



# Human osteology method statement

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## **Introduction**

*Brian Connell*

The Museum of London has been excavating human skeletal remains in the Greater London area since the mid 1970s and has accumulated an impressive archive of over 17,000 individuals. This material has been the focus of much research but, inevitably, different researchers employed different methods and recorded different data depending on any given set of research objectives.

The issue of standards in data recording was first addressed head-on in the United States where the need for standards was heightened by the increased repatriation of skeletal remains (Rose et al 1991; Buikstra and Ubelaker 1994). Recent work in the United Kingdom has been increasingly concerned with tackling issues of standards, guidelines and methods, from the preparation of MAP2 assessment and publication reports through to recommendations for data recording standards (Mays et al 2002; Brickley and McKinley 2004).

This generic method statement for analytical work carried out across the Museum of London group incorporates the recommendations of many of the current guidelines. In the past, the Museum of London utilised many different recording methods and procedures. These were first standardised into a single recording system designed by Conheaney (1997) and aimed at defining standard quantitative methods for each skeleton within a cemetery sample.

In 2003, the Museum of London group embarked on the recording of human remains on an almost unprecedented scale. The Centre for Human Bioarchaeology was created in response to the initiation of the Wellcome Osteological Research Database project (WORD), re-examining remains excavated over the preceding two decades. At the same time, the Spitalfields Market Project, involving the recording of over 5000 individuals, began. A specialist recording form was developed for these projects using the pre-existing Oracle inter-relational database. The Oracle forms, combined with the benefits of new published standards and guidelines, prompted a redesign of the methods and procedures employed in the recording of human remains (Connell and Rauxloh 2003). The principal aim was to standardise the recording of all metric and morphological variability expressed in the human skeleton. The system was designed to be applicable to any skeleton regardless of age at death.

This document represents a further advance, providing details of the references used to determine age and sex, palaeopathological diagnostic criteria, morphological landmarks and so forth. As such, it supersedes all previous method statements and should be considered standard for all material recorded by the Museum of London group from 2003, unless otherwise stated: site specific variations from this method statement will be discussed in the relevant reports.

# 1 Preservation and archaeological data

*Brian Connell*

Every skeleton recorded is identified by the archaeological site code and context number throughout. Photographs (digital record shots and publication photographs) and radiographic plates taken in support of differential diagnosis are given a reference number following standard archive procedures (Museum of London in prep). Such data is cross-referenced with the context number to enable association of the record with the original skeleton.

The general state of bone preservation is visually assessed using a three-point grading system (Table 1). Where the degree of preservation is variable throughout the skeleton, the predominant grade is assigned.

Grade	Description
1	bone surface in <b>good</b> condition with no erosion; fine surface detail such as coarse woven bone deposition would (if present) be clearly visible to the naked eye
2	bone surface in <b>moderate</b> condition; some post-mortem erosion on long bone shafts. Erosion of articular surfaces and some prominences
3	one surface in <b>poor</b> condition; extensive post-mortem erosion resulting in pitted cortical surfaces; articular surfaces missing or severely eroded

*Table 1 Skeletal preservation grading system*

Archaeological information of interpretive importance is noted. If a skeleton is truncated this is recorded to enable a distinction to be made from those skeletons where elements are absent due to poor preservation or recovery.

Occasionally intrusive bone from the general cemetery soil may be included with the primary burial, as may animal bone. The presence of intrusive bone is indicated with a brief description of the intrusive material. Detailed recording of intrusive human bone is not carried out unless specified by the site method statement or where it is considered to have funerary significance.

When a skeleton comes from an area of a cemetery with a high burial density it is not unusual to find intrusive human bones from other individuals. In some cases it is not possible to distinguish between intrusive and non-intrusive bones, due to similar morphology, preservation and size of the elements: this is particularly true of the small elements of the hands and feet. In instances where separation was not possible, the mixed elements from the primary burial and intrusive individual are not fully recorded, but the location of the mixing is noted (eg mixed hands) and referred to as 'elements artificially deleted'. This enables all further analyses to be conducted using only those elements where the integrity of the individual is certain.

The location and details of any staining present on the skeleton is recorded. If the causative agent can be determined, this is also noted – eg green staining from a copper-alloy object.

Where biographical data is available for any individual, most usually in the form of a coffin plate, this is also noted.

Subsamples (bones, bone segments or teeth) removed from the skeleton for any analytical purpose are noted by location, together with the date the samples were taken, purpose and name or location of the researcher in question.

## 2 Catalogue of completeness

*Brian Connell*

Each anatomical element, or component thereof, is recorded on a presence or absence basis. For the purposes of this recording protocol at least 50% of a bone or bone segment must be present before it is counted as such.

### 2.1 Cranial elements

The smaller cranial bones (ethmoid, lacrimal, palatine, vomer and turbinates) are excluded from the catalogue. The selected components reflect those elements most likely to be recovered from infants and young children, which will also allow an estimate of the proportion of the adult skull present to be made. The sphenoid is recorded as five separate elements: the basisphenoid or body; and for both right and left sides, the greater wing; and the lesser wing. The occipital bone is recorded relative to its four primary centres: the basioccipital (*pars basilaris*); the right and left exoccipitals (*pars lateralis*); and the squamous portion. The hyoid bone is catalogued in three parts: the two greater horns and the body. As no attempt is made to remove auditory ossicles, only the presence of loose ear ossicles is noted; the bones of the ear are not systematically recorded.

### 2.2 Post-cranial elements

The sternum is recorded in six sections: the manubrium; the four sternbrae of the mesosternum or body; and the xiphoid process. Although all rib fragments are thoroughly examined for evidence of pathology, only the heads of ribs, the portion of the ribs with the costo-vertebral and costo-transverse articular facets, are catalogued by side, where known. The vertebrae (from cervical to coccygeal) are catalogued by their constituent parts and divided into three groups: complete vertebra with both the centrum and neural arch; centrum only; and neural arch only.

The pelvic girdle is divided into each of its component elements: the ilium; ischium; and pubis. The scapula is divided into four parts: the glenoid fossa; the coracoid process (or unfused coracoid bone in a subadult); the acromion; and the infraspinous portion of the blade. The clavicle is recorded in three parts: the medial (or sternal) third; the middle third; and the lateral (or acromial) third.

For each of the long bones (humerus, radius and ulna, femur, tibia, fibula) the diaphysis (shaft) is subdivided into three equal parts: proximal; middle; and distal. The epiphyses and joint surfaces are catalogued separately.

The patellae, carpals, tarsals, metacarpals and metatarsals are recorded. The phalanges of the hands and feet are divided into proximal, intermediate and distal groups for the right and left limbs; as confidently assigning a side to individual phalanges is not always possible, un-sided elements are also noted. Hallucial sesamoids are likewise counted as right, left or un-sided.

### 2.3 Cartilage

The presence of ossified thyroid, cricoid, costal and other cartilage is noted. Underlying pathological causes of the ossification are recorded as outlined in section 8.

### 2.4 Dentition

The *Fédération Dentaire Internationale* (FDI) system (1971) of tooth identification is used to allow the use of a unique numeric identifier for each tooth (Hillson 1996, 8).

The dental inventory consists of a count of all teeth and tooth positions available for examination, to permit the calculation of the true prevalence of dental pathology. A set of numeric codes enable the osteologist quickly to record presence or absence (Table 2). Post-mortem loss is indicated by a sharp-edged socket with no indications of healing, ante-mortem loss by the full or partial healing of the empty socket. Congenital absence is established by the surface morphology of the bone where eruption would have been anticipated. Radiographic examination is not undertaken in such cases unless otherwise stated.

Code	Description
1	tooth present
2	post-mortem loss
3	ante-mortem loss
4	congenital absence
5	tooth present (no socket observable)
6	tooth erupting
7	deciduous retention
Null	area absent

*Table 2 Dental catalogue*

### 3 Age at death estimation

Natasha Powers

#### 3.1 Subadult age at death

For all ageing methods requiring metric data, measurements are taken in millimetres (mm).

Foetal and neonatal remains are aged using linear regression equations for diaphyseal length as cited in Scheuer and Black 2000 (after Scheuer et al 1980). Measurements are taken of all complete long bones and the average age estimate expressed in weeks.

Diaphyseal length data from Maresh 1970 is used to estimate the age at death of subadults over the age of two months. In addition to long bone length, the maximum dimensions of the ilium and the basioccipital are recorded to enable age estimation (Buikstra and Ubelaker 1994, 45–6; Scheuer and Mac Laughlin-Black 1994; Scheuer and Black 2000). The resulting age estimates are pooled with that derived from the long bones and an average age at death estimate expressed in weeks or years as appropriate.

Dental eruption data from Gustafson and Koch (1974) is also utilised. The Moorees, Fanning and Hunt (MFH) code (1963a; 1963b) is noted for all teeth where the stage of crown or root completeness was visible: loose teeth or those which can easily be removed from the socket to allow observation. The tooth position is recorded using the FDI (1971) method and coded following the system set out by Buikstra and Ubelaker (1994; 50). Age estimation is based on the average age attained from both male and female data.

Epiphyseal fusion data (Connell and Rauxloh 2003, with reference to Scheuer and Black 2000) are consulted (Table 3). The state of fusion in all observable epiphyses is visually quantified as fused, fusing or unfused as defined in Buikstra and Ubelaker (1994, 41). This is compared with tabulated data and the average or best fit of this range recorded as the overall age for this method.

Epiphyseal fusion		Timing	
Element	location	♂	♀
Skull	mandibular synchondrosis		<12 months
	spheno-occipital synchondrosis	13–18 years	11–16 years
Vertebrae	neurocentral synchondroses		3–4 years
	thoracic neurocentral synchondroses		3–4 years
	lumbar neurocentral synchondroses		2–4 years
Clavicle	medial epiphysis*		16–21 years
Scapula	coracoid process		15–17 years
	acromion		18–20 years
Humerus	proximal epiphysis ( <i>composite</i> )	16–20 years	13–17 years
	distal epiphysis ( <i>composite</i> )	12–17 years	11–15 years

	medial epicondyle	14–16 years	13–15 years
Radius	proximal epiphysis	14–17 years	11.5–13 years
	distal epiphysis	16–20 years	14–17 years
Ulna	proximal epiphysis	13–16 years	12–14 years
	distal epiphysis	17–20 years	15–17 years
Metacarpals	proximal epiphysis (base) MCP 1	16.5 years	14–14.5 years
	distal epiphyses (heads) MCP 2–5	16.5 years	14.5–15 years
Os coax	ischio-pubic ramus		5–8 years
	iliac crest		20–23 years
	ischial epiphysis		20–23 years
Femur	proximal epiphysis ( <i>head</i> )	14–19 years	12–16 years
	greater trochanter	16–18 years	14–16 years
	lesser trochanter		16–17 years
	distal epiphysis	16–20 yrs	14–18 years
Tibia	proximal epiphysis ( <i>plateau</i> )	15–19 yrs	13–17 years
	distal epiphysis	15–18 years	14–16 years
Fibula	proximal epiphysis	15–20 years	12–17 years
	distal epiphysis	15–18 years	12–15 years
Calcaneum	epiphysis	18–20 years	15–16 years
Metatarsals	proximal epiphysis (base) MTS 1	16–18 years	13–15 years
	distal epiphyses (heads) MTS 2–5	14–16 years	11–13 years

\* Fusion of the sternal clavicle was defined when the epiphyseal ‘flake’ adhered to the centre of the joint surface (O’Connell 2004, 19).

### Table 3 Epiphyseal fusion data

Liversidge (1994) indicated that dental development data was the most reliable method of age estimation. In light of this, where contradictory results were obtained from the different aging methods, dental data were given priority.

For older subadults (1 month and older) age attained using each method of estimation is expressed in years and months (as fractions of years in decimals). Age estimation data are combined to produce an overall age estimate in weeks or years, as appropriate; these are grouped as shown in Table 4. All individuals under the age of 18 years at death are referred to collectively as subadults.

Description	Age range
Inter-uterine/neonate	<4 weeks
Early post-natal infant	1–6 months
Later post-natal infant	7–11 months
Early child	1–5 years

Later child	6–11 years
Adolescent	12–17 years
Subadult	<18 years

*Table 4 Subadult age groups*

### 3.2 Adult age at death

Adult ages are estimated from a combination of pubic symphysis degeneration (Brooks and Suchey 1990; Buikstra and Ubelaker 1994, 24–32), auricular surface degeneration (ibid; Lovejoy et al 1985), sternal rib morphology (Iskan et al 1984; Iskan et al 1985) and dental attrition data (Brothwell 1981, 72).

Descriptive text and diagrams are referred to when establishing the phase of change present in the joints of the pelvis. Dental attrition data is only recorded where a complete quadrant of mandibular molars is present. It is considered the least reliable of the ageing methods and the other ageing methods are referred to in preference when establishing an overall age estimate.

In fragmentary or incomplete remains, adulthood is defined by the complete fusion of the epiphyses, with the exception of the late fusing centres of the *os coxa* and medial clavicle. The complete eruption of the third permanent molars is also used to signify adulthood. The late fusing epiphyses are used to enable estimation of young adulthood.

As many different ageing methods are used as the preservation of the skeleton allows and a ‘best fit’ approach taken when placing an individual into an age category. If the relevant skeletal area was unobservable, it is recorded as such.

It is recognised that difficulties exist in ascribing age estimates to adult remains, particularly older adults; therefore, broad age groups are used for analysis (Table 5).

<b>Description</b>	<b>Age range</b>
Young adult	18–25 years
Early middle adult	26–35 years
Later middle adult	36–45 years
Mature adult	≥46 years
Adult	>18 years
Subadult	<18 years

*Table 5 Adult age groups*

## 4 Sex determination

*Jelena Bekvalac*

Sex is estimated for adult individuals only. The methods employed are based on a macroscopic assessment of selected features of the skull (including the mandible) and the pelvis. Where metric data is also utilised (section 5.4; Bass 1987) it is stated in the site specific methods.

A total of 14 sexually dimorphic features are assessed (Table 6).

Skeletal element	Morphological feature	Reference
Skull	supraorbital ridges	Brothwell 1981
	inion protuberance	
	nuchal crest	
	mastoid processes	Bass 1987, 82
	slope of forehead	
	zygoma root	Ferembach et al 1980
Mandible	gonions	Brothwell 1981
Pelvis	ventral arc	Phenice 1969
	medial portion of pubis	
	greater sciatic notch	Bass 1987, 203–05
	preauricular sulcus	
	subpubic angle	
	subpubic concavity	
	median ischiopubic ridge	

*Table 6 Cranial and pelvic features used to determine adult sex*

When observation is possible, each of the selected features is graded using a five-point scale. Where an element could not be observed a sex code of ‘9’ was assigned. An overall estimate of sex is derived from a combination of the data from the individual features with more weight given to the significance of the pelvis than of the cranial features. Overall sex is also assigned on the five-point scale with ‘9’ signifying an adult of undetermined sex (Table 7).

Grade	Sex
1	male
2	probable male
3	intermediate
4	probable female
5	female
9	element not observable/undetermined sex

*Table 7 Estimation of adult sex*

## 5 Ancestry

This code should be used to record the results of CRANID or FORDISC analyses. However, it can also be used to record skeletal and dental morphology that is considered to be suggestive of an individual's ancestry. The determination of ancestry is based upon the forensic anthropology guidelines published by Byers (2005), which rely upon the macroscopic assessment of discernible skull differences between White, Black and Asian ancestry groups. It is recognised that the characteristics employed to assess an individual's ancestry is subjective and influenced by inter-observer error (see Elliot and Collard 2009; Ramsthaler et al, 2007; Ubelaker et al, 2002).

In the absence of CRANID or FORDISC analyses, the following data should be recorded, where available:

- Metric analysis: maximum length and breadth, minimal frontal breadth, bizygomatic breadth, cranial base length, basion-prosthion length, nasal height and breadth, orbital height and breadth, chords (frontal, parietal and occipital bones), internal palatal length and breadth (see chapter 6)
- Dental morphology: in addition to the variations recorded usually on the database, the presence of features such as talon cusps, large molar cusp size (Irish and Nelson 2008) should be recorded
- Craniofacial morphology: shape of the ascending ramus (wide/narrow, oblique/vertical); palatine suture (straight/jagged/arched); zygomatic tubercle (presence/absence); incisor rotation (present/absent); sagittal arch (low and sloping); nasal profile (concavo-convex/straight); nasal aperture (medium/wide/narrow); nasal bones (low/high, arched/flat); nasal sill (sharp/absent); nasion (depressed/absent); post-bregmatic depression (present/absent); zygomaticomaxillary suture (angled/curved)

## 6 Metric data

*Richard Mikulski*

The stated references (Table 8; Table 9; Table 10) should be consulted for the equipment used. All measurements are taken in millimetres or degrees, as appropriate.

### 6.1 Cranial measurements

Where preservation and completeness permit, a maximum of 37 measurements are taken from the adult skull (31 single and 3 paired left and right) and a further 12 measurements on the mandible (Table 8, Table 9). Skulls exhibiting post-mortem deformity are not measured. Where pathological deformation of the cranium has occurred, metric data are recorded on a separate paper record, for ease of separation from the main metric dataset.

Measurement	Abbreviation	Landmarks	Reference
Porion-bregma height	PBH (left/right)	po-b	Bass 1987, 70
Total facial height	TFH	n-gn	
Palatal breadth (end-end)	G'2	-	Bass 1987, 78
External length	ext-L	-	
Maximum length	L	g-op	Buikstra and Ubelaker 1994, 74
Maximum breadth	B	eu-eu	
Basi-bregmatic height	H'	ba-b	
Bizygomatic breadth	J	zy-zy	
Basi-nasal length	LB	ba-n	
Basi-alveolar length	GL	ba-pr	
Nasal height	NH'	n-ns	
Nasal breadth	NB	al-al	
Upper facial height	G'H	n-pr	
External breadth	ext-B	Ecm-ecm	
Upper facial breadth	fmt-fmt	Fmt-fmt	
Palatal length	G'1	st-o	
Bi-dacryonic chord	DC	d-d	Buikstra and Ubelaker 1994, 76
Frontal chord	S1'	n-b	
Parietal chord	S2'	b-l	
Occipital chord	S3'	l-o	
Orbital length	O'2 (left/right)	-	
Orbital breadth	O'1 (left/right)	d-ec	
Bi-orbital breadth	ec-ec	ec-ec	
Foraminal breadth	FB	-	Buikstra and Ubelaker 1994, 77
Foraminal length	FL	ba-o	

Bimaxillary breadth	GB	-	Brothwell 1981, 82
Biasterionic breadth	Bias B	as-as	Brothwell 1981, 83
Frontal arc	S1	-	
Simotic chord	SC	-	
Parietal arc	S2	-	
Occipital arc	S3	-	
Transverse biporial arc	BQ'	po-po	Brothwell 1981, 83
Bi-dacryonic arc	DA	da-da	
Maximum circumference	U	-	

*Table 8 Cranial metrics*

Measurement	Abbreviation	Landmarks	Reference
Bicondylar width	W1	cdl-cdl	Buikstra and Ubelaker 1994, 78
Symphyseal height	H1	id-gn	
Bigonial breadth	GoGo	go-go	
Mandibular angle	M<	-	
Minimum ramus breadth	RB' (left/right)	-	
Ramus height	RH (left/right)	-	
Biforamen breadth	ZZ	-	Brothwell 1981, 83
Maximum length	ML	-	
Coronoid height	CrH (left/right)	-	

*Table 9 Mandibular metrics*

## 6.2 Calculation of cranial indices

Cranial indices are calculated according to Bass (1987, 69) and head shapes are referred to as dolichocranic, mesocranic, brachyranic and hyperbrachyranic.

## 6.3 Dental measurements

Dental measurements are recorded using sliding callipers on the maxillary and mandibular canines and first molars (both permanent and deciduous). The mesiodistal and buccolingual crown diameters are recorded. In the canine teeth these are defined as the maximum diameter of the crown relative to the tooth long axis, not the crown long axis (Buikstra and Ubelaker 1994, 62). In molar teeth, the mesiodistal crown diameter is taken between interproximal contact points. The buccolingual diameter is taken perpendicular to the mesiodistal plane. When possible, loose teeth are extracted to enable mesiodistal measurements to be acquired. If pathological change, extreme attrition, post-mortem damage or the proximity of adjacent teeth obscured or compromised the crown at the point of measurement, metric analysis is not attempted.

## 6.4 Post-cranial measurements

A maximum of 29 paired measurements are recorded for the adult post-cranial skeleton and three single measurements of the sacrum (Table 10). Long bone length is measured for intact elements or those with a single close fitting break only. Measurements of the calcaneum are not taken where there is marked enthesopathy development at the heel. Glenoid breadth is defined as the maximum width of the articular surface and radial head diameter as the maximum diameter. The maximum length of the talus is measured from the posterior margin of the talo-crural articular surface to the anterior aspect of the talar head.

Element	Measurement	Abbreviation	Reference
Clavicle	length	CLL1	Buikstra and Ubelaker 1994, 79
Scapula	glenoid length	GLL	Bass 1987, 123
	glenoid breadth	GLB	(see text)
Humerus	length	HuL1	Buikstra and Ubelaker 1994, 80
	head vertical diameter	HuHD	
	shaft maximum diameter	HuD1	
	shaft minimum diameter	HuD2	
	mid shaft circumference	HuC	
	epicondylar width	HuE1	
Radius	length	RaL	Buikstra and Ubelaker 1994, 80
	head maximum diameter	RaHD	(see text)
Ulna	length	ULL1	Buikstra and Ubelaker 1994, 81
Sacrum	length	SaL	Buikstra and Ubelaker 1994, 81
	breadth	SaB	
	S1 body width	BW	
Femur	maximum length	FeL1	Buikstra and Ubelaker 1994, 82
	oblique length	FeL2	Bass 1987, 219
	head vertical diameter	FeHD	Buikstra and Ubelaker 1994, 82
	ant-post proximal diameter	FeD1	
	med-lat proximal diameter	FeD2	
	ant-post mid-shaft diameter	FeD3	
	med-lat mid-shaft diameter	FeD4	Buikstra and Ubelaker 1994, 83
	mid shaft circumference	FeC	
	bicondylar width	FeE	
Tibia	length	TiL1	Bass 1987, 234
	length (medial)	TiL2	Buikstra and Ubelaker 1994, 83
	ant-post diameter at foramen	TiD1	
	med-lat diameter at foramen	TiD2	
	epicondylar breadth	TiE	

Fibula	length	FiL1	Buikstra and Ubelaker 1994, 84
Calcaneus	length	CaL	Buikstra and Ubelaker 1994, 84
Talus	length	TaL	(see text)

*Table 10 Post-cranial metrics*

## **6.5 Stature**

Adult stature is calculated for adult males (codes 1 and 2) and (females (codes 4 and 5) based on maximum length of the right femora and using the regression formulae for ‘white’ males or females derived by Trotter (1970).

## **6.6 Subadult skeletal measurements**

The maximum lengths of the long bone diaphyses of the limbs are recorded, using sliding callipers or an osteometric box as appropriate, where the element is intact or contains a single, close fitting break. The maximum length of the clavicle and epicondylar width of the distal humerus and femur are also recorded (Buikstra and Ubelaker 1994, 46). The width and height of the ilium (ibid) and the width and depth of the greater sciatic notch are noted (Mays 1998, 39). Measurement of the length and breadth of the basioccipital follows Buikstra and Ubelaker (1994, 45), and Scheuer and MacLaughlin-Black (1994).

## 7 Non-metric skeletal traits

*Bill White*

The non-metric traits considered to be of primary interest in population studies are summarised in Buikstra and Ubelaker (1994, 85–94) and in Brothwell and Zakrezewski (2004, 27–33), and refer to Berry and Berry (1976) and Finnegan (1978) in particular.

A suite of 39 cranial variables, six mandibular and 47 other post-cranial traits was selected, based upon the recognised standards, but selecting only those traits that previously showed significant prevalence in the initial examination of the skeletal material from St Mary Spital (Connell 2002) (Table 11; Table 12; Table 13; Table 14). Each trait was noted as present, absent or unobservable.

<b>Mid-line traits</b>	<b>Reference</b>
Metopism	Buikstra and Ubelaker 1994, 87–9
Lambdoid bone	
Inca bone	
Bregmatic bone	
<b>Bilateral traits</b>	
Asterionic bone	Buikstra and Ubelaker 1994, 87–9
Epipteric bone	
Sagittal wormian bones	
Coronal wormian bones	
Lambdoid wormian bones	
Squamo-parietal wormian bones	Brothwell 1981, 94
Parietal notch bone	Buikstra and Ubelaker 1994, 87–9
Torus auditivus	Buikstra and Ubelaker 1994, 91
Torus maxillaris	Brothwell and Zakrezewski 2004, 28
Torus palatinus	

*Table 11 Cranial sutural variation*

<b>Bilateral traits</b>	<b>Reference</b>
Supraorbital foramen	Buikstra and Ubelaker 1994, 87
Accessory infraorbital foramen	
Supraorbital groove	Buikstra and Ubelaker 1994, 93
Mastoid foramen	Buikstra and Ubelaker 1994, 91
Foramen of Huschke	Buikstra and Ubelaker 1994, 90
Parietal foramen	Buikstra and Ubelaker 1994, 88
Posterior condylar canal	Buikstra and Ubelaker 1994, 89

*Table 12 Cranial foraminal variation*

<b>Bilateral traits</b>	<b>Reference</b>
Multiple mental foramina	Buikstra and Ubelaker 1994, 91
Torus mandibularis	
Mylohyoid bridge	

*Table 13 Mandibular variation*

<b>Trait</b>	<b>Reference</b>
<b><i>Mid-line traits</i></b>	
Sternal foramen	Barnes 1994, 223
Manubrio-corpae synostosis	Barnes 1994, 213
<b><i>Bilateral traits</i></b>	
Os acromiale	Buikstra and Ubelaker 1994, 94
Acromial articular facet	
Septal aperture	Buikstra and Ubelaker 1994, 92
Supracondylar process	Brothwell 1981, 99
Atlas: posterior bridge	Buikstra and Ubelaker 1994, 92
Atlas: lateral bridge	
Atlas: transverse foramen bipartite	
Atlas: double facet	Brothwell 1981, 98
Accessory sacral/iliac facets	Buikstra and Ubelaker 1994, 94
Acetabular crease	Brothwell 1981, 99
Third trochanter	Buikstra and Ubelaker 1994, 94
Allen's fossa	
Hypotrochanteric fossa	Brothwell 1981, 98
Patella: Vastus notch	Buikstra and Ubelaker 1994, 94
Patella: bipartite	Aufderheide and Rodríguez-Martín 1998, 74
Tibia: squatting facet medial	Buikstra and Ubelaker 1994, 94
Tibia: squatting facet lateral	
Calcaneal facet absent	
Calcaneal facet double	
Talus: Os trigonum	Brothwell 1981, 98
Talus: talar facet double	
Talus: squatting facet	Buikstra and Ubelaker 1994, 94

*Table 14 Post-cranial variation*

## 8 Dental pathology

*Tania Kausmally*

Five main aspects of dental pathology are recorded (caries, calculus, enamel hypoplasia, periodontitis and periapical lesions). All pathologies are recorded to an individual tooth level stating location and advancement of the condition (Connell and Rauxloh 2003). To allow accurate calculation of true prevalence rates, it is noted when a particular pathology is unobservable by virtue of being obscured by another pathological trait or by dental wear, eg a gross calculus deposit obscuring the observation of enamel hypoplasia.

### 8.1 Caries

Dental caries is defined as destruction of the enamel, dentine and cement (resulting from acid production by bacteria in dental plaque) manifesting as a cavity in the crown or root surface (Hillson 1996, 269). Caries is recorded at individual tooth level noting the position and severity of the largest carious lesion visible. Location is divided into seven categories (Table 15), based on a recording system by outlined by Buikstra and Ubelaker (1994, 55).

Location of caries
Occlusal
Lingual
Buccal
Mesial
Distal
Gross (site of origin no longer identifiable)
Root surface

*Table 15 Location of caries*

The severity is expressed in four categories with root caries recorded using the three latter categories only (Table 16).

Severity of caries
Enamel destruction only
Destruction of dentine without exposure of pulp chamber
Destruction of dentine with pulp chamber exposed
Cross destruction (crown largely destroyed)

*Table 16 Severity of caries*

### 8.2 Calculus

Calculus is noted where mineralized plaque can be seen adhering to the tooth surface (Hillson 1996, 255). Calculus is recorded on an individual tooth level stating the location

and severity of the formation. The location is recorded as supra- or sub-gingival based on the location of the deposit (on the crown or the root) and the characteristics of the calculus (ibid, 257). The severity is recorded as slight, medium or considerable deposition following Brothwell (1981, 155).

### 8.3 Enamel hypoplasia

Diagnosis of hypoplastic defects refers to Hillson (1996, 167) for description of linear and pit-shaped interruption in the enamel formation. Enamel hypoplasia is recorded on individual tooth level. Each tooth crown is divided into three sections (cusp, middle and lower). Location was recorded according to the most severe defect. Three grades indicate the severity of linear hypoplasia, defects following the trend of the perikymata (Table 17). Unusual pit formation defects presenting as circular defects on the crown surface are also noted (Hillson 1996, 172).

Severity of linear hypoplasia	Definition
Just discernable	can be seen but not felt with a fingernail
Clear groove on the tooth surface	clearly felt with the fingernail
Gross defect (ridges/dentine exposed)	clear brown ridges on enamel surface

*Table 17 Enamel hypoplasia*

### 8.4 Periodontitis

Gingivitis (inflammation of the gum) will, if left untreated, develop into periodontitis causing loss of alveolar bone (Regezi et al 2000, 144). Periodontal disease is recorded on an individual tooth level, visually estimating the distance between the cemento-enamel junction (CEJ) on the buccal and labial surfaces and the alveolar bone and only when there was no damage to the latter. This method is not considered to be flawless owing to the nature of dental eruption, attrition and development during life and these factors should be taken into consideration prior to analysis (Hillson 1996, 263). Recording is based on Brothwell (1981, 155) noting three stages (grade 1: 2–3mm; grade 2: 3–5mm; grade 3: >5mm (majority of root exposed)).

### 8.5 Periapical lesions

Periapical lesions, most commonly developed from periapical granuloma by the accumulation of pus, are diagnosed following (Hillson 1996, 285). Lesions are recorded at the parent tooth position according to the location of the largest sinus drainage (external, internal or maxillary sinus). The underlying aetiology of the lesion is noted, where possible, as dental caries or dentine exposure resulting from advanced wear.

## **8.6 Dental anomalies**

### *Lynne Cowal*

Permanent and deciduous dentitions are examined for the presence of a number of the more commonly observed dental anomalies, coded on a presence/absence basis unless otherwise stated.

### ***Rotation***

Dental rotation is identified when a tooth has rotated from normal anatomical position. The degree of rotation was estimated from the buccal surface of the tooth, categorised in 45 degree increments in a mesial or distal direction, and subdivided by those changes occurring in a single tooth and bilateral winging (Hillson 1996, 112).

### ***Crowding***

Crowding is noted as present when a permanent tooth (or teeth) is located in an abnormal position to the rest of the dental arcade (misaligned) due to a decrease in the dental space for that tooth. The abnormality is recorded relative to the tooth mesial to the relevant position.

### ***Impaction***

The identification of impaction follows Hillson (1996). Impaction of a tooth in its most correct form implies that a tooth remains inside the maxilla or mandible and does not emerge into the mouth at all; however, many variations occur and a tooth may erupt sideways partially exposing one of its crown surfaces (ibid, 113). Impaction in the archaeological material is observed macroscopically where there is partial exposure of the tooth crown surface, or if post mortem damage to the maxilla or mandible exposes the impacted crown.

### ***Transposition***

Transposition is identified where two neighbouring teeth swap normal position (Hillson 1996, 113). The FDI (1971) code of the position to which each tooth had moved was recorded.

### ***Enamel pearl***

The presence of enamel pearls (small nodules of enamel formed on the tooth root: Hillson 1996, 98) is recorded according to tooth position.

### ***Peg molar***

Peg-shaped teeth, appearing as a single rounded cusp, are noted as present or absent for maxillary molars. Peg deformities in other teeth (Hillson 1996, 19) are separately documented as a general dental pathology.

### ***Incisor shovelling***

Shovelling is defined by the presence of pronounced marginal ridges, resulting in a deep lingual fossa and is recorded by presence or absence only when observed on the

permanent maxillary incisors (Hillson 1996, 87). No differentiation between grades of shovelling is made.

***Carabelli cusp***

The presence or absence of Carabelli cusps (Hillson 1996, 91), of all types is recorded for the maxillary permanent molars only, with no further details of size or type recorded.

***Protostylid cusps***

Protostylid cusps on the buccal crown below the mesiobuccal cusp are recorded for the mandibular permanent molars only, with no further details of size recorded (Hillson 1996, 97).

## **9 Skeletal pathology**

### **9.1 Introduction**

*Natasha Powers*

The following section outlines the criteria referred to when diagnosing the main classes of disease observed in the skeleton. Individual recording requirements are outlined by pathology where applicable. Some conditions are noted on a presence/absence basis and this is stated in the relevant section. All other pathological lesions are recorded via a written description on an Oracle database, supported by digital photographs and drawings as required. Where appropriate, radiographs of the skeletal elements are taken to ascertain a definitive diagnosis. In all cases, lesions are macroscopically examined, the size of any discreet lesions is measured and the location on the bone surface is described using standard anatomical terms. The step-by-step procedure in diagnosis was followed (Roberts and Connell 2004, 34).

Paper records supplement the database where particularly significant or unusual pathological changes necessitate. All suspected cases of specific infection are fully documented with digital record photographs and a paper recording form. All pathological lesions are digitally photographed in those collections destined for reburial. Differential diagnosis of all pathology is considered and noted when appropriate.

In some cases, identification of specific diseases is hampered, if not prohibited, by incomplete or poorly preserved remains, although a fragmentary nature does not necessarily preclude diagnosis.

All measurements are taken in millimetres or degrees as appropriate.

The crude prevalence is calculated by individual for different demographic and/or stratigraphic groups as appropriate. Where appropriate, true prevalence is calculated by element (or segment) affected. The dataset used is stated wherever statistical figures are used in the analytical reports produced.

### **9.2 Congenital and developmental abnormalities**

*Rebecca Redfern*

The diagnosis of congenital and developmental abnormalities in the axial or appendicular skeleton is dependent upon skeletal preservation and completeness. Congenital abnormalities are defined as those observable at birth, and developmental those which become apparent during skeletal maturation (Barnes 1994, 2).

#### ***Skull malformation***

When an individual displays a congenital malformation of the skull which cannot be specifically diagnosed, due to taphonomic damage or because it falls outside the following criteria, it is classed as a 'general skull malformation'. Such cases are drawn or photographed and cranial measurements are provided on a paper recording form.

### *Sutural agenesis*

This is defined following Barnes (1994) and diagnosed by the presence of the features summarised in Table 18, where absence of the suture and/or an unusual cranial shape is observed macroscopically.

<b>Suture(s) affected</b>	<b>Condition</b>	<b>Diagnostic features</b>
Sagittal	scapholcephaly (complete agenesis)	long, narrow skull with a median ridge in adults
Sagittal and lambdoid	scaphocephaly (complete agenesis)	long, narrow low skull with prominent occipital region
	plagiocephaly (partial agenesis)	asymmetrical posterior skull
Coronal suture	plagiocephaly (partial, unilateral agenesis)	asymmetrically shaped frontal
	brachycephaly (complete, bilateral agenesis)	rounded cranium
Coronal and lambdoid	oxycephaly	increased cranial height and width, producing 'pointed tower' skull
Coronal and sagittal	Crouzon's syndrome	frontal bossing with steep forehead, short calvarium and flat occipital
Lambdoidal	plagiocephaly (partial, unilateral agenesis)	asymmetrical rhomboid shape
	complete bilateral agenesis	high, flat occiput; external indented suture markings with internal bony build-up for suture markings
Squamosal	(partial or complete)	may alter dimensions of cranial width
Metopic	trigonocephaly (complete agenesis)	triangular, pointed forehead (Barnes 1994, 152–7, fig 4.9)

*Table 18 Sutural agenesis*

### *Bathrocephaly*

This variation of posterior sagittal synostosis, is characterised by the development of a 'podium' in the occipital region. Diagnosis is based on the posterior portion of the parietal sloping inferiorly and the occipital bone protruding superiorly (Jane et al 2000).

### *Anencephaly*

This failure of the posterior embryonic neural tube to develop the cranial vault is diagnosed using the descriptions of Aufderheide and Rodríguez-Martín (1998, 55–6).

### *Microcephaly*

Microcephaly is defined as failure of brain development that results in a statistically significant subnormal skull circumference resulting in cranial capacity of less than 1000cc (Barnes 1994, 160). Diagnosis follows metric assessment and is reliant upon the following features (Aufderheide and Rodríguez-Martín 1998, 56):

***head circumference less than 46cm;***

***recession*** of the frontal and parietal bones;

***flattening*** of the occipital bone;

***prominent nose;***

***receding mental protuberance;***

***early synostosis*** of all or most of the cranial sutures and bregmatic fontanel;

***large face*** in comparison to the head;

the skull has a ***conoidal shape***.

### *Hydrocephalus*

Following hydrocephalus the skull becomes large and globular, with frontal bossing. Where possible, attempts are made to distinguish between communicating and non-communicating hydrocephalus following the criteria published in Richards and Anton (1991, 195). Diagnosis is reliant upon the following features (Aufderheide and Rodríguez-Martín 1998, 57–8):

***enlargement*** of the head;

***thinning*** of the skull bones;

***bulging fontanelles;***

***widely separated sutures***, often showing wormian bones;

***atrophy*** of the supraorbital ridges;

***flattening*** of the cranial base.

### *Mandibular abnormalities*

Developmental delays in the mandible can cause disruption in union and growth. Where a cleft mandible is noted this is categorised as full separation, partial cleft or diastema (Barnes 1994, 162, fig 4.13).

Where possible hypoplasia or aplasia (*hemifacial microsomia*) of the mandible is diagnosed by type (Table 19) following Barnes (1994, 161–3, fig 4.14).

Type	Description
I	mild hypoplasia of one or both sides of the mandible and temporo-mandibular joints; abnormal occlusion
II	small and abnormally shaped mandibular ramus, with an underlying temporo-mandibular joint; the mandibular arch is narrow and non-occluding on the affected side
III	aplasia of one half of the mandibular angle; the ramus and temporo-mandibular joint are absent

*Table 19 Mandibular hypoplasia*

Bifid condyles are noted where two distinct articular facets separated by a depression or groove are present. Condylar hypoplasia is represented by a larger than normal condyle, confirmed by measurement of the maximum dimensions and could be unilateral or bilateral. Coronoid hyperplasia is noted where one or both coronoid processes was elongated, reaching the posterior aspects of the zygomatic bones (Barnes 1994, 165–9, figs 4.16, 4.18).

#### *External auditory meatus abnormalities*

Where external auditory meatus defects are observed they are subdivided as unilateral or bilateral and partial (hypoplasia of ectodermal groove) or complete atresia (aplasia of ectodermal groove) (Barnes 1994, 199, fig 4.35).

#### *Cleft palate*

Defined as a disruption in the development of the palatal processes, which leads to hypoplasia or aplasia of one or both sides resulting in abnormal fusion, variation in cleft palate type is described as unilateral or bilateral and by type (dorsal notch, partial or full cleft with the vomer not attached) according to Barnes (1994, 171–4, fig 4.21).

Changes secondary to cleft lip are considered likely if there is a small unilateral or mid-line fissure, for example between the canine and lateral incisor, or a complete cleft in either location, associated with agenesis of the right central incisor (Barnes 1994, 184–6, fig 4.27). The presence of a deviated nasal septum, disruption of nasal floor or disturbance of the lateral incisor(s) are noted (ibid 196–7).

#### *Craniofacial abnormalities*

Facial clefts, disruptions of the mid face, are subdivided according to Barnes (1994, 182):

***nasomaxillary facial cleft*** – non-union of the maxillary prominence and lateral nasal prominence, from eye orbit to oral cavity;

***naso-ocular facial cleft*** – non-union between lateral nasal prominence and medial nasal prominence along nasal groove, from eye orbit to oral cavity;

***median facial cleft*** – aplasia (hypotelorism) or hypoplasia (hypertelorism) of one or both parts, or mild hypoplasia observed by widely spaced orbits and nares, and a broad nasal area. The nasal bones are attached at unusual angles with notching of the nasal alae.

Where observation was possible macroscopically, the frontal sinuses were atypical/absent. The vomer and perpendicular plates may be hypoplastic; premaxillary alveolus is notched or cleft. May be associated with oxycephaly.

Hypoplastic defects of the median nasal prominence are considered where a flat glabella, short and fat nose, absence of the anterior nasal spine, hypoplasia of the frontal sinuses, crescent-shaped nares and small pre-maxilla resulting in a prognathic mandible are observed. Nasal and lacrimal hypoplasia are defined as unilateral or bilateral and subdivided by hypoplasia, severe hypoplasia or aplasia (Barnes 1994, 192–7, fig 4.31, 4.32).

Ossification of the stylohyoid chain (Reichert's cartilage) is subdivided as: that affecting the entire chain; complete but separate ossification of each element; or as complete ossification and unification to the hyoid (Barnes 1994, 206, fig 4.38).

A variety of developmental fissural cysts, resulting from delay in the retraction of the overlying ectodermal tissue, are considered and defined by element affected as follows (Barnes 1994, 175–80):

***median anterior maxillary cyst*** – indicated by a rounded or oval lesion(s) in or near incisive canal, typically at the midline;

***median palatal cyst*** – indicated by a rounded or oval lesion(s) in or near incisive canal, typically at the midline and usually opposite the molar-retromolar region;

***globulomaxillary cyst*** – indicated by a rounded, oval, or irregular lesion, usually between lateral incisor and canine, extending into alveolar crest at junction of the premaxilla and maxilla.

### ***Spinal disorders and 'border shifting'***

Many spinal disorders are considered to be observable only in adult individuals, due to the completion of ossification or the influence of trauma or function stress (Barnes 1994, 35).

#### ***Scoliosis***

The term scoliosis is considered to indicate lateral curvature of the spine with rotation of the vertebrae and the spinous processes towards the concavity of the curvature (Aufderheide and Rodríguez-Martín 1998, 66). Vertebral border shifting associated conditions (eg osteochondrodystrophy: Aufderheide and Rodríguez-Martín 1998, 66), secondary changes and skeletal adaptations, such as robusticity or atrophy, are also noted. Diagnosis is reliant upon the following features (ibid, 67):

***lateral wedging*** of the vertebral body at the apex of the curve;

***asymmetry*** of the neural arch and vertebral processes;

***horizontal torsion*** of the vertebral body and spinous processes showing deviation from the median plane;

***variation in the morphology of the transverse processes***, in the thoracic vertebrae they are deflected backward in the convexity and forward in the concavity; in the lumbar spine they are short and plump on the convexity, and long, slender and pointed on the concavity

***sclerosis in the concavity*** of the spongiosa architecture on the wedged side of the vertebrae;

***reduction in the size and number of trabeculae*** on the convexity on the wedged side of the vertebrae.

Where possible the cause of scoliosis is diagnosed using the features outlined in (Table 20).

Cause	Subdivisions and description	Reference
Hemimetamere shifts	solitary shift, unilateral double shift	Barnes 1994, 60–62, figs 3.10, 3.13
Hemimetamere hypoplasia-aplasia	solitary aplasia, unilateral multiple hypoplasia, unilateral multiple hypoplasia with coalescence into post-lateral bar, severe multiple hypoplasia with coalescence into post-lateral bar	
Infantile idiopathic scoliosis	(onset 0–3 years) – thoracic region affected, typically the left side; where the condition persists curvatures up to 100° can occur	Aufderheide and Rodríguez-Martín 1998, 66–7
Juvenile idiopathic scoliosis	(onset 4–9 years) – thoracolumbar region involved, curvature is typically located in the right side	
Adolescent idiopathic scoliosis	(>10 yrs) – the curvature is typically located in the right side	

*Table 20 Scoliosis*

Post-paralytic scoliosis is considered as a diagnosis when unilateral vertebral rotation affecting one or two vertebrae or segmented vertebral rotation involving most vertebrae of the same spinal segment are observed, principally in the thoracolumbar region, and where associated pathology or additional skeletal adaptations, such as robusticity or atrophy are present (Aufderheide and Rodríguez-Martín 1998, 67). Secondary changes as the result of developmental hip dislocation are also considered (below).

### *Kyphosis*

Kyphosis is diagnosed when a pathological increase in the normal curvature of the thoracic spine results in the abnormal forward bending of the spine (Ortner 2003, 463). Differentiation from secondary kyphosis and Scheuermann’s disease follows (Ortner 2003, 463–6). ‘Senile’ kyphosis is considered in older adults (>50 yrs), where degeneration and attrition of the intervertebral disks, particularly the anterior portions, was observed. Diagnosis requires the apex of the curvature to be located in the upper thoracic spine and wedging of the vertebral bodies to be absent or only slight. Osteosclerosis of the anterior portions, particularly the endplates, anterior fusion of several vertebrae, and anterior marginal lipping arising directly from the vertebral end plate are also diagnostic features (Ortner 2003, 464–5).

### *Border shifting*

The presence of a variety of cranial-caudal border shifts is noted as present or absent at five levels of the vertebral column, according to the diagnostic features outlined (Table 21; Table 22; Table 23; Table 24; Table 25). Only complex defects, or combinations of defects, were fully described and photographed.

<b>Shift</b>	<b>Description</b>	<b>Reference</b>
Expression of occipital vertebra	condylar facets converge caudally; posterior and/or anterior arches incomplete; bony processes distorting the shape of the foramen magnum may also be present	Barnes 1994, 82–8, figs 3.22, 3.25
Precondylar process	a blunt rounded bony extension from the inferior aspect of the anterior rim of the foramen magnum	
Bipartite occipital condyles	two distinct and separate condyles are present	
Transverse basilar cleft	transverse cleft along one or both sides of the basioccipital; normally incompletely formed, as narrow slits with smooth edges, 3–5mm in length	
Atlas occipitalised	atlas is partially/completely incorporated into the base of the occipital	
Paracondylar processes (right/left/bilateral)	broad base, a conical shape, can be quite large	
Hypoplasia of condylar facets	condyles are abnormally small	
Precondylar facets	forms on the rim of the foramen magnum of the occipital, when the odontoid fails to descend and protrudes on/into the foramen magnum	
Odontoid process displacement	dens presents as a separate element or ossicles which may attach to the anterior rim of the foramen magnum, or agenesis of the dens or odontoid; no differentiation made between types (i–v )	

*Table 21 Occipitocervical border shifts*

<b>Shift</b>	<b>Description</b>	<b>Reference</b>
Expression of a cervical rib: bony tubercle	a small tubercle that does not extend beyond the normal lateral dimensions of the transverse processes	Barnes 1994, 100–04, fig 3.31
Expression of a cervical rib: blunt bony projection	a bony extension 40–50mm in length	
Expression of a cervical rib: rib extension without costal joint	either articulating with the first rib, or attaching to the sternum via a ligamentous band	
Complete cervical rib	a separate rib, that may or may not articulate/fuse with the first rib	
Stunted transverse process on C7	transverse process is reduced in size, transverse foramina can remain	
Rudimentary first rib	first thoracic ribs are underdeveloped, usually less than 30mm in size; abnormal in shape, wider than cervical ribs, may articulate/fuse to the second rib near the scalene tubercle	
Second rib attaches to mesosternum only	cartilage shifts caudally and attaches to the side of the mesosternum, observed by a shift in costal articulation	

*Table 22 Cervicothoracic border shifts*

Shift	Description	Reference
Hypoplastic rib 12	slender, obliquely tapered small rib; transitional apophyseal facets typically shift to the 11th thoracic vertebral segment	Barnes 1994, 104, 105, 109, fig 3.34
Transitional facets on T11	see above	
Aplastic rib 12	no rib facets present on T12	
Lumbar rib and facet on L1	transitional apophyseal facets present to L1, accompanied by ribs, usually bilateral, from tubercle-like articulating processes, to 'true' articulating ribs up to 70mm in length; associated with well-formed costal joints with the transverse processes of L1	
Small blunt rib on L1	a small blunt separate projection is present on the transverse process of L1	
Transitional facets on L1	costal fovea are present on the centrum of L1	
Supernumery vertebra	T13: only recorded when the complete thoracic and lumbar vertebral column was present identified by the characteristics of the thoracic vertebrae	Barnes 1994, 78

*Table 23 Thoracolumbar border shifts*

Shift	Description	Reference
Supernumery vertebra	L6: only recorded when the complete thoracic and lumbar vertebral column is present; the supernumerary vertebra has the characteristics of the lumbar vertebrae and must not show any indication of sacralisation	Barnes 1994, 78
Apophyseal joints between S1–S2	rudimentary apophyseal joints present	Barnes 1994, 110, fig 3.36
Anterior cleft between S1–S2	cleft of any thickness is present between S1 and S2	
Apophyseal joints and anterior cleft S1–S2	as above	

*Table 24 Lumbosacral border shifts*

Sacralisation and lumbarisation are subdivided according to whether bilateral or unilateral (and if so which side was affected) and which vertebra is involved. Ala-like transverse processes of fifth or sixth lumbar vertebrae are noted as articulating or non-articulating, incomplete lumbarisation of the first sacral vertebra noted by side only. Complete sacralisation is defined as the assimilation of the fifth or sixth lumbar vertebra into the sacrum and complete lumbarisation where the first sacral vertebra is completely independent from the sacrum and has taken on the appearance of a lumbar vertebra but the alae remain (Barnes 1994, 108, fig 3.36).

Shift	Description	Reference
Incomplete separation of S5	lateral circumferences of the foramen formed between the last two sacral segments are incomplete or absent	Barnes 1994, 114, fig 3.39
Complete separation of S5	last sacral segment separated from the sacrum	
Incomplete sacralisation of the first caudal vertebra	only the body may join, or only one foramen may be created	
Complete sacralisation of the first caudal vertebra	body and alae are completely joined to the sacrum, extra foramen are created	

*Table 25 Sacrocaudal border shifts*

### *Segmentation failures (congenital fusion) and developmental errors*

A range of segmentation failures are considered and noted as present or absent at each vertebral level. As for vertebral border shifts, only complex combinations are fully described: Klippel-Feil syndrome is diagnosed according to Barnes (1994, 67–71) and recorded by a full written description and supporting photographs. Fusion is categorised as a cranial or caudal segmentation failure with complete ossification or with discrete lines of partial demarcation present. Partial or completely united centra or neural arches resulting in block vertebrae are noted (Barnes 1994, 67). A cleft neural arch is defined when there is a wide posterior separation, resulting from hypoplasia or aplasia of one or both parts of the precursors of the pedicles, laminae, or spinous process. A thin separation is noted as a bifurcate neural arch (ibid, 117, 119). Hypoplasia of the transverse processes or centrum was noted according to the side affected (ibid, 126, fig 3.45).

### ***Limb abnormalities***

#### *Achondroplasia*

This condition is suspected where there is severe shortening of the extremities but the diameter of the diaphyses and the cortical thickness are close to normal. The epiphyses and metaphyses are usually disproportionately wide relative to the length of the bones. Diagnosis is based upon the following features (Apley and Solomon 2000, 63; Aufderheide and Rodríguez-Martín 1998, 358–60; Ortner 2003, 482–9):

***brachycephalic skull***, which is also large and bulbous;

***shortened cranial base***;

***saddle shaped nose***;

***all limbs are shortened***;

***shortened fingers***;

***thickened long bones***;

increased ***lumbar lordosis***;

***adult stature rarely exceeding 140cm***.

Additional features such as lower thoracic kyphosis, equal length fingers with tapering terminal phalanges or a severely narrowed pelvis are also considered (Ortner 2003, 484).

#### *Limb aplasia, hypoplasia and malformation*

Malformation is considered to be any variation from normal physical structure, due to congenital or developmental defects (Martin 2000, 388). Limb deformities are described and broadly classified as aplasia (complete failure of development), or hypoplasia (under-development) according to Barnes (1994). Phocomelia was diagnosed if the extremities have less than the normal number of rays, and synostosis where there is congenital fusion of the limbs (Ortner 2003, 474).

#### *Developmental dislocation of the hip*

Diagnosis of developmental dislocation follows Mitchell and Redfern (2007). Primary signs, additional musculoskeletal anomalies (spinal malformations, clubfoot, and limb growth anomalies) and secondary joint changes are described by element affected. The formation of a 'true' or 'false' acetabulum is defined as follows.

**True acetabulum** – triangular or oval in shape, shallow, and smaller than a normal acetabulum. The base of the triangle lies toward the obturator foramen and the apex is directed posterosuperior. The superoinferior dimensions are larger than the anteroposterior. It is aligned roughly parallel with the lateral surface of the ilium, with a minimal posterior or superior wall. The walls may be perpendicular to the floor or overhang the cavity with lipping at the margins. The floor is irregular and shows no sign of the smooth articulating surface present in a normal acetabulum. Often a pattern due to ossification of soft tissues is visible on the floor: parallel lines or a chequered pattern.

**False acetabulum** – located superior or posterosuperior to the true acetabulum (the mean distance between the centre of the true and false acetabulum is 4.5cm (range 3.5–6cm)). When bilateral, the location is more posterior than unilateral cases. Some false acetabula are round, others are oval or crescent shaped with their long axis in the anteroposterior plane.

Four types of bony change are noted at the false acetabulum:

- 1) shallow, smooth depression, with some pitting
- 2) a fine layer of patchy bone
- 3) a raised bony plaque on the iliac cortex, with a smooth or gently concave surface
- 4) a deep rounded cup more reminiscent of a normal acetabulum

The femoral head ceases to be large and round, and does not show signs of a vertical groove compatible with *ligamentum teres*. Type 1, 2 and 3 false acetabulum are associated with small, flattened, or mushroom-shaped femoral heads, while the type 4 acetabulum is associated with heads of near normal proportions, which appear round when viewed in the coronal plane but are oval in the anteroposterior plane.

The tibial condyles on the affected side are smaller, the mid-shaft circumference reduced and tibial length shorter by a mean of 1cm (range 0.5–1.5cm).

Developmental asymmetry in the lumbar spine may be associated with hip dislocation and cause scoliosis, with lumbar concavity on the opposite side to the affected hip.

#### *Hip dysplasia (subluxation)*

This is defined when the femoral head is incompletely seated in the acetabulum. Two types of change were categorised as unilateral or bilateral:

- 1) the joint is unstable and the rim of the acetabulum may show slight deformity
- 2) the acetabulum is very shallow, the roof provides incomplete coverage, and the margin is short resulting in subluxation

In type 2 changes the acetabulum has an oblique appearance when viewed anteriorly. Additional observations are made of secondary osteoarthritis and changes to the femoral head (Resnick 2002, 4356. fig 79–1; Herring 2002, 523).

#### *Congenital talipes equinovarus*

Where articulated elements of the foot are seen to point downwards with an inverted heel and twisted forefoot congenital talipes equinovarus is diagnosed (Martin 2000, 133–4). Specific diagnostic features are as follows (Aufderheide and Rodríguez-Martín 1998, 75–6; Herring 2002, 924):

***alteration of the tibial distal articular surface***, especially the medial malleolus;

***talar malformation***, absence of the true neck and absent, fused or malformed anterior and medial facets of the subtalar joint;

***intraosseous deformity*** of the calcaneus, navicular and cuboid;

cuboid can form a ***pseudo-heel***;

***hypertrophy*** of the medial tuberosity of the navicular;

***calcaneal malformation***, small in size, the sustentaculum tali is underdeveloped, and anterior articular surface is medially deviated.

All secondary changes to the remaining foot elements, eg flattening, abnormal curvature or displacement, are also recorded.

The talus is used as the point of reference when describing foot deformities which were classified according to the direction of variation. In talipes varus, the pedal elements are orientated in a plane towards the midline of the body and in talipes valgus away from the midline of the body (Resnick 2002, 4612–13).

### **9.3 Infectious disease**

*Natasha Powers (with Amy Gray Jones)*

#### ***Non-specific infection***

Skeletal changes as the result of non-specific infectious disease are defined by the level of tissue affected (Roberts and Connell 2004, 37).

### *Periostitis*

Bony proliferation resulting from inflammation of the periosteum is classified as healed or active at the time of death (woven, lamellar or mixed) as described by Roberts and Connell (2004, 35) and by the type of bone formation seen (striated, spicular). Erosive (lytic) lesions are also noted where applicable.

Periostitis is diagnosed where the bony changes overlie the original surface of the bone cortex, though in later stages this may appear as a generalised expansion of the shaft. There are no cloacae or other indications of infection in the medullary cavity (Ortner 2003, 209–10).

Such a reaction may also be indicative of trauma, of inflammation resulting from a metabolic disorder such as scurvy, or as the result of venous stasis. Thus ‘periostitis’ is also used as a descriptive term for bony change diagnostic of inflammation of the periosteum, when this change forms part of the diagnostic criteria of a specific infectious or traumatic condition. In particular the term is used to describe visceral rib lesions (that are not pathognomic of a specific disease) and maxillary sinusitis.

### *Osteitis*

Inflammation of the bone cortex is described as healed or active at the time of death (woven, lamellar or mixed).

Osteitis is most commonly seen in conjunction with osteomyelitis and distinguishing the conditions may be problematic. Osteitis is diagnosed by the presence of expansive changes to the bone shaft without cloacae, where additional bone does not appear to overly the original cortex, rather to be contiguous. Micro-porosity resulting from expansion of the shaft is also seen. Osteitis is used as a descriptive term for bony change diagnostic of inflammation of the cortex in specific infectious conditions.

### *Osteomyelitis*

Lesions are classified as proliferative or erosive, healed or active at the time of death (woven, lamellar or mixed) as described by Roberts and Connell (2004, 35) and by the type of bone formation seen (striated, spicular). Sclerosis and sinus formation are noted and the likely direction of pus drainage commented on.

Osteomyelitis is diagnosed by the presence of gross expansion or destruction of the skeletal element. The formation of cloacae, involucrum or sequestrum clearly indicating the involvement of the medullary cavity is considered a pathognomic requirement (Ortner 2003, 199).

A diagnosis of sclerosing osteomyelitis of Garré requires the presence of significant sclerotic changes with minimal sinus formation (Aufderheide and Rodríguez-Martín 1998, 178).

Again, osteomyelitis is used as a descriptive term for specific infectious conditions (eg venereal syphilis). Periapical lesions resulting from oral pathology are not also classified as osteomyelitis (section 7.5), though generalised infections of the mandible or maxilla are.

### *Septic arthropathy*

Any non-specific infection that extends into a joint space is classified as a septic arthropathy. Destructive change with sinus formation and ankylosis, together with mono-articular involvement, are used to differentiate from degenerative joint disease (Aufderheide and Rodríguez-Martín 1998, 107). If more than one joint is affected but a non-specific infectious cause suspected, diagnostic radiography is employed following Resnick (2002, 2431).

### *Specific infection*

The element, segment and aspect affected, and the type and level of bony change are recorded and changes described following Brickley and McKinley (2004). The severity of lesion is noted by the level of tissue affected; thus the terms periostitis, osteitis and osteomyelitis are used (above, Non-specific infection). Changes are noted as proliferative or destructive.

### *Tuberculosis*

Tuberculosis is diagnosed macroscopically according to the morphology of the lesions and their distribution throughout the skeleton, using the diagnostic criteria presented by Aufderheide and Rodríguez-Martín (1998), Ortner (2003), and Resnick (2002). The main diagnostic characteristics are: predominantly destructive lesions affecting the vertebral bodies or long-bone metaphyses; a predilection for the lumbar and thoracic vertebral bodies and the large joints of the appendicular skeleton; little reactive new bone formation; and frequent vertebral collapse with kyphosis of the spine.

Rib lesions, consisting of active/remodelling new bone and/or erosive lesions, are considered to be an extension of the spinal foci where they are confined to the rib head/neck and/or adjacent to affected vertebrae. In cases where rib lesions are distributed over all or part of the rib shaft, and/or not associated with affected vertebra, they are considered to represent adjacent pulmonary infection.

### *Brucellosis*

The skeletal changes of brucellosis are difficult to distinguish from those of tuberculosis. Examination of macroscopic changes and their distribution are employed to describe the lesions of this disease with reference to Capasso (1999) and Aufderheide and Rodríguez-Martín (1998, 192–3). Significant proliferative changes in the vertebrae, together with an absence of paravertebral abscesses or collapse of the spine, are used to differentiate the two conditions as outlined by Aufderheide and Rodríguez-Martín (1998, 193). Osteolysis of the superior margin of the vertebra with sclerosis is considered a typical bony response (Ortner 2003, 217).

### *Treponemal disease*

The specific diagnosis of treponemal disease is problematic. Yaws and syphilis are closely related and many authors consider them virtually indistinguishable in skeletal material (Buckley and Dias 2002, 179). All suspected cases are recorded under the general term treponematoses, though less certain instances may also be ascribed a differential diagnosis of non-specific infection, according to the level of bony tissue involved. Certainty of diagnosis is based on the presence of a combination of these characteristics. The presence

of gumma, 'sabre shin' and/or caries sicca are considered pathognomic of treponemal disease. The specific distribution of skeletal lesions without these pathognomic changes is highly suggestive of the condition, the level of confidence of diagnosis dependent on the number of criteria fulfilled. A probable or possible diagnosis is given either because the bony changes are ambiguous or, more often, because of the absence of sufficient or specific skeletal elements to establish patterns of change. Tibial periostitis/osteitis alone, and non-specific infectious changes in a single skeletal element, are not considered diagnostic. The justification for the specific diagnosis (gumma, distribution etc) is recorded for each individual.

Diagnosis of a specific treponemal disease is attempted where the pattern of elements affected, the date, age at death and provenance of the material (urban/rural etc) allows (Aufderheide and Rodríguez-Martín 1998, 155; Hackett 1975, 237). Congenital treponematoses is most likely to indicate the presence of venereal syphilis within a population; however, yaws may also be congenitally transmitted (Ortner 2003, 277) and thus specific diagnosis is not based on this alone. The presence of infectious changes consistent with the secondary stage of the disease is taken to indicate venereal syphilis (Buckley and Dias 2002, 180) as is unilateral tibial involvement (Rothschild 2003, 107). Rothschild and Heathcote (1993) are referred to for differential diagnosis of yaws and syphilis.

Diagnostic changes of tertiary treponemal disease are considered to be:

***caries sicca*** with reference to Jaffe (1972, 930–3), Aufderheide and Rodríguez-Martín (1998, 161–2) and Ortner (2003, 280–3);

***gummatous foci***, established according to descriptions provided by Jaffe (1972, 930–41), Aufderheide and Rodríguez-Martín (1998, 160) and Buckley and Dias (2002, 180) and gummatous arthritis (Aufderheide and Rodríguez-Martín 1998, 160);

**'sabre shin'** with reference to Ortner (2003, 275–304) and Rothschild and Heathcote (1993, 202);

***Charcot joint*** (Aufderheide and Rodríguez-Martín 1998, 160; Rogers and Waldron 1989, 94);

***distribution pattern*** of both gummatous and non-gummatous changes (Aufderheide and Rodríguez-Martín 1998, 159; Buckley and Dias 2002; Steinbock 1976); as a general rule, it is considered that characteristic infectious changes in a number of the following sites are diagnostic: the clavicles, sternum, acromion, tibiae, distal humeri and proximal radius and ulnae. Distribution is also used to distinguish between the different treponemal conditions.

Also considered are:

***destructive facial/nasal changes*** particularly with perforation of the palate or periostitis, gumma formation or severe bony necrosis in the mandible (Jaffe 1972, 934);

***type of bony change*** a rough and hypervascular surface with cortical thickening reduction of the medullary canal by sclerotic trabeculae. Sequestrum formation is uncommon (Aufderheide and Rodríguez-Martín 1998, 159); marked hypervascularity may be present (Ortner 2003, 278); expanded long bones with large striae, pits, nodes of bony change and superficial cavitations may all be considered diagnostic of syphilis (Hackett 1975, 236);

***aortic aneurysm*** – sternal erosion may be due to aortic aneurysm (Hackett 1975, 237);

**dactylitis** – symmetrical dactylitis is commonly seen in yaws (Ortner 2003, 275) and less commonly in venereal syphilis than in its congenital form (Jaffe 1972, 937);

**lesions resulting from secondary venereal syphilis** are diagnosed on the basis of the skeletal distribution of periostitis and osteitis (by definition without pathognomic gumma or caries sicca) with reference to Buckley and Dias (2002, 179) and Ortner (2003, 278, 280);

**pathological fractures** (Jaffe 1972, 940) are recorded according to the methods outlined in 0 but were not considered pathognomic;

**non-specific lesions resulting from treponemal ulceration** of the soft tissues; subsequent, non-specific infection of ulcerated tissues, may increase bony necrosis leading to perforation of the cortex (Jaffe 1972, 936);

**congenital syphilis** (and/or yaws) is diagnosed by the presence of gross enamel defects (Hillson 1996, 171) in conjunction with infectious skeletal lesions (Ortner 2003, 302);

**Mulberry molars** with marked hypoplastic defects around the cusp, below which the crown is normal and above which lie gross enamel defects;

**Hutchinson's incisors** – 'pumpkin seed' shaped maxillary first incisors with a pronounced notch on the incisal edge. Peg mandibular first incisors are also considered during diagnosis.

A differential diagnosis of congenital syphilis is asserted on the basis of the distribution pattern of non-specific infectious lesions, following Lewis (2000, 51).

### *Leprosy*

The response to this disease is highly variable, the immune status of each infected person determining the type and severity of pathological change (Andersen and Manchester 1992: 123, Andersen et al 1994, 21). Diagnostic changes of leprosy are considered to be:

bilateral and symmetrical **resorption of the alveolar process** of the maxillae;

**resorption of the anterior nasal spine** (Andersen and Manchester 1992, 123–4);

**haematogenous osteomyelitis**, with multiple osteolytic lesions (leprous osteomyelitis) (Resnick 2002, 2548);

**concentric remodelling** of the bones of the extremities due to the absorption of cancellous bone resulting in a tapered appearance (ibid, 2550);

**volar grooves** as the result of claw hand deformity (Andersen and Manchester 1987).

Also considered are:

**tarsal disintegration** involving the fragmentation of the articular surfaces of the tibia, talus and calcaneus (Resnick 2002, 2550–1);

**secondary infectious changes** particularly in the extremities.

### *Mycotic infection*

Bone involvement in this disease is an uncommon finding resulting either from the extension of soft tissue lesions or haematogenous spread. Ortner (2003, 325) states that diagnosis between different fungal diseases is often impossible in dry bone, thus as a general rule, no attempt is made to differentiate between specific diseases. Where

differentiation is attempted it is carried out with reference to Ortner (2003, 325–8) and Aufderheide and Rodríguez-Martín (1998, 212–22). The bilateral but random distribution of lytic lesions with little or no sclerosis is considered to be diagnostic.

### *Actinomycosis*

Bone involvement in this disease is uncommon, usually resulting from the extension of soft tissue lesions. A diagnosis of actinomycosis is made with reference to Ortner (2003, 319) and Aufderheide and Rodríguez-Martín (1998, 194).

Diagnostic changes are considered to be:

***periosteal involvement*** ossifying in the later stages;

***hypervascularity***;

superficial erosion of the cortex with ***little or no sclerosis***;

***vertebral lytic lesions*** limited to the centrum (Ortner 2003, 319) or posterior elements (Aufderheide and Rodríguez-Martín 1998, 194) with no subsequent collapse and primarily affecting the thoracic or lumbar spine; the absence of involvement of the intervertebral discs differentiates from tuberculosis;

***mandibular or maxillary lesions*** consisting of small lytic lesions with a sclerotic margin; ***'worm eaten'*** lesions in the flat bones;

***rib lesions*** most often in the right hemi-thorax (Aufderheide and Rodríguez-Martín 1998, 194).

Also considered are:

***secondary joint involvement***;

***metaphyseal lesions*** with extensive destruction and periosteal bone proliferation.

### *Nocardosis*

Bony lesions are rare and result from the haematogenous spread of infection. Diagnosis of nocardosis is made with reference to Ortner (2003, 323). The presence of lytic cavitations of cancellous bone with little reactive bone formation is considered diagnostic. Fistula extending from lytic lesions and joint involvement are also considered.

### *Typhoid*

Osteomyelitis as the result of typhoid infection occurs in a very small number of clinical cases. A differential diagnosis of typhoid is considered when osteomyelitic changes are noted in the ribs, tibiae (with diaphyseal abscess) or spine (erosion of the vertebral plates with sclerosis, reduction in disc space and marginal osteophytes) (Aufderheide and Rodríguez-Martín 1998, 191).

### *Smallpox*

The skeletal involvement of smallpox varies from 2–20%, and 80% of those with skeletal lesions are less than 5 years old. Diagnosis of smallpox was based on the age of the individual affected and the distribution of osteomyelitis (Ortner 2003, 333–4).

Diagnostic changes are considered to be:

*osteomyelitis of the elbow* (80% of reported skeletal cases involve this joint, though smallpox was also considered where bilateral changes were present in other locations);

*epiphyseal involvement* and destruction;

*metaphyseal destruction* when lesions located or initiated near the growth plate spread;

*bilateral changes*;

*absence of massive sequestration*;

*reactive changes* and involvement of all three bones of the arms (tuberculosis will tend to affect only the humerus and ulna).

Also considered are:

**secondary joint changes** and **limb deformity** (Ortner 2003, 334; Jackes 1983).

### *Rubella*

Skeletal changes as the result of in utero infection are seen in infants less than 3 months. A diagnosis of rubella is considered where metaphyseal lesions are present in the distal femora and proximal tibiae, without periostitis, sometimes leading to pathological fracture. The diaphysis is not involved. Diagnosis is strengthened if congenital abnormalities are also present (Aufderheide and Rodriguez-Martin 1998, 210). Differentiation from congenital syphilis is made on the basis of the absence of ossifying periostitis. Poor mineralisation, enlargement of the anterior fontanel and growth retardation are also considered as indicative (Ortner 2003, 336)

Rubella is also considered a differential diagnosis for arthritic changes in the hands, wrist, knee or ankle of adults.

### *Poliomyelitis*

Where there are skeletal indications of asymmetrical paralysis, demonstrated by atrophy or limb shortening with osteopenia, an underlying diagnosis of polio is considered (Aufderheide and Rodriguez-Martin 1998, 212). Post-paralytic deformities such as *coxa valga* and *pes calva* are considered to support this diagnosis.

### *Parasitic infection*

When the remains of a hydatid cyst are recovered with a skeleton, parasitic (tapeworm) infection is diagnosed only when the contextual integrity of the grave is good. Differential diagnosis follows Aufderheide and Rodriguez-Martin (1998, 243).

Parasitic infection, including malaria, is considered as a differential diagnosis where non-specific skeletal changes are seen, particularly in conjunction with cystic, lytic lesions, and with reference to specific conditions outlined by Aufderheide and Rodriguez-Martin (1998, 222–4) and Ortner (2003, 337–41).

## **9.4 Trauma**

### *Don Walker*

The recording and analysis of traumatic lesions in archaeological remains has been the subject of much debate over recent years (Buikstra and Ubelaker 1994, 119–20; Grauer

and Roberts 1996, 532–5; Lovell 1997, 139–53; Judd 2002, 1255–65). There are problems of standardisation, especially concerning the recording of long bone trauma. The methods employed at the museum of London follow those of Buikstra and Ubelaker (1994, 119–20).

### **Fractures**

All fractures are examined macroscopically and radiographically. Where possible, both antero-posterior and medio-lateral radiographs are taken. Recording follows Roberts (2000), summarised below.

**Note the bone and side affected** (if applicable).

**Identify location** using standard anatomical terms. For long bones, metacarpals, metatarsals and phalanges, location was described as proximal, middle or distal shaft, and proximal or distal epiphysis or joint surface. A note is made when fractures occur at the junction of two segments. The location of fractures in other areas of the skeleton is recorded using description combined with measurements from anatomical landmarks.

**Identify type** following (Lovell 1997, 141–4) eg transverse, oblique, spiral, comminuted, greenstick, compression, avulsion, and depression.

**Note the state of healing** (healed, healing, unhealed), the size and type of bone forming the callus (woven, lamellar or mixed). Where the broken ends have not unified, whether the fracture was in the process of unification (healing) or had permanently failed to unite (healed) is noted. Non-union is divided into hypertrophic (some healing potential) or atrophic (diminished healing potential).

**Record evidence of infection** on the fractured bone (or any closely associated bones), categorised as outlined in section 8.3, together with the proximity to the fracture site.

**Note evidence of pre-existing pathology**, which may be related to the fracture

**Note any evidence of joint disease adjacent to the fracture**, contour change, osteophytosis, subchondral pitting or eburnation. The presence of any pseudoarthroses, or joints affected by avascular necrosis is also noted.

**Record any evidence of atrophy** in the affected limb, established by visual examination and measurement of paired elements. For diagnosis it is considered that atrophy may result from disuse or biomechanical dysfunction of the limb itself, or in another area of the body secondary to nerve damage.

Deformity is measured relative to the proximal or medial section from the corresponding radiographs (Grauer and Roberts 1996, 534–5). The excavation records for the skeleton are also referred to, to establish evidence of deformity or unusual limb orientation. Deformity is established by quantification of:

- the direction and angle of linear deformity (in degrees)
- the angle of any rotational deformity (in degrees)
- the overlap of the fractured ends (in mm), and whether the overlap is anterior, posterior, medial or lateral
- the amount of apposition (100% = full apposition)

### *Fracture separation of epiphysis*

Subadult epiphyseal separation and fracture can damage a bone's growth plate and is recorded following Salter and Harris (1963, 587). Any shortening or other deformity as a result of growth plate damage is noted.

### *Pathological fracture*

Fractures occurring as a secondary consequence of a pre-existing disease are noted according to Lovell (1997, 144). Care is taken to distinguish these types of fracture from those caused by traumatic injury.

### *Dislocation*

Dislocation is recorded as full or partial (subluxation) defined according to Lovell (1997, 140). Description includes all changes within and around the affected joint. Associated fractures and complications are fully recorded. Care is taken to distinguish traumatic subluxations from those caused by congenital weakness (above, 8.2, Hip dysplasia (subluxation)).

### *Soft tissue injury*

The location and dimensions of all examples of bony reaction (both lytic and blastic) in response to soft tissue injuries are recorded. These may occur in isolation or in association with bone fracture and have been found to affect muscle (myositis ossificans), cartilage, tendons and ligaments (Resnick 2002, 4727). Where the form of an ossified structure did not resemble that of soft tissue, a diagnosis of an ossified haematoma is considered.

### *Enthesopathies (musculoskeletal markers)*

Enthesophytes are defined as small bony projections at the site of muscle attachment resulting from chronic or acute trauma or a variety of other pathological conditions (eg in association with DISH; see section 8.5). The location and distribution is recorded together with relation to any other pathological change identified on the skeleton. They are described as 'slight' or 'pronounced'.

### *Blunt force, sharp force and projectile injury*

Lesions are distinguished from post-mortem damage with reference to Byers (2002) and recorded following Boylston (2004). A full written description details the size, shape, location and state of healing of each lesion. The written record is supported by diagrams, using standard proforma (Buikstra and Ubelaker 1994, chapter 2: attachment 3a–10b), and photographs as required.

In cases of cranial injury, endocranial inspection is carried out whenever possible, and changes such as evidence of infection, internal bevelling or flakes of still-attached bone around the lesion are recorded. Care is taken in examining the edges of cranial fragments, to allow differentiation of peri-mortem from post-mortem fracture (Boylston 2004, 40). Injuries are characterised according to the types outlined in Table 26.

Type of force	Fracture types	Reference
Blunt	linear cracks in the cranial surface	Lovell 1997, 153–7
	depressed (pond) fractures	
	depressed (stellate) fractures	
	depressed (comminuted) fractures	
Sharp	deep wounds slicing (partially or completely) through bone	Boylston 2004, 40
	glancing wounds leaving shallow indents	
	deep angled wounds removing a slice of bone or leaving a flap of bone attached	
Projectile	slow velocity – similar to blunt trauma	Berryman and Haun 1996, 4
	high velocity – complete penetration of the vault with radiating fractures and internal bevelling	
	gunshot – complete penetration of the vault with radiating fractures and external bevelling	

*Table 26 Types of cranial injury*

Recognition of the location and angle of a wound may assist in the interpretation of the direction of the blow and/or the position of the affected individual at the time (Boylston 2000). Radiating fractures are observed to attempt patterning of the sequence of multiple injuries (Boylston 2004, 41). Observations of subtle widening between the cranial bones, or of basal ring fractures (where fractures lines ‘jump’ across the foramen magnum) are also recorded (Lovell 1997, 155).

A smooth and polished edge indicates where the blade edge has come into contact with the bone, and should therefore display the angle of the blow. This is recorded in degrees from the sagittal plane wherever possible. Careful measurement and description of terminal fractures is carried out to assist in the interpretation of the type of weapons used (Boylston 2004, 40).

Sharp force trauma relating to decapitation is considered where single or multiple cut marks are observed on any of the cervical vertebrae, the base of the cranium, the mandible or clavicles.

Projectile injuries caused by slow moving objects can produce lesions similar to blunt force trauma. Recording follows the same methods, noting the size and location of any lesion, radiating fractures and location of any bevelling.

### ***Surgical intervention, autopsy and anatomical specimens***

Evidence of amputation, trepanation, or any other surgical intervention is recorded with written description, diagrams and photographs, and with reference to Roberts (2000, 344–5). Where possible, evidence relating to the type of instrument used to perform the intervention is recorded: striations, skip marks and re-starts, the direction, angle and order of cut-marks. The state of healing and any evidence of infection are noted.

## 9.5 Joint disease

### *Spinal joint disease*

*Lynne Cowal*

The recording vertebral pathology can be problematic due to the number of individual joint surfaces which may be affected. In consideration of this, joint disease is recorded at the vertebral joint interface level: if any of the small joints are affected in an opposing pair of vertebrae (eg first to second cervical vertebrae); the presence of joint disease is scored for that interface. Changes in the atlanto-axial joint and the apophyseal joint of the first and second cervical vertebrae are recorded individually. Conditions outlined here use a coded system to record presence/absence.

### *Osteoarthritis*

Degenerative joint changes are observed as macroscopic changes to the zygoapophyseal joints and recorded following Brothwell 1981, 150 (after Sager 1969). True osteoarthritis is defined by the presence of eburnation. Degenerative changes were not recorded for uncovertebral, costovertebral or costotransverse joints.

### *Osteophytes*

Osteophytes affecting the margins of vertebral bodies are recorded at the joint interface level following Brothwell 1981, 150 (after Sager 1969) and scoring formation as intermittent, continuous or extensive. Where changes can be definitely associated with a specific condition (eg DISH, kyphosis, AS), they are not recorded under vertebral joint disease but are fully itemised in the description of the relevant condition.

### *Intervertebral disc disease*

Dehydration of the nucleus pulposus in the disc causes premature hardening, rupturing and herniation of the disc, manifest on the vertebrae as pitting on and around the centrum. Diagnosis of intervertebral disc disease follows Rogers and Waldron (1995, 26–7) and is recorded by the location on the centrum: under the footprint of the annulus fibrosus; on the central articular surface; or covering the entire surface.

### *Schmorl's nodes*

The recording of a Schmorl's node was based on the maximum dimension of depressed circular or linear lesions in the articular surfaces of the centra: less than 15mm (small), 15–25mm (medium) and greater than 25mm (large) (Connell and Rauxloh 2003, 18).

### *Fusion*

Fusion of two or more vertebrae when identified as a general degenerative change, is recorded by location: whether the centrum and/or zygoapophyseal joints are involved. A distinction is made between pathological and congenital segmentation failure (above, 8.2, Segmentation failures (congenital fusion) and developmental errors).

## *Extra-spinal joint disease*

*Jelena Bekvalac*

Joint diseases are identified and observed at a macroscopic level in dry bone and diagnosed following Ortner (2003, 545–88); Aufderheide and Rodríguez-Martín (1998); Rogers and Waldron (1995); and Rogers et al (1987).

A clear description is made, classifying changes as proliferative or erosive prior to any attempt at further diagnosis, as proposed by Rogers et al (1987, 180–2).

Joint diseases are diagnosed considering the age and sex of the individual and any concurrent pathology. The criteria followed to identify specific conditions are based upon location, severity, skeletal distribution (elements affected, unilateral or bilateral change) and type of the pathological lesions: bone formation (including ankylosis) or destruction (erosive). Specific diagnosis therefore often relies on good preservation and completeness of the individual.

Diagnostic considerations are:

**proliferative bone formation** – described with reference to location, severity and grade and if ankylosis occurred (Sager 1969);

**erosive destruction** – the size, shape and location of any lesion is recorded. A description of the lesion is made with respect to the type of destruction observed (eg lytic or granuloma foci), surface changes observed within the erosive lesion surface and the appearance of the margins of the lesion;

**inflammation** – a record is made of location and distribution in relation to the joint with a description of the type of bone formation present and if active or healed.

Ankylosis (fusion) is identified if two normally articulating elements are partially or fully fused and the ankylosis emanates from the joint surfaces (intra-articular) or the joint margins. The presence of ankylosis is considered as a potential precursor or indicator of another underlying disease. The location of fusion of the joint margins (eg superior, anterior) and is also noted.

## *Osteoarthritis*

Degenerative joint changes are recorded for each joint surface using a presence/absence system and catalogued by the severity of marginal osteophyte formation and pitting each on a three-point scale (slight, moderate or severe), and the proportion of the joint surface eburnated (<>50%). The small joints of the hands and feet are described and recorded separately with reference to the same system. Eburnation is considered a diagnostic requirement for osteoarthritis (Rogers and Waldron 1995, 13, 43 and 99). Details of eburnation are also recorded: which part of the articular surface is affected; any grooving visible; and the degree of severity of such grooving. Sub-chondral cysting resulting from the breakdown of cartilage is indicated by the presence of micro and macro porosity.

## *Inflammatory osteoarthritis*

Inflammatory osteoarthritis is defined following Rogers and Waldron (1995, 69). If indications of inflammation are present, the location and distribution, proximity to the joint, the size of the area affected and a description of the type of periosteal new bone formation are recorded.

### *Ankylosing spondylitis (AS)*

This condition is identified by the observation of bilateral sacroiliac ankylosis and fusion of the anteriolateral surface of the vertebrae, the latter occurring from the lumbar vertebrae upwards (bamboo spine). The involvement of the apophyseal joints and ankylosis and ossification of the interspinous and supraspinous ligaments are a requirement for diagnosis (Ortner 2003, 571–2; Resnick 2002, 1026).

### *Reiter's disease*

Where changes are identified predominantly in the hands, feet, vertebrae and sacroiliac joint and involve proliferative change and enthesophyte development at the iliac crest and Achilles tendon, a diagnosis of Reiter's disease is considered. Diagnostic lesions include the following (Aufderheide and Rodríguez-Martín 1998, 104-105; Resnick 2002, 1110–24):

***periosteal new bone*** on the bone shaft surfaces of the metacarpals, metatarsals and phalanges;

***asymmetrical vertebral syndesmophytes***;

***costovertebral and apophyseal joint ankylosis***;

***ossification of the supraspinous ligament***.

### *Psoriatic arthropathy*

This condition principally affects distal interphalangeal joints of the hands and feet, with the main diagnostic feature the erosion of the distal interphalangeal joints. Lesions associated with this condition are rounded cup-like erosions with tapering of the bone into a pencil point shape: 'pencil in cup' deformity (Aufderheide and Rodríguez-Martín 1998, 104).

### *Rheumatoid arthritis (RA)*

Diagnosis is heavily dependent upon the completeness of the skeleton studied. Erosive lesions predominantly affecting the metacarpophalangeal and proximal interphalangeal joints of the hands and the metatarsophalangeal joints of the feet are considered indicative of RA (Aufderheide and Rodríguez-Martín 1998, 99; Resnick 2002, 891–974). A full description of the location, type and severity of erosive lesion and any consequent ankylosis is made.

### *Gout*

Principal diagnostic criteria are the observation and description of sharply defined lytic lesions of the para-articular joint surfaces of the first metatarsal, with unilateral joint involvement (Aufderheide and Rodríguez-Martín 1998, 108).

### *Enteropathic Arthropathy*

This condition is identified by small erosive lesions of the minor and major joints of lower limbs, predominantly the distal interphalangeal joints of the feet, with unilateral involvement (Aufderheide and Rodríguez-Martín 1998, 105). Skeletal distribution is again key.

### *Rotator cuff disease*

Defined when damage and pathological change to the muscle insertions and attachments of the rotator cuff muscles (supraspinatus, infraspinatus, teres minor and subscapularis) destabilise the shoulder joint. Diagnostic criteria for rotator cuff disease include the following (Aufderheide and Rodríguez-Martín 1998):

***porosity***;

***marginal osteophytosis*** graded as for all degenerative joint change (8.5, Extra-spinal joint disease);

***enthesophytic development at the muscle attachment*** recorded as slight, moderate or severe;

partial or complete ***ankylosis***;

partial or complete ***dislocation***.

### *Diffuse idiopathic skeletal hyperostosis (DISH)*

This condition is classified for recording purposes as a joint disease with further discussion as required. Excessive amounts of bone formation at the joint entheses and ankylosis of the spine ligament with no intervertebral disc involvement or apophyseal fusion indicate this condition. DISH is only diagnosed if at least four contiguous vertebrae are fused, with bony bridges rooted from the anterolateral aspect of the vertebral bodies on the right side of the vertebrae ('dripping candle wax' bony fusion) (Aufderheide and Rodríguez-Martín 1998, 97; Resnick 2002, 1477).

### *Neurotrophic arthropathy (Charcot joint)*

This secondary proliferative arthropathy, results from the loss of sensory nerve supply and continual mechanical stress of the affected area produces an exuberant bony response (Ortner 2003, 587). For diagnosis, consideration is made with respect to concurrent pathology. If involved with leprosy, articular grooving and eburnation may be particularly evident in the knee joint. The elements affected and distribution are also considered. Diagnostic criteria include the following (Aufderheide and Rodríguez-Martín 1998, 107):

***proliferative bone exostoses formation***;

***bone fragmentation*** of the subchondral bone;

***eburnation***;

***irregular destruction***.

### *Ochronotic arthropathy*

This deposition of organic pigment in the cartilage of the diarthrodial joints, identified in adults later in life, does not affect the small joints of the hands and feet or the sacroiliac joints (Aufderheide and Rodríguez-Martín 1998, 111). Diagnostic criteria include:

***vertebral disc space narrowing***;

involvement of the ***major joints***;

***eburnation*** due to loss of pigment cartilage, indicates the later stages of the condition.

### *Haemochromatosis*

Primary diagnostic criteria are small destructive lesions of the metacarpophalangeal and interphalangeal joints with inflammatory response on the associated bone surface. Skeletal completeness is again an important factor for a more conclusive diagnosis. The age of the individual is considered relevant, as the condition affects older adults.

### *Calcium pyrophosphate deposition disease (CPPD or pseudogout)*

This results from deposition of crystals but is non-lytic and does not affect the para-articular surfaces. It is differentiated from gout by the location of the lesion and joint affected, the knee being most commonly involved. Intra-articular ossified cartilage is also a diagnostic feature (Aufderheide and Rodríguez-Martín 1998, 114).

### *Sarcoidosis*

Primary diagnostic criteria are destructive lesions in the second and third phalanges of the metacarpals and metatarsals. Scalloped lytic lesions with a round or slightly lobular shape, between 1mm and 1cm in size are considered diagnostic (Ortner 2003, 341). The age and ancestry of the individual are considered where possible: sarcoidosis affects adults more commonly between the ages of 20–40 years with a predilection for individuals of African descent (Resnick and Niwayama 1995, 4337).

### *Amyloidosis*

This is considered as a diagnosis for an erosive arthropathy affecting the joint surfaces of the hand, wrist, shoulder and hip, with subchondral cyst formation and peri-articular osteopenia. Joint destruction is not common. The condition primarily affects the elderly, and the age of the individual is, therefore, also considered. As it is secondary to other chronic infections and inflammatory diseases, concurrent pathology is also considered (Aufderheide and Rodríguez-Martín 1998, 115).

## **9.6 Metabolic and nutritional disease**

### *Richard Mikulski*

Metabolic diseases are observed and identified at a macroscopic level in dry bone and assessed following the criteria and guidelines described in Ortner (2003, 383–418) and Aufderheide and Rodríguez-Martín (1998, 305–44). The age and sex of the individual is considered when attempting diagnosis.

### *Deficiency diseases*

#### *Rickets*

Rickets is diagnosed following Mays et al (2006) and Ortner and Mays (1998). The definition of the nature of the vitamin D deficiency (ie active versus residual/healed) is based on the characteristics of the bony changes and age of the individual. Diagnostic changes include:

***bowing of long bones***, particularly the legs and the humerus and forearm in infants;

*lack of cortical density* towards the metaphyses;

*thickening of cranium* by subperiosteal bone deposition (usually external but occasionally internal);

*flaring and cupping* of metaphyseal ends;

*decreased curvature of the ribs*;

*rachitic rosary* characterised by enlarged or flared sternal rib ends.

### *Osteomalacia*

Osteomalacia is considered where evidence of stress, hair-line or healed fractures to the base of the acromial spine is present, especially where such changes are bilateral (Brickley et al 2007). Other strong evidence includes:

*healed and unhealed fractures* at or around the base of the acromial spine of the scapulae;

*vertebral collapse*;

angular '*kinking*' of *sacrum*;

*stress fractures*;

*accentuated cupping of vertebral endplates* leading to angular kyphosis in severe cases, usually at the level of the eighth or ninth thoracic vertebrae;

*decreased curvature in ribs*;

*angular deformation to sternum*;

*abnormal morphology of the pelvic girdle* severe reduction of subpubic angle and narrowing of pelvic canal.

### *Scurvy*

Infantile scurvy is considered according to Brickley and Ives (2006). Diagnostic changes include:

*profuse irregular porous new bone* plaques on the ectocranial surfaces (with 'honeycomb' appearance), pitting to lingual aspects of mandibular rami and new bone on the diaphyses of long bones (in particular the humeri, femora, tibia and fibulae);

*new bone formation in the orbits*;

*flaring of sternal rib ends* 'scorbutic rosary'.

Other evidence of possible scurvy is considered to include porosity or porous new bone to muscle attachment sites, such as the suprascapular fossae and the external surfaces of the iliac blade.

Symmetrical periosteal lesions with a shell of lamellar bone attached to the original cortex via woven bone, evidence of haematoma, pathological fractures, gingival haemorrhage leading to tooth loss and orbital lesions are all considered indicative of scurvy in adults (Aufderheide and Rodríguez-Martín 1998, 313).

### *Cribra orbitalia*

Cribra orbitalia is a descriptive term for porotic hyperostosis lesions of the orbit. The characteristic lesions of cribra orbitalia were identified as pitting of the compact bone varying in size from capillary like impressions to coalescing outgrowths (Stuart-Macadam 1989, 217). The orbital roof is examined macroscopically for evidence of pathological change. Each orbital roof is recorded as a single unit with cribra orbitalia noted as present, absent or unobservable. Lesions are recorded following the grading system defined by Stuart-Macadam (1991) (types 1–5).

### ***Metabolic disorders***

#### *Osteoporosis*

Cases of osteoporosis are considered where generalised cortical thinning is observed in addition to vertebral compression fractures or femoral neck fractures. Where possible, the trabeculae are observed using low-level magnification. Diagnostic changes include the following (Aufderheide and Rodríguez-Martín 1998, 314–15):

- diminished bone mass;***
- sclerotic atrophy*** in trabeculae;
- vertebral micro fractures;***
- concave/biconcave appearance to vertebral bodies;***
- compression fractures*** and secondary kyphosis;
- cortical thinning;***
- multiple healed/unhealed rib fractures;***
- increased diameter of medullary cavity;***
- increased porosity/lamination of cortex;***
- femoral neck fractures.***

#### *Infantile cortical hyperostosis (Caffey's disease)*

Where massive deposition of layered periosteal woven bone is seen in one or more element of neonatal or infant remains, a diagnosis of infantile cortical hyperostosis is considered (Lewis 2007, 143–5). Changes affecting the mandible, clavicle, long bones and ribs are considered typical, where the diaphyses are affected but the metaphyses are not involved.

#### *Fluorosis*

Fluorosis is considered where widespread profuse periosteal new bone formation is observed (broad flat sheets, nodular excrescences or a combination of both), affecting the axial skeleton in particular (Aufderheide and Rodríguez-Martín 1998, 317). Diagnostic changes include:

- narrowing of medullary canal;***
- ossification of interosseous ligaments;***

*calcification of joint capsules;*

*increased fracture frequency;*

*expansion of tables in cranium*, with obliteration of the marrow space.

#### *Leontiasis ossea*

This condition is diagnosed where excessive bone formation is noted on the cranial and facial bones, with uniform or nodular periosteal deposition and possible narrowing of the orbits and paranasal sinuses (Aufderheide and Rodríguez-Martín 1998, 417).

#### *Generalised hyperostosis with pachydermia*

Excessive formation of periosteal bone which blends with the original cortical surface, a marked increase in long bone diameters, retention of the medullary canal, and sparing of the joint ends and joint fusion are considered to be indicative of this condition. This condition usually manifests during puberty and progresses through adult life so the age of the individual is considered during diagnosis (Aufderheide and Rodríguez-Martín 1998, 419).

## **9.7 Endocrine disease**

*Bill White*

### *Pituitary disorders*

Tumours of the pituitary gland in an adult can lead to hyper-pituitary disorders and increased growth. These are considered where adult stature lies three or more standard deviations above the population mean. Acromegaly is diagnosed where thickening of bone, facial changes, enlarged ribs and vertebrae and enlargement or erosion of the *sella turcica* are observed (Aufderheide and Rodríguez-Martín 1998, 327–8). Gigantism as a result of hyper-pituitarism is noted if non-uniform epiphyseal fusion leading to growth asymmetry has occurred. Secondary kyphosis, with or without scoliosis, is also considered to support diagnosis (ibid, 326–7). Dwarfism resulting from hypo-pituitarism is signified by extreme shortened stature with elements maintaining normal relative proportions and enlargement or erosion of the *sella turcica* (ibid, 329).

### *Disorders of the parathyroid glands*

Primary hyperparathyroidism is characterised by excessive osteoclastic activity. This is established by diffuse osteopaenia or *osteitis fibrosa cystica* (Aufderheide and Rodríguez-Martín 1998, 331–3). Hypoparathyroidism, an extremely rare condition, is diagnosed following Ortner (2003, 429).

### *Disorders of the thyroid glands*

Hyperthyroidism may cause osteoporosis and is considered as an underlying aetiology for changes outlined in 8.6, Osteomalacia. Differential diagnosis distinguishing hyperthyroidism from hyperparathyroidism may be problematical, but is attempted utilising the descriptions in Ortner (2003, 427). Dwarfism with abnormal growth of the

long bones, short extremities and cranio-facial abnormalities are considered typical of hypothyroidism (Ortner 2003, 426–7, Aufderheide and Rodríguez-Martín 1998, 339).

## 9.8 Neoplastic disease

*Don Walker*

Radiographs were taken of all lesions lacking a secure diagnosis in order to reveal their inner structure and allow accurate classification.

The age and biological sex of the individual concerned is considered when attempting diagnosis of neoplasms. Neoplastic disease is classified as primary or secondary bone tumour or secondary (non-metastatic) bony response to a primary soft tissue tumour Anderson (2000, 204–05). Where specific diagnosis is not possible, neoplastic conditions are classified by the level of tissue from which they originated (Table 27). Initial diagnostic investigations are carried out with the aid of Ortner (2003, 503–44) and Resnick (2002, 3745–4351).

Origin	Neoplasm included
Bone	osteoma; button osteoma; osteoid osteoma; osteoblastoma; osteosarcoma
Cartilage	chondroma; chondroblastoma; osteochondroma; chondrosarcoma
Fibrous connective tissue	fibrous cortical defect
Others	giant cell granuloma; meningioma; osteoclastoma; Ewing's sarcoma; bone cysts

*Table 27 Classification of neoplastic disease*

## 9.9 Circulatory disease

*Brian Connell*

Circulatory disease covers a range of conditions where the circulatory system produces lesions affecting bone tissue. Diagnosis of the following conditions utilise the descriptions of Aufderheide and Rodríguez-Martín (1998, 85–8), as summarised below, and with reference to Resnick (2002, 3597–686) and Ortner (2003, 343–57).

### *Osteochondroses*

#### *Perthes' disease*

Osteochondrosis of the femoral head, as a result of avascular necrosis, affecting superior and anterolateral aspects of the femoral head is considered diagnostic of Perthes' disease. Changes are characterised by a flattened and widened femoral neck and mushroom-shaped femoral head, often lower in position than the lesser trochanter

### *Osgood-Schlatter's disease*

This is recognised as osteochondritis of the tibial tubercle producing a thickened, irregular surface, with calcification in adjacent areas or actual detachment of bone from the tubercle.

### *Sinding-Larsen's disease*

Where changes similar to those of Osgood-Schlatter's disease affect the inferior pole of the patella, this condition is diagnosed.

### *Blout's disease*

This is recognised as osteochondritis of the posteromedial surface of the tibial proximal epiphysis, with concavity of the epiphysis and widening of the epiphyseal plate.

### *Sever's disease*

Osteochondritis of the calcaneal tuberosity producing irregularity of the tuberosity with fragmentation is considered diagnostic. Confirmation by radiograph of increased density is carried out wherever possible.

### *Kohler's disease*

This is recognised as osteochondritis of the navicular, producing a flattened and sclerotic appearance, with the element assuming the shape of a biconcave lens with an irregular contour.

### *Freiberg's disease*

Where the second metatarsal is shortened with a collapsed head and irregular articular surface, Freiberg's disease is diagnosed, with reference to Anderson and Carter (1993).

### *Scheuermann's disease*

Scheuermann's disease is recognised by the presence of osteochondritic erosion and epiphyseal ring fragmentation with loss of anterior height in the vertebrae.

### *Calves' disease*

Flattening and cuneiform deformation secondary to aseptic necrosis of the vertebral body is considered diagnostic of this condition.

### *Keinbock's disease*

Collapse and flattening of the lunate with the formation of sub-chondral cysts and increased bone density indicates Keinbock's disease.

### *Preiser's disease*

This condition is diagnosed where osteochondrosis of the scaphoid bone characterised by an irregular and deformed shape is noted.

## ***Osteonecroses***

### ***Femoral condyle necrosis***

Necrotic destruction of the femoral condyle, usually medial, is characterised as the presence of any concavity 10–25mm in size and surrounded by osteosclerosis.

### ***Slipped femoral epiphysis***

This is defined as an inferior-posterior displacement of the femoral head, often associated with secondary degenerative joint disease.

### ***Hypertrophic osteoarthropathy***

This is defined as symmetrical new bone formation on the diaphyses of the tubular bones of the appendicular skeleton. The following features are considered characteristic of this disease (Aufderheide and Rodriguez-Martin 1998, 91):

***periosteal bone formation*** on the tubular bones without endosteal bone deposition;

***clubbing of fingers and toes*** especially the distal phalanges;

***symmetrical distribution*** of lesions;

***new bone separated from cortex easily.***

## **9.10 Miscellaneous disease**

### ***Tania Kausmally (with Rebecca Redfern)***

Classification of diseases at times poses inherent problems owing to the complexities of the conditions described (Ortner 2003, 435). The diseases included in this category are those in which the cause is poorly understood or those which appear to be determined by cultural factors.

### ***Paget's disease of bone***

Paget's disease is identified from distinct thickening, resulting from an increase in the rate of bone remodelling due to a primary defect in the osteoclasts, and affecting one or more bones but never the entire skeleton (Fraser 1997, 348; Ortner 2003, 435). As this disease is mainly seen in individuals over 40 years of age (Salter 1999, 199), age was considered during diagnosis.

The distribution of the pathology is recorded and the extent of thickening where possible. Any bowing of the weight-bearing limb bones is noted, together with the surface texture (presence of pumice-like, fine porous bone). Radiographs are taken to identify the classic 'cotton wool' appearance of the trabeculae and diploe (*ibid*) and any reduction in the medullary cavity (Ortner 2003, 437). Transverse fractures and secondary osteoarthritis are recorded. Diagnosis refers to Ortner (2003, 435), Aufderheide and Rodríguez-Martín (1998, 413), Salter (1999, 199) and Resnick (2002, 1947–95).

### ***Hyperostosis frontalis interna (HFI)/Morgagni syndrome***

Hyperostosis frontalis interna is diagnosed in adults with a distinct thickening of the frontal bone, composed of the expansion of the diploic trabecular mass bulging endocranially (Aufderheide and Rodríguez-Martín 1998, 419). The condition is mainly seen in middle aged or elderly women (Eldridge and Holm 1940), thus the profile of the individual is considered during diagnosis.

Extent is recorded through descriptive analysis (Aufderheide and Rodríguez-Martín 1998, 419; Hershkovitz et al 1999):

stage 1 – ***rice-grain*** like nodules;

stage 2 – ***moderate bulging*** on the endocranial surface;

stage 3 – ***excessive bulging*** on the endocranial surface (possibly extending to the parietal and temporal regions).

The condition is recorded in skeletal remains where the endocranial portion of the frontal bone was visible. Prevalence rate is crudely calculated based on total number of frontal bones present and does not account for the quantity of fragmentary material present.

### ***Artificial cranial abnormality***

Cranial abnormalities created in infancy as the result of cultural practices were documented following attachment 28 of Buikstra and Ubelaker (1994, 160–63). The recording of pad impressions drew on the descriptions of Aufderheide and Rodríguez-Martín (1998, 36) who suggest that a ‘band’ of 1–1.5cm wide, which is sharply demarcated and located immediately adjacent to the affected suture is characteristic.

### ***Spondylolysis and spondylolisthesis***

It is considered unclear whether these conditions are caused by micro-trauma, a congenital defect, or a combination of both (Ortner 2003, 148; Aufderheide and Rodríguez-Martín 1998, 63; Roberts and Manchester 1995, 78).

Spondylolysis is recognised as an ossification union failure of the *pars interarticularis* of the vertebrae (usually the fourth or fifth lumbar) causing separation of the vertebra into two parts (the vertebral body, the pedicles, and the transverse and superior articulating processes and the dorsal part formed by the laminae, spinous process and inferior articular processes). Spondylolysis is recorded as bilateral or unilateral, the vertebrae affected and any secondary joint changes are noted.

Spondylolisthesis expresses the same pathological traits as spondylolysis but is distinguished by the dislocation of the unstable vertebral body. Diagnosis may be difficult, but is attempted where obvious changes in rim formation or fusion as a result of the forward slip are visible (Roberts and Manchester 1995, 79; Merbs 1996, 217).

Prevalence is calculated as a percentage of the number of laminae present at the affected vertebral level.

### ***Endocranial lesions***

The term endocranial lesions encompasses abnormalities in the skull which do not correspond to post-cranial changes and so cannot be allocated a specific cause or

pathological category. Lesions are recorded describing the location, size and appearance, as well as degree of remodelling and healing, following the four types outlined by Lewis (2004, 89–90). Where possible, suggestions are given for any possible association with another disease. Prevalence is recorded against the number of skulls present.

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