Ancient Monuments Laboratory
Report 77/90

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Summary

Twelve skeletons (10 male, 1 unsexable and 1 female), excavated from Launceston Castle between 1969 & 1972 are reported upon.

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# TWELVE INHUMATION BURIALS FROM LAUNCESTON CASTLE, CORNWALL

## Introduction to the site

A total of 14 inhumations were recovered in excavations during 1969-1972 at Launceston Castle. All were interred sometime during the period c1700-1775AD; the castle was used as a gaol at this time, and it is thought that the interments may represent prisoners. Two of these burials have been the subject of a previous report (Keepax 1973), hence the present work is primarily concerned with the remaining 12.

## The human remains

### 1. Sex, age and bone preservation

Bone preservation was scored as good, moderate or poor on the basis of gross inspection of the remains.

Sex was determined using dimorphic aspects of the pelvis and skull (Workshop of European Anthropologists 1980).

For immature individuals age at death was estimated using dental development (Ubelaker 1978: Fig. 62) and epiphysial fusion (Workshop of European Anthropologists 1980: Fig. 6). Dental wear is probably the most reliable basis for estimating age at death for adults in archaeological collections. However dental wear could not be used to age the Launceston Castle adults: insufficient immature individuals are present to estimate the rate of wear in the group under study, as recommended by Miles (1963). Research by Brothwell yielded a chart (Brothwell 1981: Fig. 3.9) relating molar wear to age in archaeological material, however this is only applicable up to and including the Mediaeval period; dietary changes during the Post-Mediaeval period mean that Brothwell's standards cannot be applied here. Thus, although it was recorded, molar wear is of little use in age estimation in the present case. The morphology of the pubic symphysis (Suchey et al. 1987, 1988) and the state of closure of the skull sutures (Perizonius 1984) were used to estimate adult age at death; these methods are somewhat imprecise, hence the rather broad age categories for many adults shown in Table 1.

Table 1: Preservation and demographic composition of the assemblage

| Skeleton | Sex | Approx. Age | Preservation | Approx. Completeness |
|---|---|---|---|---|
| TJM:B | Female | 25-40 | Moderate | 60-80% |
| TJM:D | Male | 15-18 | Good | 60-80% |
| TJM:E | Male | 30+ | Moderate | 40-60% |
| TJM:F | Male | 21-24 | Good | 60-80% |
| TJM:G | Male? | About 20 | Good | 40-60% |
| TJM:I | Male | 40+ | Moderate | 80%+ |
| TJM:J | Male | 60+ | Moderate | 60-80% |
| TJM:L | Male | Adult | Moderate | 60-80% |
| TJM:M | Unsexable | Adult | Moderate | <20% |
| TJM:N | Male | 50+ | Moderate | 80%+ |
| TJM:O | Male | 20-30 | Good | 60-80% |
| TJM:P | Male | 50+ | Good | 80%+ |

A skeletal element was scored as present if it was represented by a complete or incomplete bone. A few stray human bones were present in some contexts; these are not included in Table 2.

Table 2: Representation of skeletal elements

| Skeletal element | Number represented |
|---|---|
| Skull | 11 |
| Mandible | 9 |
| Cervical vertebrae | 63 |
| Thoracic vertebrae | 119 |
| Lumbar vertebrae | 44 |
| Sacrum | 9 |
| Sternum | 11 |
| L ribs | 98 |
| R ribs | 97 |
| L clavicle | 12 |
| R clavicle | 12 |
| L scapula | 12 |
| R scapula | 12 |
| L humerus | 12 |
| R humerus | 11 |
| L radius | 9 |
| R radius | 10 |
| L ulna | 8 |
| R ulna | 10 |
| L carpals | 41 |
| R carpals | 48 |
| L metacarpals | 46 |
| R metacarpals | 37 |
| L hand phalanges | 27 |
| R hand phalanges | 23 |
| U hand phalanges | 99 |
| L pelvis | 10 |
| R pelvis | 8 |
| L femur | 10 |
| R femur | 9 |
| L patella | 7 |
| R patella | 4 |
| L tibia | 9 |
| R tibia | 9 |
| L fibula | 9 |
| R fibula | 9 |
| L calcaneus | 5 |
| R calcaneus | 9 |
| L talus | 5 |
| R talus | 8 |
| L tarsals* | 19 |
| R tarsals* | 37 |
| L metatarsals | 21 |
| R metatarsals | 33 |
| L foot phalanges | 16 |
| R foot phalanges | 11 |
| U foot phalanges | 40 |

L=left R=right U=unknown side *=excluding talus and calcaneus

Although the bones were generally fairly well preserved, in most cases they were rather fragmented. This severely limited the amount osteometric (particularly craniometric) data which could be obtained. The fragmentary state of the remains appears to be partially due to factors operating during the skeletons' sojourn in the soil (e.g. breakage by tree roots, soil pressure etc) although there are a significant number of fresh-looking breaks, suggesting damage during excavation or storage. The major reason why some skeletons are rather incomplete is damage to the burials by later features.

Ten skeletons are male (including one probable male) and only one is female; this sex imbalance is significant according to the binomial distribution ($p=5.86\times10^{-3}$). If, as seems probable, the interments are of prisoners from the gaol then this might reflect an overall preponderance of male inmates in the gaol, or, perhaps, that more female burials are located in other, unexcavated, areas of the site.

## 2. Metric variation

### (a) Stature

Stature was estimated from long-bone length using the formulae of Trotter & Gleser (1952, 1958). The results are show in Table 3.

Table 3: Stature (cm)

| Skeleton | Sex | Stature |
|---|---|---|
| TJM:B | Female | 151.3 |
| TJM:E | Male | 169.0 |
| TJM:F | Male | 173.7 |
| TJM:G | Male? | 163.6 |
| TJM:I | Male | 167.5 |
| TJM:J | Male | 156.7 |
| TJM:L | Male | 159.6 |
| TJM:N | Male | 176.6 |
| TJM:O | Male | 162.8 |
| TJM:P | Male | 164.2 |

The mean male stature at Launceston is 166.0cm (5'5"), with a range of 156.7-176.6cm (5'2"-5'10"). Archaeological material from London dated 1650-1750 gave a figure for mean male stature of 169.2cm (computed from mean femur length (Pearson & Bell 1919, quoted in Huber 1968) using Trotter & Gleser's (1958) formula). Steegmann (1985) conducted a study of individual stature in 18th century military recruits from all over Great Britain using documentary sources. Accepting war-time recruiting cohorts as unbiased stature estimates of the male population from which they were drawn (which Steegmann argues is probably reasonable) a mean male adult stature of 169.5cm is obtained (for the period 1776-1782). The formulae of Trotter & Gleser give an estimate of maximum adult stature, achieved during early adulthood (probably at about 21 years in the 18th century according to Steegmann's figures). In middle and later years stature declines slightly,

mainly due to degenerative changes in the soft tissues, hence care is required in comparing stature figures from archaeological with those from documentary sources. The mean age of Steegmann's 1776-1782 cohort was 25.2 years, hence his figures should form a valid comparison for archaeological data. The Launceston Castle stature figures are not exceptional in the light of documentary and other archaeological data.

(b) Meric and cnemic indices

The meric index is a measure of the anterio-posterior flattening of the femur in the sub-trochanteric region; cnemic index expresses the transverse flattening of the tibia in the region of the nutrient foramen. The precise significance of these indices is uncertain, although they are probably explicable in terms of adaptation of the bones to mechanical stresses. The indices were taken according to the definitions of Brothwell (1981: 88-89). The results are shown in Table 4.

Table 4: Meric and cnemic indices

| | Meric index | | Cnemic index | |
|---|---|---|---|---|
| Individual | L | R | L | R |
| TJM:B | 84.6 | 76.5 | 59.7 | 65.1 |
| TJM:D | 83.5 | 93.0 | 84.9 | 78.5 |
| TJM:E | 83.5 | 79.6 | 80.3 | - |
| TJM:F | 76.8 | 77.5 | 77.8 | 69.6 |
| TJM:I | 88.2 | 82.3 | 73.6 | 74.9 |
| TJM:J | 71.8 | 74.2 | - | - |
| TJM:L | 80.1 | 77.7 | 69.4 | - |
| TJM:N | 87.3 | 83.2 | 78.4 | - |
| TJM:O | 80.9 | - | - | - |
| TJM:P | 76.7 | 81.1 | 60.8 | 68.5 |

(c) Craniometric measurements

In only one instance could the cranial index be determined: TJM:P had a cranial index of 74.7 (dolichocranic). A few other cranial and mandibular measurements were also taken, the former according to the definitions of Howells (1973) and the latter according to the definitions of Brothwell (1981). The results are listed in the Appendix.

## 3. Non-metric variation

Non-metric traits take the form of minor variations in skeletal form such as presence or absence of bony spurs or foramina. For at least some of these variants there is evidence that they are to some extent inherited, although the causes of many remains obscure.

Thirty-three cranial and 20 post-cranial non-metric traits were scored on a presence/absence basis. Trait definitions were taken mainly from Berry & Berry (1967) and Finnegan (1978).

## Table 5: Cranial non-metric traits

| | TJM:B | TJM:D | TJM:E | TJM:F | TJM:G | TJM:I | TJM:J | TJM:L | TJM:N | TJM:O | TJM:P |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Metopic suture | 0 | 1 | | 0 | P | 0 | 0 | | 0 | 0 | 0 |
| Ossicle at lambda | 1 | | | | 1 | | 1 | | 0 | | 0 |
| Lambdoid ossicle | 1 | 1 | | | 1 | 1 | 1 | | 0 | 1 | 1 |
| Inca bone | 0 | 0 | | 0 | 0 | 0 | 0 | | 0 | 0 | 0 |
| Sagittal ossicle | 0 | 0 | | | 0 | | | | | 0 | |
| Ossicle at bregma | 0 | 0 | | 0 | 0 | | 0 | | 0 | 0 | 0 |
| Coronal ossicle | | 0 | | 0 | 0 | | 0 | 0 | 0 | | 0 |
| Fronto-temporal articulation | | -/0 | | | 0/- | | | | | | 0/- |
| Squamo-parietal ossicle | | -/0 | | | 0/0 | | | | | | 0/- |
| Epipteric bone | | -/1 | | | 0/- | | | | | | |
| Parietal notch bone | -/0 | | | | 0/0 | | 0/0 | | 0/- | | 0/0 |
| Auditory torus | 0/0 | 0/0 | | 0/- | 0/0 | 0/0 | 0/0 | | 0/0 | 0/- | 0/0 |
| Foramen of Hushke | 1/- | 1/1 | | 0/- | 0/0 | 0/0 | 0/0 | | 0/0 | 0/- | 0/0 |
| Ossicle at asterion | | | | | 0/- | | 0/0 | | | | 0/0 |
| Clinoid bridging | | | | | 1/- | | | | | | |
| Palatine torus | 0 | 0 | 0 | | 0 | 0 | 0 | | | 0 | |
| Maxillary torus | 0 | 0 | 0 | | 0 | 0 | | | | 0 | |
| Mastoid foramen extra-sutural | 0/- | 1/1 | | | 0/0 | 1/1 | 1/1 | | -/1 | 1/1 | 0/0 |
| Mastoid foramen absent | -/0 | 0/0 | | | 0/0 | 0/0 | 0/0 | | -/0 | 0/0 | 1/0 |
| Double condylar facet on occipital | -/0 | -/0 | | 0/0 | 0/0 | -/0 | | | | 0/0 | 0/0 |
| Parietal foramen | 1/0 | 0/1 | | 1/- | 1/0 | | 0/1 | 1/1 | 0/1 | 0/0 | 0/1 |
| Accessory infra-orbital foramen | | -/0 | | | 0/0 | | | | | | |
| Zygomatic-facial foramen | 0/1 | 0/1 | | -/1 | 1/1 | -/1 | 1/0 | | 0/0 | 1/1 | 1/- |
| Divided hypoglossal canal | 1/0 | -/1 | | 0/0 | 0/0 | | 0/0 | | 0/0 | 0/0 | 0/0 |
| Posterior condylar canal patent | 0/1 | 0/1 | | 1/- | 0/1 | -/1 | | | 1/1 | 1/1 | 0/0 |
| Precondylar tubercle | 0/0 | | | | 0/0 | | | | | 0/0 | 0/0 |
| Foramen ovale incomplete | | -/0 | | 0/1 | 0/0 | -/1 | -/1 | | 0/- | | |
| Accessory lesser palatine foramen | 1/- | -/0 | | | 1/- | | | | | | 0/1 |
| Supra-orbital foramen complete | 0/- | 0/0 | | P/- | -/0 | 0/0 | 0/0 | -/0 | 1/0 | | 1/1 |
| Maxillary M3 agenesis | 1/0 | -/1 | | | 0/0 | 0/0 | | | 0/- | 0/0 | 0/0 |
| Mandibular M3 agenesis | 0/0 | 0/1 | | | 1/0 | 1/0 | 0/0 | | 0/0 | 0/0 | 0/0 |
| Mandibular torus | 0 | 0 | 0 | | 0 | 0 | 0 | | 0 | 0 | 0 |
| Mylohyoid bridging | | | | | | | | | | | |

1=trait present P=partial trait manifestation 0=trait absent -=no observation possible. Scores for bilateral traits are presented as score for left side/score for right side.

Table 6: Postcranial non-metric traits

| | TJM:B | TJM:D | TJM:E | TJM:F | TJM:G | TJM:I | TJM:J | TJM:L | TJM:M | TJM:N | TJM:O | TJM:P |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fossa of Allen | 0/0 | 1/1 | 0/0 | 1/1 | | 0/0 | 0/0 | 0/- | | 0/0 | 0/- | 0/0 |
| Plaque formation | 0/0 | 0/0 | 0/0 | 0/0 | | 0/0 | -/0 | 0/- | | 1/1 | 0/- | 0/0 |
| Exostosis in trochanteric fossa | 0/- | | 0/0 | 0/0 | | -/0 | 1/1 | 1/- | | 0/1 | 0/- | 1/1 |
| Supra-condyloid process | 0/0 | -/0 | 0/0 | 0/0 | -/0 | 0/0 | 0/0 | 0/0 | | 0/0 | 0/0 | -/0 |
| Septal aperture | 0/0 | -/0 | 0/0 | 1/0 | -/0 | 0/0 | 0/0 | -/0 | | 0/0 | 0/0 | -/0 |
| Acetabular crease | | | 0/- | 0/0 | | 0/0 | 0/0 | 0/- | | 0/0 | 0/- | 0/0 |
| Accessory sacral facets on ilium | | 0/0 | 0/- | 0/0 | | 0/0 | 0/- | | | | | |
| Spina bifida occulta | | 0 | | 0 | | 0 | 0 | | | | | 0 |
| Sixth sacral segment | | | | 0 | | P | 0 | | | | 0 | |
| Acromial articular facet | 0/0 | | 0/- | 0/0 | -/0 | 0/- | 0/- | | 0/- | 0/0 | | 0/0 |
| Os acromiale | 0/0 | | 0/- | 0/0 | -/0 | 0/- | 0/- | | 0/- | 0/0 | | 0/0 |
| Supra-scapular foramen | -/0 | 0/0 | 0/0 | -/0 | 0/0 | -/0 | -/0 | | | 1/- | | 1/- |
| Vastus notch | 1/- | -/1 | | | -/0 | 0/- | 1/1 | 0/0 | 0/- | 0/- | | 1/- |
| Vastus fossa | 1/- | -/0 | | | -/0 | 0/- | 0/0 | 0/0 | 0/- | 0/- | | 0/- |
| Emarginate patella | 0/- | -/0 | | | -/0 | 0/- | 0/0 | 0/0 | 0/- | 0/- | | 0/- |
| Anterior calcaneal facet double | 1/1 | -/1 | 0/0 | -/0 | | 0/0 | | 0/1 | | 0/0 | -/0 | -/1 |
| Anterior calcaneal facet absent | 0/0 | -/0 | 0/0 | -/0 | | 0/0 | | 0/0 | | 0/0 | -/1 | -/0 |
| Atlas facet double | 0/0 | 0/0 | 0/0 | | 0/0 | 1/1 | | | | 1/1 | 0/0 | 0/0 |
| Posterior atlas bridging | 0/0 | 1/0 | 0/0 | | 0/0 | 0/0 | 1/- | | | 0/- | 0/- | |
| Lateral atlas bridging | 0/0 | 0/0 | | | 0/0 | 0/0 | 0/- | | | 0/- | 0/0 | 0/0 |

1=trait present 0=trait absent P=partial trait manifestation -=no observation possible. Scores for bilateral traits are presented as score for left side/score for right side.

The vertebral columns of TJM:D and TJM:I show variations in the areas of transition between vertebral types. In TJM:I there is a supernumary facet for articulation with the occipital bone on the left transverse process of the atlas; the area of the occipital bone with which it would have articulated is missing. The last thoracic vertebra bears lumbar-type facet joints. There is partial sacralisation of the 5th lumbar vertebra via an articulation between its left transverse process and the left sacral ala. Lipping and porosis suggestive of degenerative changes are present, implying that movement was possible at this articulation.

TJM:D shows what might be classified as a cervico-thoracic transition variation. The right transverse process of the last cervical vertebra is thickened and elongated; the left is of more normal appearance but bears an unfused epiphysis near its tip. There is also a projection on the left side of the vertebral body bearing an unfused epiphysis at its tip. The morphology of the left transverse process and the left side of the centrum is suggestive of articulations for a true cervical rib, although the rib itself was not recovered. The enlarged right transverse process might be considered a rudimentary cervical rib (Schmorl & Junghanns 1971: 57-8).

## 4. Pathology

(a) Dental pathology

(i) Dental caries. Dental caries was scored as present or absent in each tooth, and as present or absent in individuals with one or more fully erupted teeth available for study. On this basis 9 out of 11 individuals for whom the condition could be scored showed dental caries.

Table 7: Distribution of dental caries

MAXILLA

| | LEFT | | | | | | | | | | RIGHT | | | | | | un |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | M3 | M2 | M1 | PM2 | PM1 | C | I2 | I1 | I1 | I2 | C | PM1 | PM2 | M1 | M2 | M3 | ident. |
| Teeth | 1 | 5 | 5 | 7 | 5 | 7 | 3 | 5 | 6 | 3 | 6 | 6 | 6 | 3 | 5 | 2 | 0 |
| Carious | 1 | 2 | 2 | 2 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 1 | 1 | 2 | 1 | 0 | 0 |
| Teeth | 4 | 5 | 4 | 5 | 5 | 6 | 5 | 3 | 5 | 7 | 6 | 7 | 6 | 6 | 5 | 2 | 2 |
| Carious | 2 | 0 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 1 | 2 |

MANDIBLE

Of a total of 158 teeth 28 (17.7%) are carious

Although it must be considered a multifactorial disease, many studies have shown a strong correlation between caries rates and consumption of carbohydrates, particularly sugars. During the Post-Mediaeval period sugar became more widely available in England than it had been hitherto; in addition food became softer and more refined (Hardwick 1960; Moore & Corbett 1978). Consistent with the above rates of dental caries generally rose and dental attrition fell compared with earlier periods. Estimates of caries rates with respect to total teeth in skeletal material dating from 1600-1800 vary from 10-20% (Hardwick 1960; Brothwell 1959); thus the figures for Launceston Castle cannot be considered atypical for the period. Dental attrition tends to inhibit formation of caries cavities on the occlusal surfaces of the teeth by obliterating the pits and fissures which may act as foci for caries, and exerting a cleansing effect on the teeth. Moore & Corbett (1978) noted an increasing predilection for occlusal caries during the Post-Mediaeval period and associated it with a reduction in dental attrition. In the Launceston material many of the caries cavities are rather large, destroying considerable areas of dental crown; in such cases it is impossible to determine their point of origin on the tooth. In cases where cavities are sufficiently small to determine their initial location many were seem to have originated on the occlusal surface (Plate 1).

Although difficulties in accurately determining age in adults make firm conclusions impossible, the impression was that the rate of dental wear varies markedly between individuals, perhaps suggesting marked differences in the consistency of the diet between individuals buried at Launceston.

(ii) Ante-mortem tooth loss. This was scored on a presence-absence basis for each erupted tooth position, and as present or absent in individuals with one or more tooth positions available for study. On this basis 8 out of 10 individuals showed ante-mortem tooth loss.

Table 8: Distribution of ante-mortem tooth loss

| | MAXILLA | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | LEFT | | | | | | | | RIGHT | | | | | | | |
| | M3 | M2 | M1 | PM2 | PM1 | C | I2 | I1 | I1 | I2 | C | PM1 | PM2 | M1 | M2 | M3 |
| Tooth posits. | 5 | 8 | 8 | 9 | 9 | 9 | 7 | 7 | 8 | 7 | 8 | 8 | 8 | 7 | 7 | 4 |
| A-m loss | 3 | 3 | 3 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 | 3 | 1 | 1 |
| Tooth posits. | 7 | 9 | 9 | 9 | 9 | 9 | 7 | 7 | 9 | 9 | 8 | 9 | 9 | 9 | 9 | 7 |
| A-m loss | 2 | 3 | 4 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 3 | 3 | 4 | 2 |
| | MANDIBLE | | | | | | | | | | | | | | | |

Of a total of 254 tooth positions, 72 (28.3%) show ante-mortem tooth loss

Major causes of ante-mortem tooth loss include dental caries and diseases of the periodontal tissues. Brothwell (1959) found a rate of ante-mortem tooth loss of about 13% with respect to total tooth positions in skeletal material dated 1600-1800AD. Against this the level in the Launceston material looks rather high, however little can be inferred from this given the small size of the assemblage. The rate of ante-mortem tooth loss at Launceston is also inflated by the presence of 2 edentulous individuals (TJM:J and TJM:P).

(iii) Periapical dental abscesses. These are scored in terms of number of affected tooth positions in an analogous fashion to ante-mortem tooth loss. Five out of 10 individuals for whom observation could be made showed one or more periapical dental abscesses.

Table 9: Distribution of periapical abscesses

| | MAXILLA | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | LEFT | | | | | | | | RIGHT | | | | | | | |
| | M3 | M2 | M1 | PM2 | PM1 | C | I2 | I1 | I1 | I2 | C | PM1 | PM2 | M1 | M2 | M3 |
| Tooth posits. | 5 | 8 | 8 | 9 | 9 | 9 | 7 | 7 | 8 | 7 | 8 | 8 | 8 | 7 | 7 | 4 |
| Alv. abscesses | 0 | 0 | 0 | 1 | 2 | 2 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 2 | 1 | 0 |
| Tooth posits. | 7 | 9 | 9 | 9 | 9 | 9 | 7 | 7 | 9 | 9 | 8 | 9 | 9 | 9 | 9 | 7 |
| Alv. abscesses | 1 | 0 | 2 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| | MANDIBLE | | | | | | | | | | | | | | | |

Of a total of 254 tooth positions, 18 (7.1%) show a periapical abscess.

Of the 18 abscess cavities, 8 were at the apices of teeth whose pulp cavities had been exposed to infection by dental caries, one was situated as the base of a tooth whose pulp cavity appeared to

have been exposed by excessive wear and in the remaining 9 cases cavities were located at sockets whose teeth were lost post-mortem so their cause was unclear.

(iv) Dental calculus. Dental calculus is a concretion on the teeth consisting of calcium salts and, in life, organic matter in which flourish numerous bacteria. It may be considered as mineralised dental plaque and is associated with poor oral hygiene. Of 8 individuals who could be scored for the condition 6 showed it to grade I, and 1 to grade II, on Dobney & Brothwell's (1987) scale. Hillson (1979) has emphasised that while a low oral pH favours the development of caries cavities, calculus tends to form under conditions of high pH. In this light it is interesting to note that in TJM:D the mandibular left M1 shows gross approximal caries and the occlusal surface is mainly covered by calculus deposits, as to a lesser extent is the crown of the neighbouring M2. In addition, dental calculus in the Launceston assemblage is generally confined to the cervical regions of the tooth crowns; in TJM:D the ante-mortem loss of the occlusal partners of the above teeth facilitated the accretion of deposits on their occlusal surfaces.

(v) Periodontal disease. TJM:E and TJM:I show pitting and porosis of the interdental septa, together with some probable alveolar resorbtion, suggestive of periodontal disease. Periodontal disease is an inflammation of the gums and other periodontal tissues associated with poor oral hygiene.

(vi) Dental enamel hypoplasias. These appear macroscopically as linear bands of depressed enamel or areas of pitting (Sarnat & Schour 1941). They are associated with a wide variety of stressors including infectious diseases and nutritional deficiencies (Pindborg 1970: 138-210). The anterior dentition appears to be more susceptible to hypoplasias than the posterior teeth (Goodman & Armelagos 1985). Eight individuals had anterior dentition in sufficiently unworn state for hypoplasias to be scored; of these 4 showed the presence of defects. The location of defects on the tooth was used to estimate the age at which the individual suffered the stress episode which gave rise to the hypoplasia, using the methodology of Goodman et al. (1980).

Table 10: Dental enamel hypoplasias

| Burial | Defect(s) | Approximate age when formed |
|---|---|---|
| TJM:B | Pitted area/several heavy lines | 6 months-2.5 years |
| TJM:I | 3 lines | 2.0, 2.5, 2.8 years |
| TJM:N | 3 lines | 3.4, 3.8, 4.3 years |
| TJM:O | 1 line | 4.1 years |

The severity of the defects on the anterior dentition of TJM:B is notable, particularly the areas of pitted, malformed enamel on the canine teeth (Plate 2), suggesting a prolonged and perhaps severe period of disease and/or poor nutrition. The defective enamel seems to have precipitated the formation of a caries

cavity on the lingual surface of the crown of the left mandibular canine.

The crowns of the 1st permanent molars of TJM:G show rather more than the usual numbers of pits and fissures (particularly the mandibular molars). It may be that these are simply non-pathological variants, but it is more likely that they represent dental enamel hypoplasias, in which case the stress episode(s) which gave rise to them occurred around time of birth.

(vii) Miscellaneous. TJM:O has a large cavity (approximately 1cm internal diameter) in the anterior part of each maxilla, continuous with, and partially destroying the sockets of the incisor and canine teeth (Plate 3). The internal surfaces of the cavities are slightly roughened and sclerotic. There is slight cavitation in the root of the left maxillary medial incisor. The lateral maxillary incisors are missing. When the canines and medial incisors are placed in their sockets (Plate 4) their crowns almost touch, leaving insufficient room for lateral incisors. There is a small socket filled with bone just distal to each canine; these are probably sockets for deciduous canines whose exfoliation was delayed, forcing the permanent canines to erupt mesial to their usual positions.

One possible explanation for the cavities in the maxillae is that they are a consequence of periapical abscessing. The crowns of the incisors and canines are intact, hence the cavities cannot be a result of alveolar infection via these teeth, although one could argue that they may have originated via infected lateral incisors which were both subsequently exfoliated. Several factors argue against this explanation: there seems to be insufficient space in the maxillae for the lateral incisors, suggesting that they may have been congenitally absent or at least failed to erupt, and the very large size of the lesions and, more particularly, their bilaterally symmetrical nature is atypical of cavities associated with periapical infection.

It seems possible that the maxillary lateral incisors were impacted, or, more likely that they were congenitally absent. When a tooth fails to develop a primordial cyst may form via degeneration of the dental follicle (Shafer et al. 1983: 259); it may thus be that the cavities in the maxillae of TJM:O represent primordial cysts associated with the degeneration of the enamel organs which would have formed the crowns of the lateral incisors. A dentigerous cyst originates after the crown of a tooth has formed as a result of accumulation of fluid between the reduced enamel epithelium and the tooth crown; dentigerous cysts are associated with an unerupted or impacted teeth (Shafer et al. 1983: 260-261). Hence if one accepts the scenario that the lateral incisors failed to erupt (and were resorbed or lost post-mortem) the cavities might represent dentigerous cysts.

Thus, although the most likely of the above diagnoses of the cavities in the maxillae of TJM:O is perhaps that they are primordial cysts, a firm diagnosis remains elusive.

The hard palate of TJM:E has a small area of increased pitting on its inferior surface and a similar-sized (approx. 2x4mm) pitted area on the superior surface; these are indicative of small, localised areas of infection.

The right third molar in a mandible from context ZJ 20 684, present as a stray bone in burial TJM:E, is erupting at an angle 45 degrees mesially and is impacted against the distal root of the neighbouring second molar. The left M3 is also angled 45 degrees mesially but has erupted fully. The crown of the left M2 has suffered destruction by dental caries - only one root remains in the socket. It is probable that this destruction of the crown of the left M2 allowed the left M3 to erupt despite its abnormal orientation. The maxillae associated with this mandible show bilateral absence of the third molars.

(b) Arthropathies

Degenerative joint disease may be divided into two categories: that affecting the vertebral bodies is termed osteophytosis and that affecting the other joints is termed osteoarthritis (following Collins 1949). Both human and animal studies have shown that mechanical stress is an important factor in the aetiology of degenerative joint disease. The most usual cause seems to be repeated minor traumata as might result from day to day activities; this leads to degeneration of the intervertebral disc or joint cartilage with subsequent macroscopic bony changes, including marginal lipping and joint surface irregularities. Factors implicated in the prevalence of degenerative joint disease include individual age and the degree of physical stress on the joints in life.

Degenerative joint disease was distinguished from other arthropathies using criteria described by Steinbock (1976), Ortner & Putschar (1985) and Rogers et al. (1987).

The presence of osteophytosis and osteoarthritis was scored as grade I, II or III with reference to the scheme of Sagar (1969, reproduced in Brothwell 1981: Fig. 6.9). The results (adults only) are shown below).

Table 11: Osteophytosis: maximum severity by individuals

| Maximum severity | | | |
|---|---|---|---|
| 0 | I | II | III |
| 4 | 2 | 3 | 2 |

Table 12: Osteophytosis: prevalence by vertebrae

| Cervical | | | | Thoracic | | | | Lumbar | | | | Total | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 0 | I | II | III | 0 | I | II | III | 0 | I | II | III | 0 | I | II | III |
| 29 | 4 | 10 | 1 | 58 | 30 | 5 | 1 | 10 | 16 | 1 | 0 | 97 | 50 | 16 | 2 |

Table 13: Osteoarthritis: maximum severity by individual

| Maximum severity | | | |
|---|---|---|---|
| 0 | I | II | III |
| 4 | 3 | 2 | 2 |

Table 14: Osteoarthritis

| | | Severity | | |
|---|---|---|---|---|
| Skeletal element | 0 | I | II | III |
| L mandibular condyle | 5 | 0 | 0 | 0 |
| R mandibular condyle | 5 | 0 | 0 | 0 |
| L ribs | 73 | 8 | 3 | 3 |
| R ribs | 72 | 10 | 3 | 1 |
| Cervical vertebrae | 47 | 1 | 4 | 2 |
| Thoracic vertebrae | 81 | 6 | 3 | 3 |
| Lumbar vertebrae | 29 | 2 | 1 | 1 |
| L medial clavicle | 9 | 1 | 0 | 0 |
| L lateral clavicle | 3 | 1 | 1 | 1 |
| R medial clavicle | 9 | 0 | 1 | 0 |
| R lateral clavicle | 3 | 1 | 1 | 2 |
| L glenoid cavity | 5 | 2 | 0 | 1 |
| R glenoid cavity | 9 | 1 | 0 | 0 |
| L proximal humerus | 9 | 0 | 0 | 0 |
| R proximal humerus | 9 | 0 | 0 | 0 |
| L distal humerus | 7 | 0 | 0 | 0 |
| R distal humerus | 10 | 0 | 0 | 0 |
| L proximal radius | 7 | 0 | 0 | 0 |
| R proximal radius | 6 | 0 | 0 | 0 |
| L distal radius | 8 | 0 | 0 | 0 |
| R distal radius | 7 | 0 | 0 | 0 |
| L proximal ulna | 6 | 0 | 0 | 0 |
| R proximal ulna | 8 | 0 | 0 | 0 |
| L distal ulna | 7 | 0 | 0 | 0 |
| R distal ulna | 8 | 0 | 0 | 0 |
| L carpals | 34 | 3 | 0 | 2 |
| R carpals | 39 | 2 | 2 | 2 |
| L metacarpals | 40 | 0 | 2 | 0 |
| R metacarpals | 32 | 1 | 1 | 0 |
| L hand phalanges | 22 | 2 | 2 | 1 |
| R hand phalanges | 16 | 2 | 4 | 1 |
| U hand phalanges | 93 | 0 | 0 | 0 |
| L acetabulum | 7 | 2 | 0 | 0 |
| R acetabulum | 5 | 2 | 0 | 0 |
| L proximal femur | 9 | 0 | 0 | 0 |
| R proximal femur | 8 | 0 | 0 | 0 |
| L distal femur | 9 | 0 | 0 | 0 |
| R distal femur | 8 | 0 | 0 | 0 |
| L patella | 7 | 0 | 0 | 0 |
| R patella | 3 | 0 | 0 | 0 |
| L proximal tibia | 7 | 0 | 0 | 0 |
| R proximal tibia | 6 | 0 | 0 | 0 |
| L distal tibia | 7 | 0 | 0 | 0 |
| R distal tibia | 7 | 0 | 0 | 0 |
| L proximal fibula | 3 | 0 | 0 | 0 |
| R proximal fibula | 6 | 0 | 0 | 0 |
| L distal fibula | 6 | 0 | 0 | 0 |
| R distal fibula | 6 | 0 | 0 | 0 |
| L calcaneus | 5 | 0 | 0 | 0 |
| R calcaneus | 8 | 0 | 0 | 0 |
| L talus | 5 | 0 | 0 | 0 |
| R talus | 7 | 0 | 0 | 0 |

| Skeletal element | 0 | Severity I | II | III |
|---|---|---|---|---|
| L tarsals* | 19 | 0 | 0 | 0 |
| R tarsals* | 33 | 0 | 0 | 0 |
| L metatarsals | 21 | 0 | 0 | 0 |
| R metatarsals | 33 | 0 | 0 | 0 |
| L foot phalanges | 15 | 0 | 0 | 0 |
| R foot phalanges | 11 | 0 | 0 | 0 |
| U foot phalanges | 40 | 0 | 0 | 0 |

L=left R=right U=unknown side *=excluding talus and calcaneus

(c) Trauma

(i) Fractures. Three individuals, TJM:I, TJM:J & TJM:P, show a total of 6 fractures, a prevalence with respect to total identifiable bones (calculated as described for Table 2) of 6/1178.

TJM:I shows 2 rib fractures and a fracture to the base of the right 1st metacarpal. All are firmly healed. The metacarpal fracture is oblique with a fissure running across the dorsal margin of the proximal joint surface so that the articular facet is displaced in a volar/distal direction (Plate 5). There also seems to be another fracture line across the volar edge of the articular facet. Fractures of the 1st metacarpal involving the proximal articular surface are termed Bennett's fractures. They may be a consequence of a blow to the tip of the thumb and tend to be more frequent in the dominant hand (Lipscombe 1982: 760-762).

TJM:J shows healed fractures of a left and a right rib, both situated near their sternal ends. The skeleton of this individual was rather osteoporotic (see below). The rib fractures are united by callus composed of new, woven bone, suggesting that they occurred not long before death. It thus seems possible that loss of bone mineral was a predisposing factor to these fractures.

TJM:P shows an oblique fracture of the middle third of the left clavicle. Although it is firmly united the bone is 1cm shorter that its counterpart on the right hand side. The line of fracture is clearly visible on the radiograph (Plate 6), as is the over-riding of the fractured ends. Clavicle fractures of this sort are frequently a result of a fall on an outstretched hand or on the point of the shoulder.

(ii) Schmorl's nodes. An intervertebral disc consists of a tough outer layer (the annulus fibrosus) surrounding an inner core (the nucleus pulposus) which, until early adulthood, is composed of semi-gelatinous material. In younger individuals excessive compression of the spine (as might occur due to heavy lifting) may result in extrusion of material from the nucleus pulposus into the adjacent vertebral body. The bony manifestation of this is a bony pit or cleft - the Schmorl's node. In some individuals congenital weakness in the cartilage plate of the vertebral body may increase the likelihood of formation of Schmorl's nodes, but there is no doubt that a single trauma may rupture a healthy disc (Schmorl & Junghanns 1971: 158-168).

Table 15: Distribution of Schmorl's nodes

| Individual | No of affected vertebrae | No of nodes (i=inferior (s=superior surface) |
|---|---|---|
| TJM:J | 1 lumbar | 1(i) |
| TJM:N | 2 lumbar | 2(1i, 1s) |
| TJM:O | 7 thoracic | 8(7i, 1s) |

(iii) Soft tissue trauma. The left femur of TJM:E shows a smooth exostosis on its lateral surface just superior to the greater trochanter. It is about 5cm long and rises about 1cm proud of the surface of the normal cortex. This probably represents myositis ossificans circumscripta. Myositis ossificans is a soft tissue ossification; although it may arise spontaneously, in about 60-75% of cases there is a history of single or repeated traumata (Resnick & Niwayama 1988: 4247-4253). The calcified fragment often initially lies near, but is separate from, a bone but subsequently merges with it (ibid.). The lateral surface of the proximal femur is an area exposed to trauma and hence is a not infrequent site for the lesion (discussion in Norman & Dorfman 1970).

(d) Cribra orbitalia

Cribra orbitalia takes the form of small pits or perforations in the orbital roofs. Of the 9 individuals who could be scored for the condition 1 (TJM:D) showed lesions, of Brothwell's (1981: Fig. 6.17) cribriotic type. In addition this individual shows pitting and some striations composed of coalesced apertures on the outer table of both parietal bones (the inner table is normal). The lesions are mainly confined to the area between the parietal eminences and the sagittal suture. It seems probable that these lesions represent porotic hyperostosis of the cranial vault - cribra cranii. It seems probable that both cribra orbitalia and cribra cranii share a common aetiology: iron deficiency anaemia (Hengen 1971; Stuart-Macadam 1989). It also seems probable that lesions in the orbital roofs may represent initial changes whereas severe or prolonged anaemia leads to additional lesions on the cranial vault (Hengen 1971; Stuart-Macadam 1989). In addition to deficient dietary intake of iron, anaemias may be caused by gut parasites, frequent in the unhygienic living conditions which were doubtless common in antiquity.

(e) Infections

(i) Non-specific lesions. When it is not possible to identify a particular micro-organism as responsible for an inflammation it is termed a non-specific infection. In the Launceston Castle assemblage 2 types of non-specific infection are found: periostitis, inflammation of the periosteum which results in laying down of new bone on the underlying cortex, and osteomyelitis, inflammation involving the marrow cavity (Steinbock 1976: 60).

The left tibia of TJM:I bears a smooth swelling on its anterior

border about one third of the way from the distal end; there is slight pitting and striation of the bone surface in the area of the lesion. The radiograph shows the swelling to be deposition of new bone upon the underlying cortex, the endosteal surface is normal. The left fibula shows a deposit of woven bone on its posterior surface, just above the distal end.

The right tibia of TJM:P shows slight, well remodelled deposits of pitted bone, mainly confined to the lateral surface, as does the midshaft area of the left fibula.

The above lesions probably represent periostitis. Periostitis may be a consequence of systemic disease or of local trauma and infection. The localised nature of the lesions in the 2 cases above suggest the latter as the more likely cause; the swelling on the sub-cutaneous surface of the tibia of TJM:I is probably an ossified haematoma, consistent with the thesis of local trauma. Documentary evidence suggests that chronic infection of the lower legs was a common health problem in the 18th century among the "lower classes of the community" (Loudon 1981), however the slight nature of the lesions in the Launceston material should be emphasised.

The right humerus of TJM:J is irregularly swollen over much of its length (Plate 7), only the epiphyses and the proximal metaphysis remain unaffected. Near the middle of the shaft the bone is rather roughened but elsewhere it is pitted but smooth. The cortex is pierced by 2 small holes in the area of the deltoid tuberosity. A post-mortem break in the mid-shaft area shows the medullary cavity to be narrowed by cancellous bone. These lesions probably represent osteomyelitis. In osteomyelitis the pathological process is one of bone destruction, pus formation and bone repair, hence the enlarged, deformed appearance of the osteomyelitic bone. Abscess cavities develop in the bone interior and in general eventually discharge into the surrounding soft tissues via a sinus in the cortex. It is likely that the 2 holes observed in the cortex of the humerus of TJM:J represent such sinuses. Osteomyelitis may arise from infection following local bony or soft tissue injury, or via the bloodstream from a primary infection elsewhere in the body. In the latter case the great majority of cases start in childhood or adolescence, although the untreated infection can persist for decades so in archaeological assemblages adults may show lesions from disease initiated in childhood (Ortner & Putschar 1985: 104f; Steinbock 1976: 60f). X-ray revealed no evidence for any bony trauma which might have initiated the infection. Most of the original cortex seems to have been removed by remodelling, although traces of it were visible in places on the radiograph. Both the radiographic and gross appearance of the bone are suggestive of disease of long-standing.

(ii) Tuberculosis. The upper and much of the lower surface of the body of the 5th lumbar vertebra of TJM:O has been destroyed ante-mortem, as have the pedicles, so that the neural arch is separate from the remainder of the bone. There is a little reactive bone on the anterior wall of the centrum. The lower parts of the body of the 4th lumbar vertebra show extensive destruction, and there is a small erosion on the superior surface of the body of the first sacral segment. The walls of these

lesions are irregular and of smooth, slightly sclerotic trabecular bone. In all cases bone regeneration is negligible. All the lumbar vertebrae are fragmentary and poorly preserved. The remainder of the complete vertebral column is normal. The left innominate shows deposition of woven bone over its general surface, particularly in the region of the sacro-iliac joint. The involvement of the sacro-iliac joint itself cannot be assessed due to post-mortem damage but the distribution of periostitis over the ilium suggests that it was unlikely that the joint was free from disease.

There is destruction of the 5th metacarpo-phalangeal joint (Plate 8), the surfaces of the lytic area being of fairly smooth, slightly sclerotic trabecular bone, and there is negligible new bone formation. In addition 2 intermediate phalanges and the left 1st metacarpal are markedly shortened, and 3 proximal phalanges are rather swollen and distorted, particularly towards their proximal ends (Plate 8).

The spinal lesions are strongly suggestive of tuberculosis. Skeletal tuberculosis is generally a result of secondary infection from lesions in the soft tissues, particularly the lungs, the bacilli reaching the bone via the bloodstream. The bacilli tend to locate themselves in areas of haemopoietic marrow which have high metabolic rates; thus the vertebral bodies are a favoured site for lesions. The lack of bony regeneration, sinus formation or sequestra (cf TJM:J, above) argue for tuberculosis against a differential diagnosis of osteomyelitis (Steinbock 1976: 175f; Ortner & Putschar 1985: 144f). The infection of the left ilium is likely to be an extension of the lumbo-sacral focus (Ortner & Putschar 1985: 149). The tubular bones of the hands are also fairly frequent sites for tuberculous infection, most particularly during childhood (Resnick & Niwayama 1988: 2677). In these bones a focus will rapidly occupy the whole shaft, leading to a fusiform swelling; destruction of the growth plate by the infection may lead to marked shortening and deformity of the bone on healing. Hence the changes in the hand bones are probably also a result of tuberculous infection, however involvement of the metacarpo-phalangeal joint is atypical, although it can occur (Poppel et al. 1953; Ortner & Putschar 1985: Table 5). The shortening of some of the tubular bones of the hands suggests disease contracted during childhood and the unremodelled nature of the periosteal bone on the left ilium suggests that the tuberculous infection was also active at time of death.

Pulmonary tuberculosis is population density dependent disease, and as such it may have assumed greater importance with increasing urbanisation in the later Mediaeval and Post-Mediaeval periods (discussion in Manchester 1984). "Correlations between tuberculosis and poverty, insanitary living conditions, the absence of health care, and inadequate diet are very high, while the disease has generally been more active in urban than in rural areas" (McGrew 1985: 338).

(f) Osteoporosis

All the bones of TJM:J and TJM:L are rather light, radiolucent and have thin cortices. In TJM:J there are 2 healed rib fractures (section 4c(i)), probably associated with the general depletion of bone mineral in this skeleton. In modern populations bone mass tends to decrease after the 4th decade; the point at which loss of bone mineral is classified as osteoporosis is arbitrary (Kelsey 1987). Although bone loss with advancing age is more pronounced in females, the phenomenon also occurs in males. TJM:J was the skeleton of an elderly male but in TJM:L age at death was uncertain (although this male was fully adult). Prolonged immobility may result in significant loss of bone mineral (Donaldson et al. 1970), hence if either or both these individuals had been bedridden for an extended period prior to death it may have been a contributory factor in the loss of bone mineral displayed by their skeletal remains.

## 5. Summary & discussion

Of 14 eighteenth century inhumations excavated from Launceston Castle, thought to represent inmates from when the Castle was used as a gaol, 12 were examined by the present writer and 2 were the subject of a previous report (Keepax 1973). The preservation of the bone was generally good, although most burials were somewhat fragmentary. Eleven burials were male, 2 female and one unsexable, ages at death ranged from adolescent (about 15-18 years) to elderly (probably 60+ years). Stature was unremarkable for the period. Three individuals showed healed fractures, all of the type more probably associated with accidental injury rather than inter-personal violence; in one individual osteoporosis was probably a predisposing factor to the fractures. One probable case of tuberculosis, common in the 18th century, particularly among those with poor living conditions, was identified.

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# PLATES

Plate 1: The mandible of TJM:0 showing caries on the enamel crowns of 5 of the molar teeth

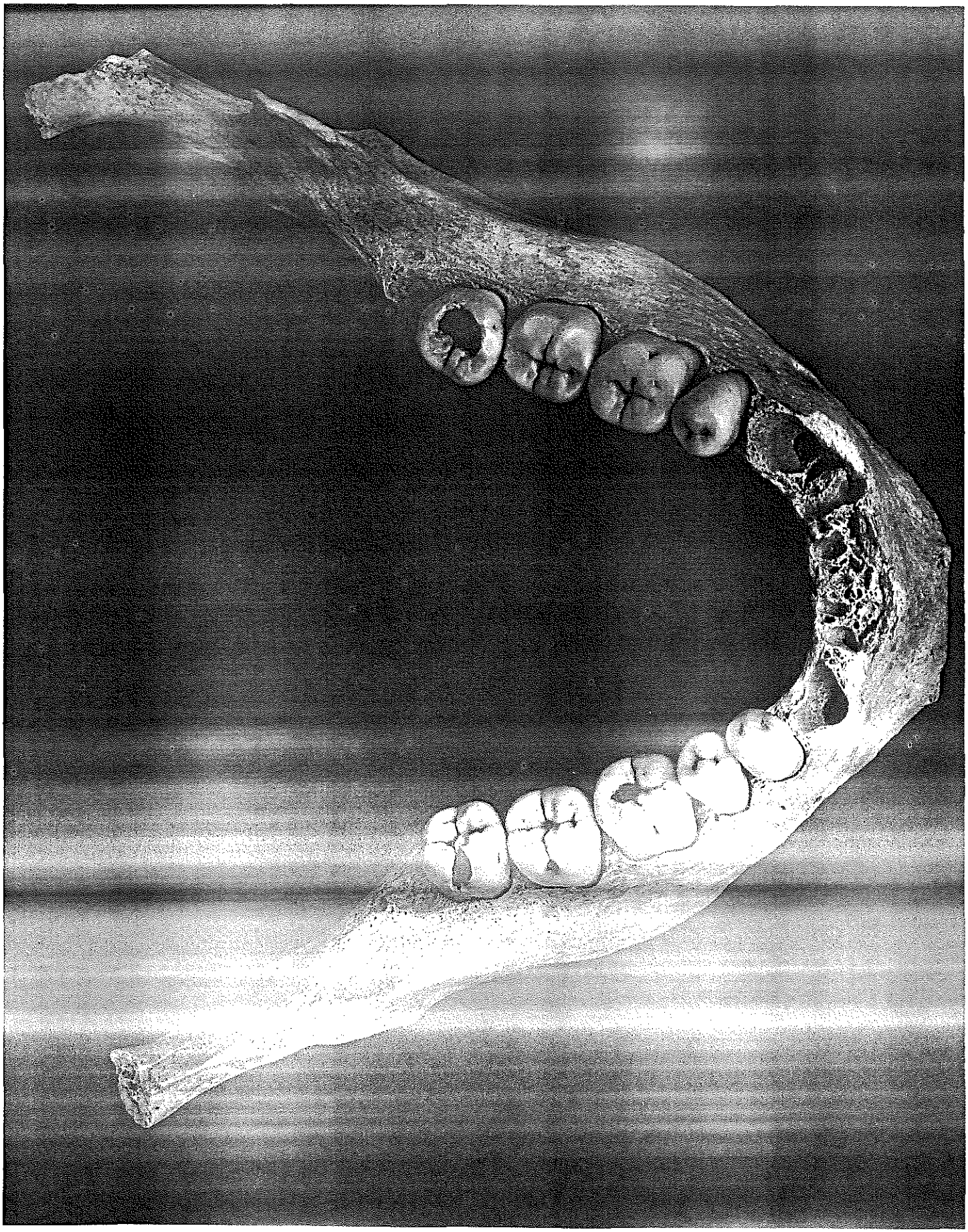


Plate 2: The mandibular canines of TJM:B (lingual view) showing severe enamel hypoplastic defects

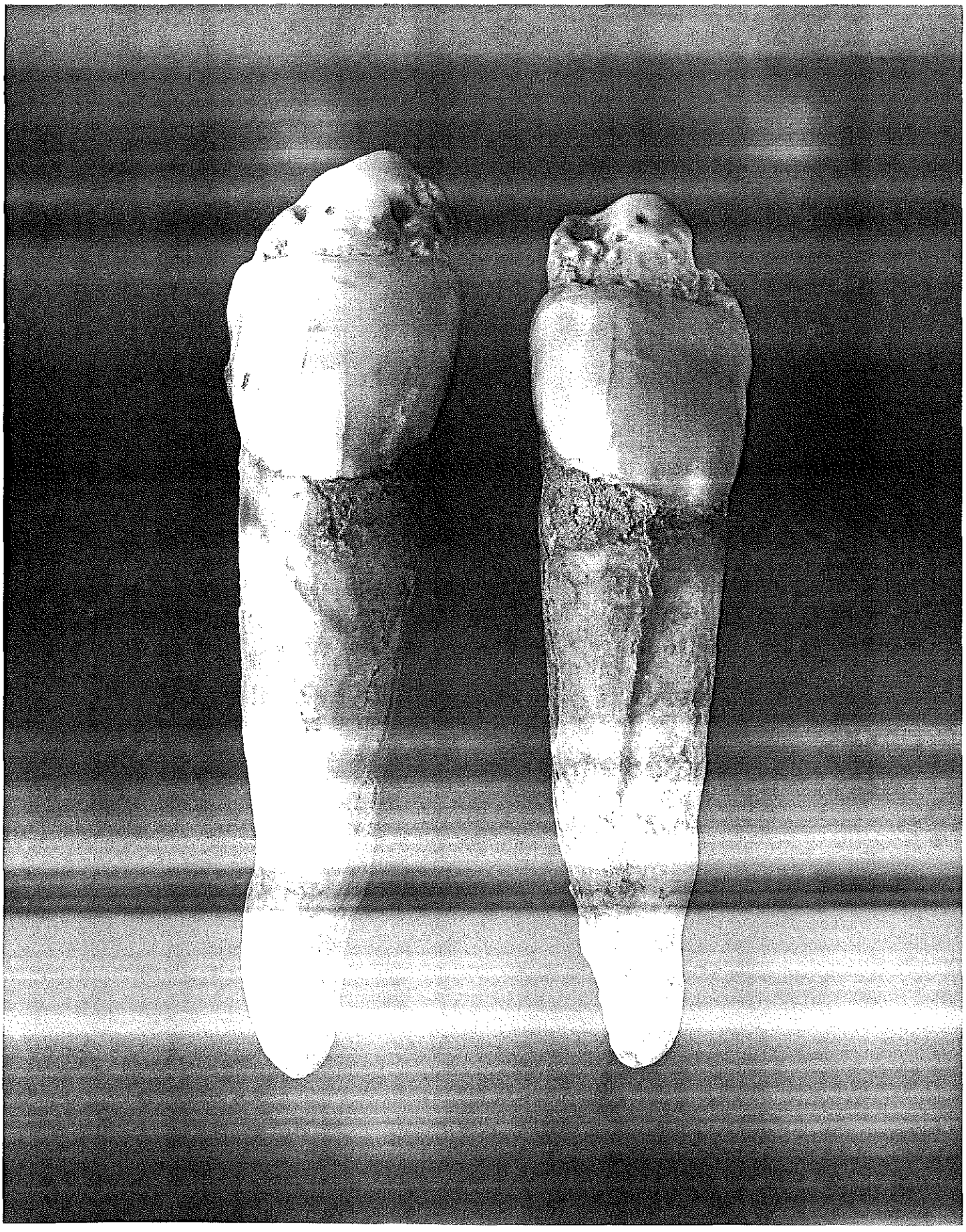


Plate 3: The maxillae of TJM:O

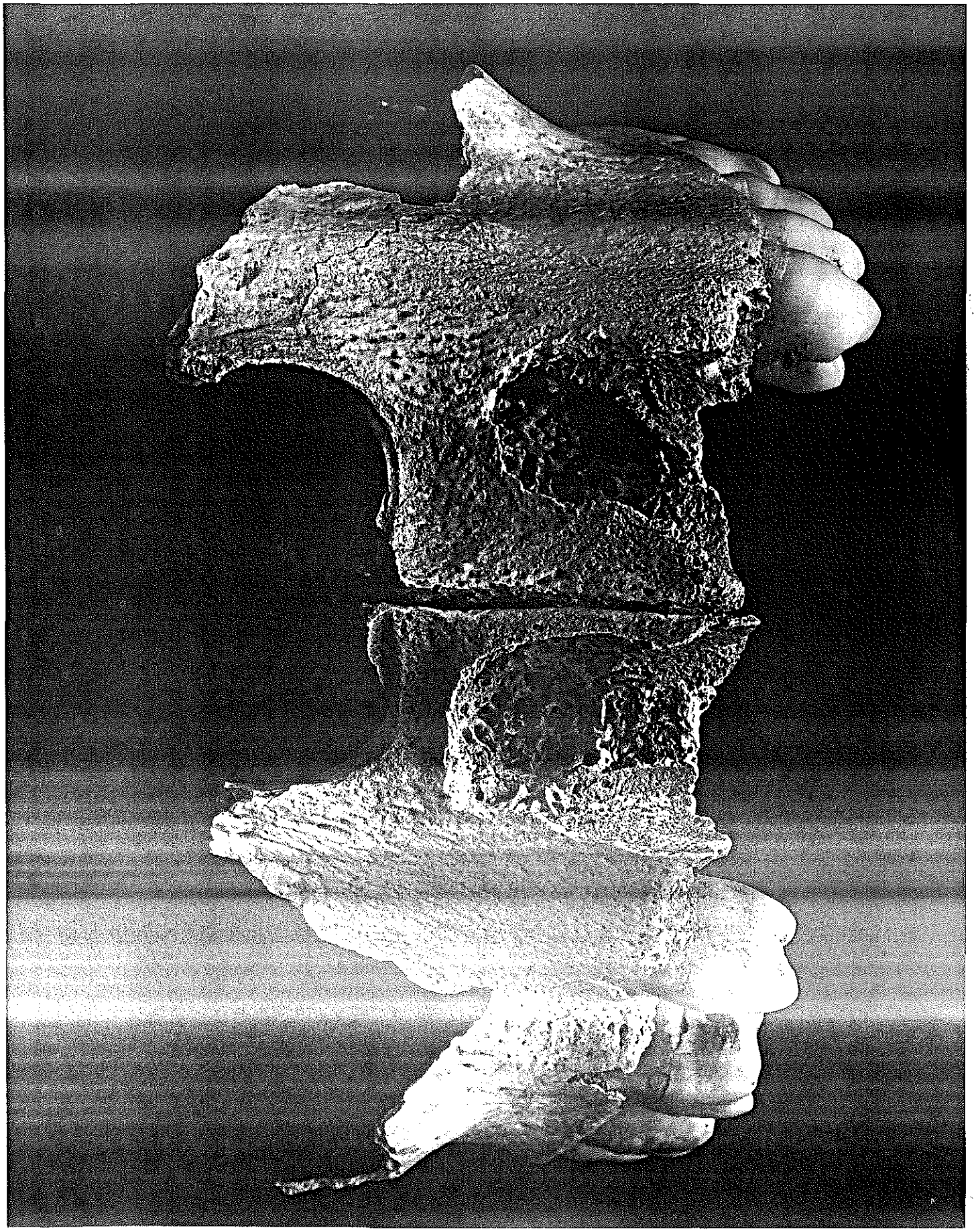


Plate 4: The maxillae of TJM:0 with the anterior teeth in position

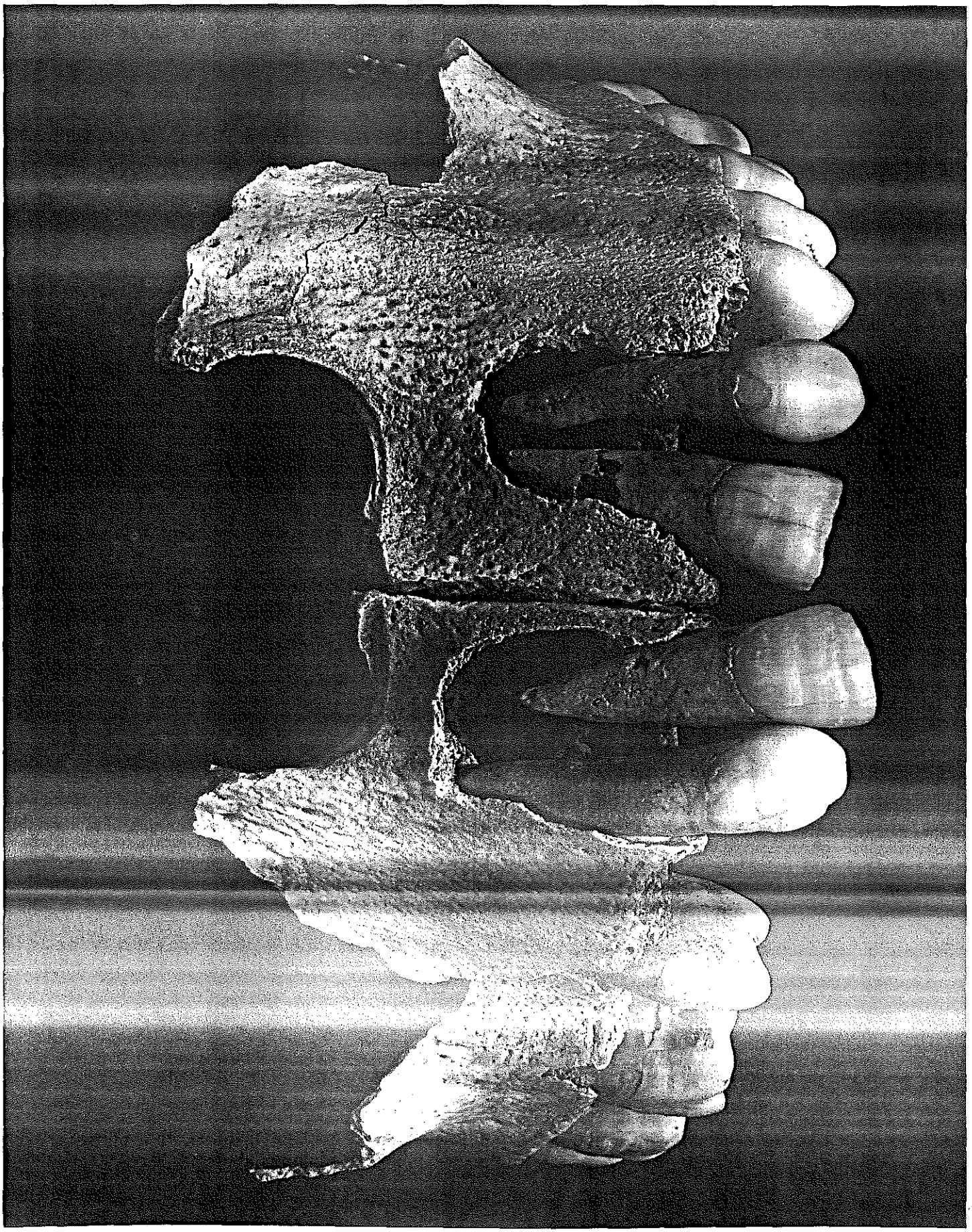


Plate 5: The right 1st metacarpal of TJM:I showing a healed Bennett's fracture

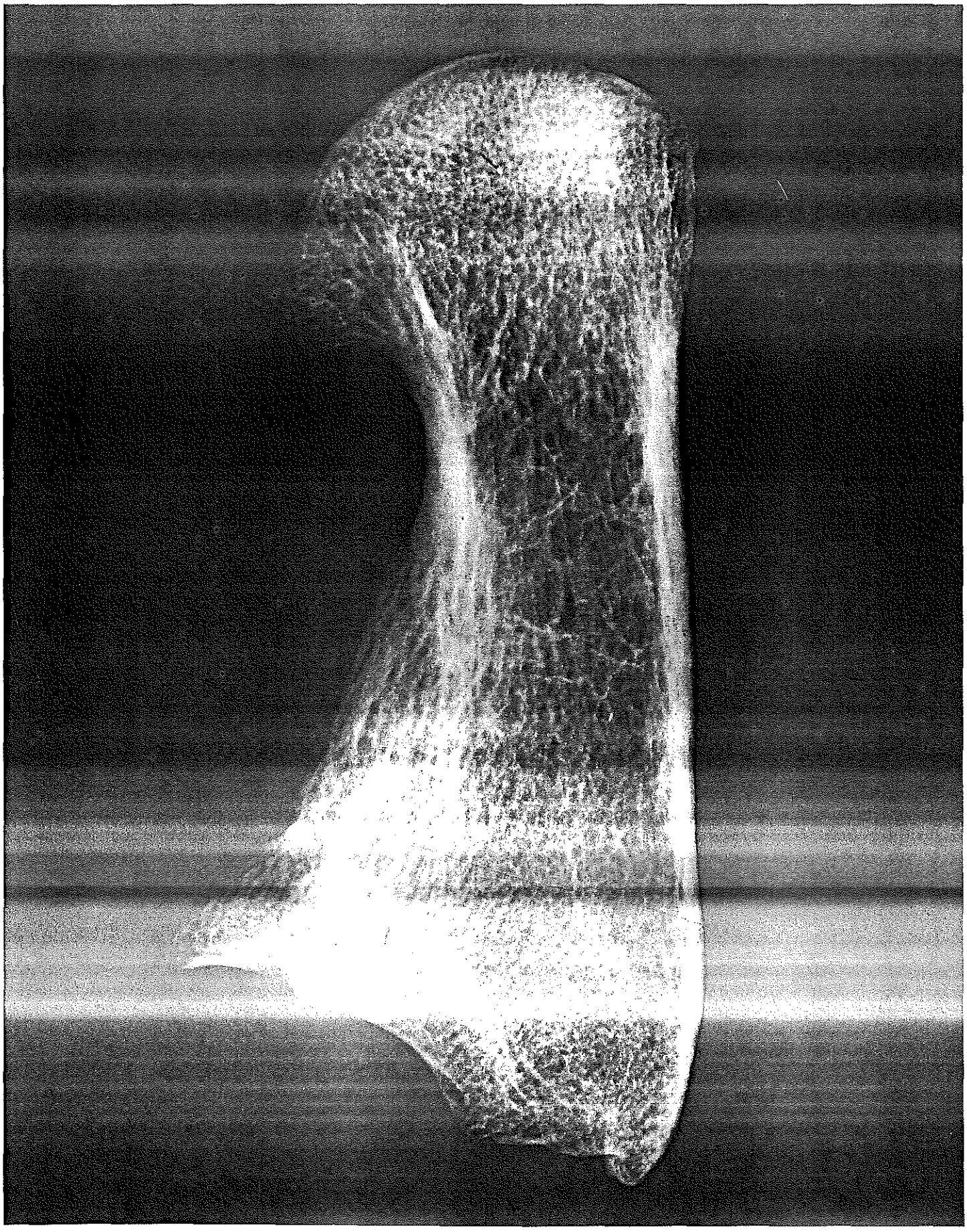


Plate 6: Healed fracture of the left clavicle of TJM:P

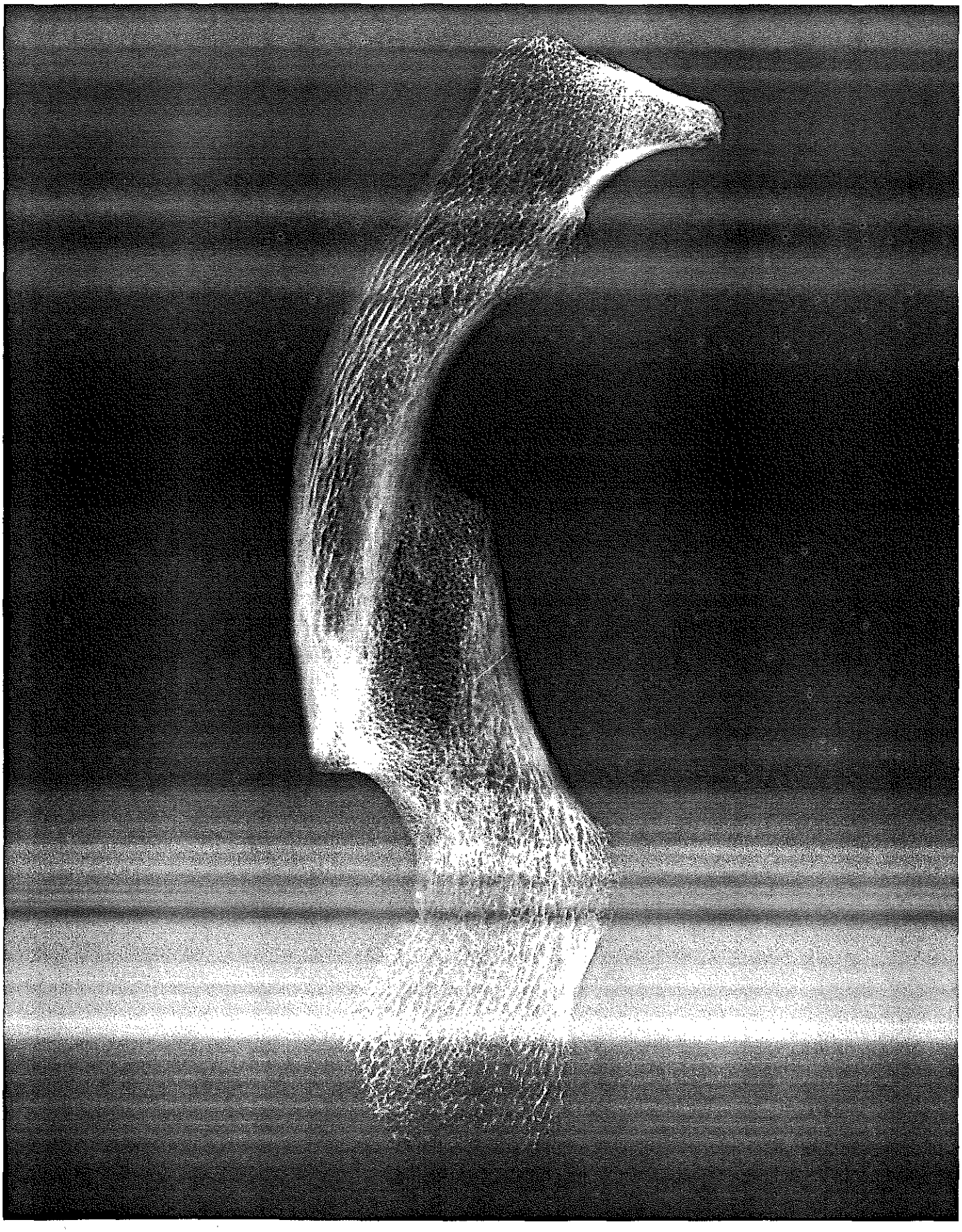


Plate 7: The right humerus of TJM:J

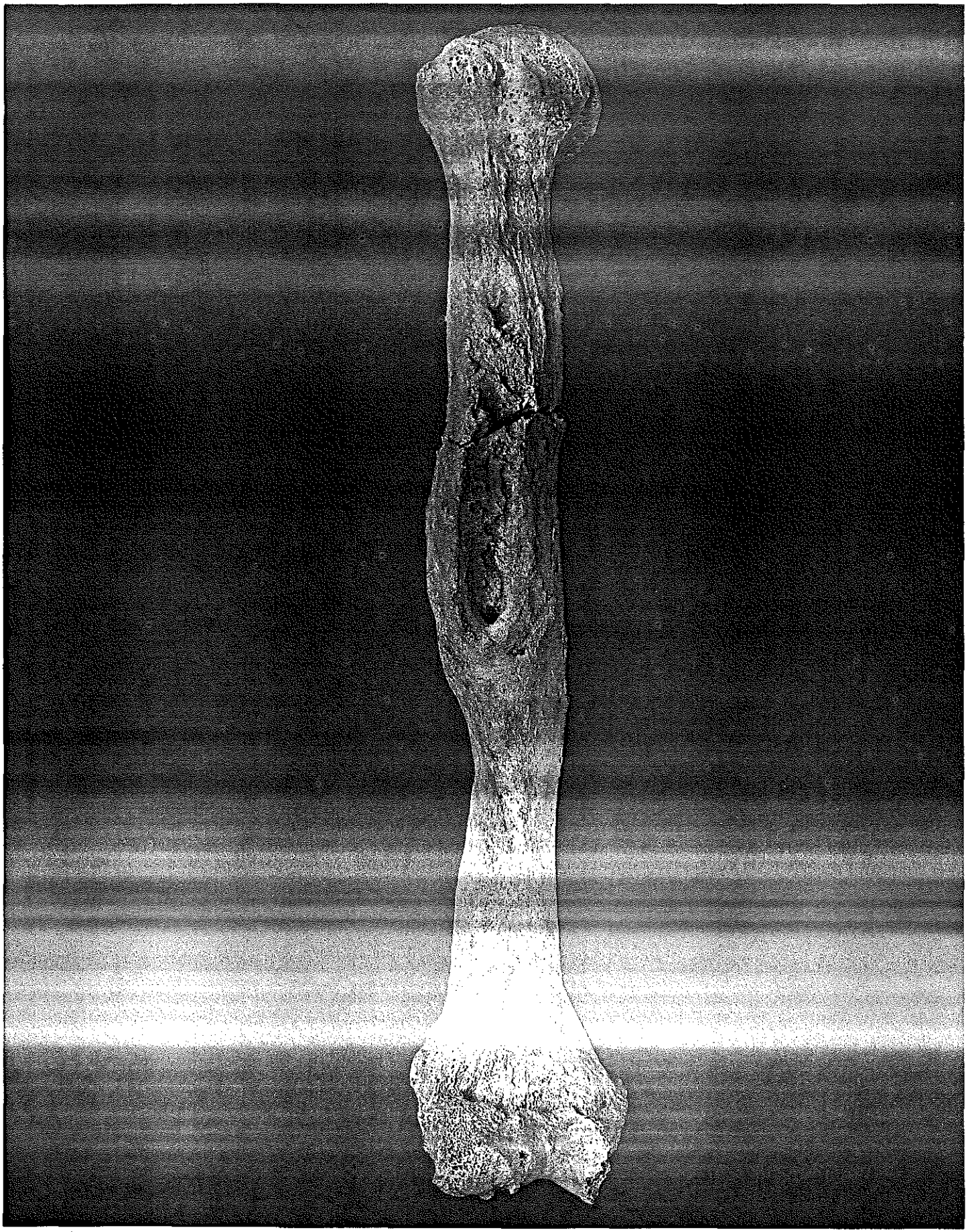
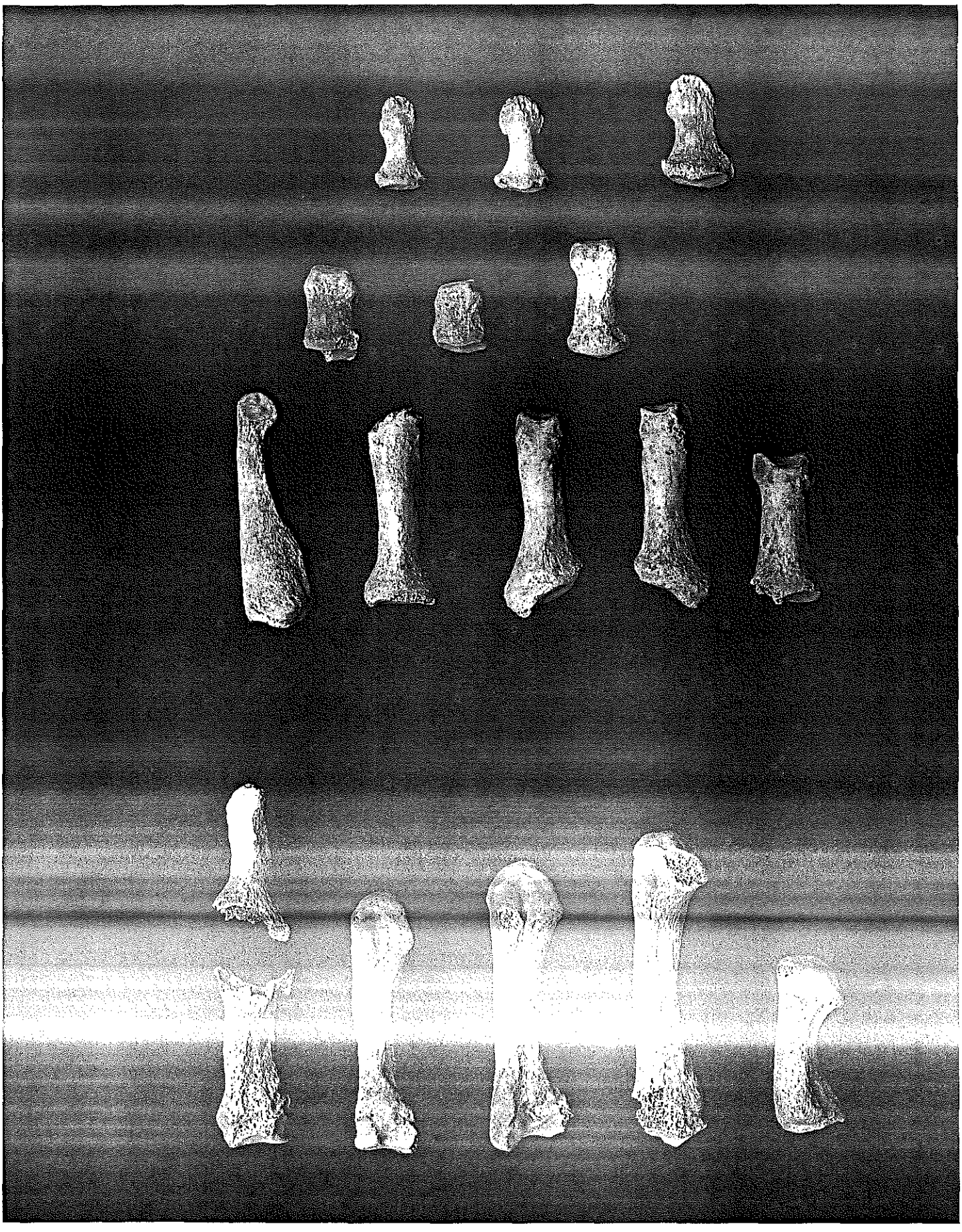


Plate 8: The metacarpals and phalanges of TJM:D



# APPENDIX: DATA FOR INDIVIDUAL BURIALS