is understandable that paleopathologists rarely choose to study perinates, and a review of their potential to provide information on disease and trauma is long overdue.

Prenatal pathology

The fetus is most vulnerable to environmental disruption during periods of rapid cell differentiation, and in particular, during the first two weeks of development when disruptive agents (teratogens) can result in spontaneous abortion of the embryo (Moore 1988). Skeletal development begins between 8-12 weeks with the formation of the skeletal matrix, followed by intramembranous (in membrane) ossification of the clavicle, mandible and bones of the skull vault (Krakow et al. 2009). Endochondral (in cartilage) mineralization forms the rest of the skeletal structure with the ossification of the appendicular skeleton, ilium and scapula by

16 weeks, metacarpals and tarsals by 16-20 weeks gestation (Krakow et al. 2009). Successful ossification relies on a good maternal oxygen supply through the bloodstream during fetal development (Waldron 2009). The homeobox (Hox) genes regulate the differentiation process of the skeleton into limb or spinal elements, and it is mutations in these genes that are responsible for malformations during embryonic development (Scheuer and Black 2000: 172).

Infections can spread to the fetus via the placenta. The placenta is highly vascular and any breach of its integrity may lead to large amounts of a pathogen reaching the fetus (Zeichner and Plotkin 1988). Pathogens may also spread through the umbilical vein, through ingestion of infected amniotic fluid, or during birth with exposure to an infected birth canal or maternal fluids (Zeichner and Plotkin 1988). The fetus may suffer damage or disruption to their developing cells, or may mount an autoimmune response, but because the fetus has a reduced inflammatory reaction to infection (Holt and Jones 2000), skeletal signs of disease are limited before birth. Rubella, smallpox, tuberculosis, syphilis, leprosy, chickenpox, mumps, measles, and scarlet fever can all be transmitted transplancentally (Lorin 1983; Al-Qattan and Thomson 1995; Naeye and Blanc 1965) and may be evident on the perinatal skeleton, either through signature (pathognomonic) signs, or generalized new bone formation. While exposure to influenza is responsible for many congenital defects, leprosy and syphilis may instead cause early spontaneous abortion or, if transmitted towards the end of the third trimester, remain latent until later infancy and childhood (Dorfman and Glaser 1990, Melsom et al. 1982). While congenital syphilis may result in characteristic notches on the unerupted permanent upper incisors on radiograph (Hutchinson 1857), cranial deformities caused by malaria or maternal smoking (Lampl 2003) are all but impossible to identify on thin and unfused perinatal cranial bones.

Enigmatic skeletal lesions

New bone formation

Traditionally, paleopathologists interpret grey bone deposits (fiber bone) on the outer bone surface as a sign of trauma or infection. For the perinatal paleopathologist, things are less straightforward. De Silva et al. (2003) warned clinicians against misdiagnosing what they termed ‘physiological’ periostitis as abuse in infants. Periostitis is a term used to denote inflammation (or –itis) of the fibrous sheath (periosteum) surrounding the bones in life. They noted symmetrical new bone formation on the long bones especially the femora, humeri and tibiae in babies aged 1-6 months old. While the new bone was concentric in most bones, the tibia was most commonly affected on the medial aspect, and in most cases bone formation was confined to the long bone shaft (or diaphysis). Shopfner (1966) examined the radiographic appearance of the long bones of 335 healthy premature and full-term infants, and noted periosteal new bone in 35 percent of cases. The bone deposits were thick, but not multilayered, and appeared on radiograph as double contours, before they became incorporated into the underlying bone surface. Gleser (1949) warned that increased formation and mineralization of the long bones during the normal growth process may mimic pathological features in a 2-5 month infant, when signs of congenital syphilis, scurvy and rickets may be suspected. For the paleopathologist examining dry bone, perinates with widespread new bone formation on the cranium, long bones and ilia are common findings,

but we are ill-equipped to determine if this indicates one of the many infections that may be responsible for neonatal death, or if this signals the child was experiencing a growth 'spurt' when they died. Common sense may dictate that a child on the brink of death is unlikely to be undergoing rapid growth, but this does not account for accidental deaths or deaths that occur shortly after a growth spurt, but before the bone can remodel. The bone turnover rate in neonates have been estimated to be high before birth and in the first 48 hours of life, and greater in the preterm neonate than full-term babies, with bone turnover rates of infants being several times higher than in adults (Mora et al. 1997). While the extent of bone turnover in perinates may be difficult to quantify, remodeling of the femur for example, significantly changes bone density within the first six months (Rauch and Schoenau 2002). Hence, any new bone formation in perinates will be rapid, with several days needed for the newly deposited organic matrix to be mineralized (Rauch and Schoenau 2002). It would also not be unreasonable to expect any trauma or inflammation experienced during the birth process to be remodeled within six months of the child's life, making identification of lesions in the perinates all the more crucial.

In some cases, localized or profuse new bone deposits that are grey in color, may point to specific conditions. Infantile cortical hyperostosis (or Caffey's disease) is an inflammatory disorder of unknown etiology causing profuse new bone formation on the long bones and mandible that heal spontaneously (Caffey and Silverman 1945). A possible fetal case, found within the abdomen of a female, was suggested by Le Bagousse and Blondiaux (2001) in Lisieux, France. All the surviving long bones have profuse new bone formation, and the tibiae and cranium are most severely affected. Layers of new bone are often identified on the internal cranial surfaces of perinatal material, confined to the occipital. As this part of the cranium is undergoing rapid growth after birth, it seems likely that many of these lesions are part of the normal bone remodeling process (Lewis 2004), but they may also be signs of inflammation or calcified blood pools (hematomas). Griffiths (1919) states that perinatal intracranial hemorrhage occurred in fifteen percent of premature births, and may also result from cerebral palsy. Birth trauma may cause localized bleeding and, if a child is laid on his or her back, then blood or pus would pool to the occipital area (Mitchell 2006). Martin et al. (1997) raised the possibility that the irregular and porotic appearance of the pars basilaris (the bone that forms part of the base of the skull), seen in children of different ages from birth to 6 years, was potentially pathological, calling for more research into this area. Without more detailed information on the pattern and timing of perinatal growth in individual skeletal elements we may never be able to untangle these issues.

Lytic cranial lesions

Kausmally and Ives (2007) highlight problems with the interpretation of destructive (or lytic) lesions in 7.4 percent (7/94) of perinates from post-medieval London. The lesions caused holes in the cranium, but only occurred on a few of the perinates from the same site and context, suggesting post-mortem damage was not to blame. Kausmally and Ives (2007) identified several possible causes including cancer, (e.g., infantile chordroma, infantile myofibromatosis, Langerhans cell histiocytosis (LCH)) and tuberculosis (TB). As the lesions were too common to support very rare cancerous conditions, they favor TB or LCH as a cause (Kausmally and Ives, 2007). Caffey (1978: 32) had previously noted these lesions

calling them ‘lacunar skull’ and describing them as most marked on the parietal and frontal bones. He linked them to spina bifida, hydrocephalus (cranial enlargement due to ‘water-on-the-brain’), meningocele, and meningio-encephocele (protrusion of the brain lining through the cranial vault), but also saw them on the radiographs of apparently health newborns. As the lytic lesions tended to heal spontaneously, Caffey (1978) considered them the result of delayed development of the membrane that eventually forms the cranial vault. Mendonça de Souza and colleagues (2008) noted destructive lesions on the skull of a 6-month-old Peruvian mummy which were accompanied by new bone formation on the internal surface of the cranium, active bone formation on the frontal and parietal bones, and a flattened occipital. They suggest cranial modification through head binding, followed by bone death and secondary infection as the cause.

Potential perinatal paleopathology: Infections

Rubella

Congenital rubella results from transmission of the rubella virus during the first trimester and would have had serious consequences for the survival of a newborn in the past. Those pregnancies ending in a spontaneous abortion or stillbirth would result in perinates entering the skeletal record without pathological changes. Those born alive suffer congestive heart failure, low birth weight and difficulty in feeding, along with deafness and cerebral palsy in the older child (Cooper et al. 1969). Rudolf et al. (1965) report that 45.3 percent of perinates with exposure to maternal rubella display bone lesions between the ages of 1 to 8 weeks and that 76 percent of the 34 perinates are male. Osseous changes include wide radiolucent bands, and ‘beak-like projections’ at the ends of the bone shafts (metaphyses) during healing, coupled with enlarged anterior fontanelles at the areas where the cranial sutures meet (Rudolf et al. 1965: 430). The ability of the rubella virus to inhibit cell multiplication of bone and fiber forming cells (i.e. osteoblasts and fibroblasts) and other tissues in the body is well known (Naeye and Blanc 1965; Reed 1969). These poorly defined zones of calcification are similar to what paleopathologists might see in children with rickets or congenital syphilis. However, once virus excretion has ceased, bone lesions can disappear in several months (Sekeles and Ornoy 1974). While slightly older than perinates, three infants aged between 3-6 months were identified with unusually large fontanelles in South Africa dating to the 20th century, leading Steyn et al. (2002) to suggest rubella as a possible differential diagnosis. Although rubella lesions are rare and transient in the perinatal period, it should be considered as a differential diagnosis in early rickets cases.

Neonatal osteomyelitis

Neonatal osteomyelitis, an abscess forming infection affecting multiple bones, was a common cause of death in the past (Trueta 1959), but has yet to be identified in perinates from archaeological contexts. Clinically, multiple bone and joint involvement occurs in 41 percent and 70 percent of cases respectively (Weissberg et al. 1974), assisted by the presence of open transphyseal vessels that allow the spread of infection across the growth plate (Trueta 1959). Ogden (1979) describes abscess and new sheath (sequestrum) formation in the first few days after birth due to a blood-born spread of infection, and from a localized infection such as a burn. The humerus in the upper arm and the knee are recognized as common sites

of infection, and radiographs of these areas may reveal abscesses inside the bone, while enlarged nutrient foramina may suggest involvement of transphyseal vessels. Sheaths of new bone should be readily identify in the perinatal skeleton, but long bone epiphyses (the growing end plates) are not ossified at birth, and smooth-based localized lesions on the metaphyseal surface caused by abscess may be more difficult to distinguish from post-mortem damage or a normal undulating surface. Caution is needed when using clinical cases to reference diseases from the pre-antibiotic era. Ogden's (1979) neonates had chemotherapy that may have allowed development of chronic lesions where more rapid death would normally occur. How common neonatal osteomyelitis was in the past is difficult to judge as the emergence of penicillin-resistant *Staphylococcus* meant a resurgence of osteomyelitis in a more virulent form and a higher number of neonatal cases in the 1950s to 1970s (Gilmour 1962).

Early onset congenital syphilis

Congenital syphilis develops in the fetus secondary to venereal syphilis in the mother. The causative organism, *Treponema pallidum*, can be transmitted as early as the ninth week of gestation. The pathogen enters the fetal bloodstream and spreads to almost every bone in the body. Toxins released from dead micro-organisms may invoke an allergic response and uterine contractions in the mother, resulting in fetal death and spontaneous abortion (17 percent) in the first half of the pregnancy (Genç and Ledger 2000). These tiny skeletal remains, if they survive into the archaeological record, will show no signs of disease. At term, a child may be stillborn (23 percent), premature, weak and sickly, or between 39-66 percent may appear perfectly healthy (Harman 1917; Hollier and Cox 1998). About 21 percent of the latter will go on to display signs of infection around two years of age ("late congenital syphilis") (Harman 1917). A trio of skeletal lesions occurring together are indicative of congenital syphilis in the perinate: joint disruption (osteochondritis), osteomyelitis and profuse new bone formation (Caffey 1939). Pálfi and colleagues (1992) describe a seven-month fetus recovered from the abdomen of a woman from Costebelle, France. The fetus displayed profuse new bone on the long bones, maxilla, ribs and cranial vault, possible destructive lesions on the parietal bones and a characteristic Wimberger's sign (thick dark band) on a radiograph. Convinced they had a case of congenital syphilis, the authors argued that the mother was in the early stages of syphilis, where clinical evidence suggests nearly all pregnancies will involve the spread of infection to the developing child. More recently, syphilis in two perinates from post-medieval Huelva, Spain (Malgosa et al. 1996) was confirmed by aDNA analysis (Montiel et al. 2012). This is the first time DNA has successfully identified a sub-species of the treponeme and may have been due to the abundance of spirochetes known to invade the bone cells of neonates (Montiel et al. 2012).

Potential perinatal paleopathology: congenital defects and skeletal dysplasia

The prenatal ossification process begins around 8-12 weeks *in utero* and from this point osteologists have the potential to recognize congenital defects in the skeleton. In particular, irregularities in the formation of the bones and replacement of the fetal spinal cord, or notochord, mean that we have the opportunity to identify lesions which may signal more serious soft tissue defects resulting in perinatal death (Figure 2). Anderson (1989) describes

axial congenital anomalies, and a possible familial relationship in three neonates from Homol'Ovi III, Arizona. The first child presented fused thoracic (chest) vertebrae, and asymmetry of the maxilla and mandible with dental overcrowding, suggesting marked facial asymmetry. The second was a perinate with fused second and third cervical (neck) vertebrae and two mid-thoracic vertebra, possibly indicating Type II Klippel-Feil syndrome. The child also had a malformed rib. Finally, a younger perinate also demonstrated fusion (or non-separation) of the spinous processes of the second and third cervical vertebrae and the fourth and fifth thoracic vertebrae. Hinkes (1983) noted flared sternal ends at the front of the rib cage 1-8 in a neonate from the Grasshopper Pueblo and Brothwell and Powers (2000) recorded merged ribs in a neonate from early medieval Lechlade, England. A tiny example of a fused radius and ulna in the arm (radio-ulnar synostosis) has been reported in a perinate from a double burial at El Molon in Spain (Lorrio et al. 2010).

Figure 2. Close-up of the ribs and spine of a 39-week-old perinate from St Oswald's Priory Gloucester, UK. The three central bodies that make up the spine are U-shaped rather than oval indicating the abnormal persistence of the early spinal cord in that area. The ribs corresponding to these bones have fused heads. All of these skeletal malformations signal more serious soft tissue deformities that would have led to the child's death.(Photograph: Lewis).

Cleft, butterfly and block vertebra, and anterior and posterior spina bifida all have the potential to be identified in the perinate (Müller et al. 1986), and the presence of extra cervical ribs may reveal information about the potential cause of death. In a study of 318 perinates born in Utah between 2006-9, Furtado et al. (2011) reported a significantly higher prevalence of cervical ribs in stillborns compared to live-born children who died within the first year (43 percent compared to 12 percent). They conclude that cervical ribs signal a disadvantageous fetal environment that leads to a greater likelihood of stillbirth with similar results presented elsewhere (Bots et al. 2011). This would require identification and careful examination of the neural arches for the 7th cervical vertebrae for facets. Although, Black and Scheuer (1997) note that cervical ribs themselves will not be found in children under the age of 10 years, as they do not fully develop until fusion of the posterior arch to the body of the vertebrae.

There are over 350 forms of skeletal dysplasia that all have the potential to be identified in perinatal remains. Differentiating between them is problematic clinically so is likely to be impossible in archaeological circumstances, although some features such as

glabella bossing at the top of the nose, flattened nasal bridge, vertebral (centrum) morphology, and poor mineralization of the cranium and skeleton may signal dysplasia (Krakow et al. 2009). Osteofibrous dysplasia of the neonate, while infrequent, may present as an expansile lytic lesion at the midshaft of a single tibia causing bulging of the bone surface macroscopically and potentially, a pathological fracture (Hindman et al. 1996). Mooney et al. (1992) presented a series of fetal facial measurements that can be used to identify cleft lip and palate, defects that are hard to recognize in the tiny unfused perinatal maxillae. These include the premaxillary length and nasal opening length, which can potentially be identified on dry bone, but techniques to test and apply this to archaeological material have yet to be developed. Although there is potential for more research to date, congenital skeletal defects are the most common form of pathology identified in archaeological perinates.

Sjøvold et al. (1974) identified a full-term fetus with multiple bony projections (osteochondromas) from St. Clements, Visby in Gotland, found within the abdominal area of a 17-20-year-old female with the same condition. The deformities may have caused an obstruction leading to the death of the mother and child. Bennett (1967) describes a case of multiple cranial suture fusion in a perinate from Utah that would have led to a clover-leaf deformity if the child had lived. Cope (2008) suggests the presence of a meningocele in the fragile cranial bones of a neonate from the Dakhleh Oasis, and a perinate with an even more severe cranial malformation (holoprosencephaly) (Cagigao 2011) buried in an urn in Palpa, Peru. The burial was normal except for the presence of another apparently normal, neonate. Although the rest of the skeleton is not described by Cagigao (2011), segmental errors in the spine are often associated with this cranial deformity (O'Rahilly et al. 1980; 1983).

The oldest archaeological case of a perinate with anencephaly (a fatal condition where the brain and the bones of the cranial vault fail to form) comes from an Egyptian catacomb in Hermopolis, built to house the mummies of sacred monkeys and ibises (Saint-Hillaire 1826). Dudar (2010) discusses a possible case of anencephaly in a child from Elmbank Pioneer cemetery in 19th century Toronto, with associated fused ribs. Detailed studies of the perinates from the Dakhleh Oasis, Egypt have revealed a variety of remarkable cranial malformations (encephalocele, iniencephaly and anencephly) (Cope 2008, Mathews 2005). Mathews (2005) provides a useful description of known anencephly in perinates housed at the Smithsonian Institution, Washington, DC. Deformities included malformed sphenoid lesser and greater wings, lack of frontal and parietal bones with isolated orbital rims, deformed squama of the temporal bones and early fusion of the elements with an elliptical rather than round tympanic ring (a small ring of bone that forms at the external opening of the ear) (Mathews 2005).

East and Buikstra (2001) discuss an achondroplastic female from Elizabeth Mounds, Tennessee with an *in utero* fetus they also suspected had achondroplasia based on metaphyseal flaring, disproportionately short limbs, and cranial and long bone measurements that were outside the normal range of the 46 perinates they compared it too. This case has not been fully published and the authors caution other dysplasias have yet to be ruled out. Keith (1913) provides a very useful comparison of the individual bones of an achondroplastic child against an unaffected child of the same age. The affected child displayed a reduced foramen magnum, absent suture mendosa on the developing occipital, premature fusion of the basi-occipital suture and short and broad wing of the pars lateralis. In the full-term child, the basi-

occiput and pre-sphenoid fused limiting further expansion of the brain. Frontal bossing was evident due to brain seeking compensatory space.

Down Syndrome

Trisomy 21 is the most common chromosomal abnormality among live-born infants and is related to increasing maternal age (e.g. 35 years). Today, Downs is estimated to occur in 1 in 700 to 1 in 1000 births in the USA (Benacerraf 1996). The vast array of skeletal and dental features associated with this syndrome has always attracted the attention of paleopathologists, and while five suspected cases of Downs have been identified, none of these are perinates. This may be because children with the syndrome tend to survive into older childhood, but given mothers of increasing age are more likely to suffer from complications at birth, we might expect some cases to enter our perinatal sample. Hook (1979) estimated 21 percent of trisomy 21 children died between mid-gestation and full-term. Sonographs of second trimester fetuses reveal some skeletal features used to predict the presence of trisomy 21 at birth. These include a shortened femur and humerus when measured against the maximum width of the skull and in comparison to the population norm, a shortened iliac length, reduced limb bone length in comparison to the axial skeleton, and shortening of the middle phalanx of the fifth digit of the hand (Keeling et al. 1997; Stempfle et al. 1999). Unfortunately, these features either require an intact cadaver, or a large enough series of well-preserved perinates to gauge the normal dimensions. Although hand phalanges ossify around 24 weeks gestation, they are so tiny they are rarely excavated making measurement or an assessment of agenesis impossible. In addition, the short and stumpy fifth finger phalanx, if recovered, may be misidentified as belonging to the foot. Clinically, the accuracy of these measurements in identifying Down syndrome is between 40-50 percent only rising to 74 percent when the soft tissue nuchal fold of the neck is included (Benacerraf 1996).

Nevertheless, some features hold promise. Absence of the nasal bone is commonly reported in Down syndrome neonates (Dedick and Caffey 1953, Otaño et al. 2002, Stempfle et al. 1999, Keeling et al. 1997), and should ossify between 15-40 weeks gestation. Otaño and colleagues (2002) found absent nasal bones in three of five (60 percent) of Down syndrome fetuses compared to only 0.6 percent of non-Down syndrome cases. A related condition, trisomy 13 can result in malformations in the lumbrosacral and thoracic spine, where small and irregular sphenoid bones have been noted (Kjaer et al. 1997). A fetus with triplody, a rare and lethal condition where there are three sets of chromosomes, can present with cranial base malformations including extra ossification centres and fusion of two or more vertebral bodies, or a disproportion in the size of the cervical bodies. Another chromosomal disorder (aneuploidy) is a possible explanation for the cranial deformations identified in a 38-40-week perinate from Andover Road in Winchester, UK. The wing of the pars lateralis is bipartite, and the posterior condyle canal is in two halves (Figure 3). The thickened and regular edges suggest this was a congenital anomaly rather than a basiliar linear fracture of the occipital (Falys 2010).

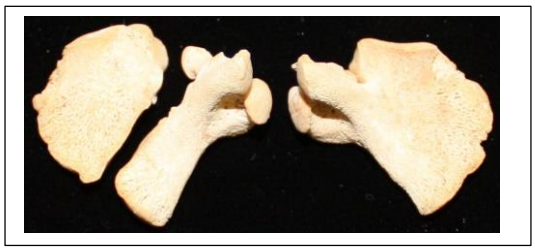

Figure 3. Left and right pars lateralis bones that form part of the occipital in a 38-40 week perinate from Andover Road in Winchester, UK. The unfused 'wing' of the bone on the left is a feature often seen in cases of children born with chromosomal disorders. (Photograph: Falys).

Osteogenesis imperfecta

Osteogenesis imperfecta (or 'brittle bone disease', a fatal congenital condition leading to multiple fractures), was suggested as a possible diagnosis for a 38-week old perinate from Dakhleh Oasis, Egypt, with severe bowing of the all surviving long bones and pathological fractures of the left ulna, femur and tibia (Cope and Dupras 2011). The baby was buried on its side in contrast to the normal supine extended burials of other children in the cemetery. Although the basilar fragments of the skull were preserved, they did not show the malformations (for example a triangular pars basiliaris) identified in a forensic case from Guatemala (Lewis 2007: 107).

Potential perinatal paleopathology: trauma

Perimortem cut marks on neonatal remains have been interpreted as indicating surgical removal (or an embryotomy). Only three cases have so far been identified; two are Romano-British examples from Poundbury Camp in Dorset and Yewden villa, Buckinghamshire. The Poundbury perinate was decapitated and has extensive cut-marks throughout the long bones (Molleson and Cox 1988; Mays et al. 2012). A possible 19th century embryotomy was identified in L'Aquila, Italy (Capasso et al. 2014) on the basis of severe jumbling of the bones in a wrapped and mummified 29-week-old fetus.

Birth Injuries

Perinates who die shortly after birth are too young for us to assess any paralysis that may occur as the result of trauma during childbirth, but some fractures may be evident. In 1950s New York, six percent of all neonatal hospital admissions of newborns were for injuries sustained at birth (Montagu 1950). Dedrick and Caffey (1953) reported fractured clavicles in 1.2 percent of their 1030 newborns; these were always unilateral and occurred more commonly on the left side. Caffey (1978) describes hematomas (cepalhematomas) on the ectocranial surface as the result of bruising during breech birth usually positioned away from midline sutures, a detail that may aid in their differentiation from meningocele. Ossified hematomas may persist for months or years. Skull fractures as a result of birth injury present more of a challenge due to the fragile and fragmentary nature of the perinatal cranium. Linear and depressed fractures may not survive fragmentation in the ground, or if the child dies shortly after birth, perimortem fractures may be indistinguishable from post-mortem breaks. Caffey (1978) also suggests that the maxilla is a frequent site for infection in the first few weeks of life due to birth trauma, and may be visible as reactive new bone formation around

the developing dental germs. Three published cases of possible birth injuries have been identified in paleopathology, involving a skull fracture in a 38-week perinate (Baxarias et al. 2010) and two cases of unilateral clavicular fractures (Soren et al. 1995; Brickley et al. 2006).

Conclusions

This paper has reviewed the challenges and potential of examining perinatal remains from archaeological contexts in order to identify skeletal pathology. Perinatal skeletons (c.26-42 weeks) are often difficult to identify and excavate, with sieving essential to ensure all of the bony elements are recovered. It is impossible for us to age perinates precisely enough to identify any dental to skeletal discrepancies that may indicate death due to renal or vascular failures, or assess the level of premature or SGA births. Although it is likely that the majority of perinates died from infectious or innate conditions, identifying pathology on the basis of new bone on sub-periosteal and endocranial surfaces is problematic as we have yet to develop criteria that allow us to distinguish pathological lesions from the normal growth process. Nevertheless, there has been increasing success in the identification of congenital malformations from archaeological contexts, with 15 of the 29 (52 percent) of the reported perinatal cases describing congenital disorders. While fetal remains recovered from the pelvic cavities of female graves hint at obstetric hazards, individual perinatal burials have the potential to tell us much about the health of the fertile maternal population, as well as the environmental factors that affect the survival of newborns. A review of the clinical literature has allowed for the identification of skeletal features that may help us recognize new conditions in the perinate, including rubella, Down syndrome and other chromosomal disorders, and birth trauma and cleft palate, but further research is needed to understand the range of pathology that can be identified on these tiny remains. Appreciating how far we still have to go in our analyses will help us set the agenda for future studies.

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