Differential Diagnosis of Holes in the Calvarium: Application of Modern Clinical Data to Palaeopathology

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In the text, a comprehensive differential diagnosis is given for the various (i) congenital and developmental, and (ii) acquired (whether pathological e.g. due to inflammation, tumour or trauma, or surgical intervention e.g. trephining) lesions which produce holes in the skull. Many of these conditions are illustrated, and their characteristic features briefly described.

Keywords: HOLES IN SKULL, CONGENITAL DEFECTS, PATHOLOGICAL LESIONS, BENIGN TUMOURS, MALIGNANT TUMOURS, TREPHINING, TRAUMA (MILITARY AND CIVIL), PSEUDOPATHOLOGY, PALAEOPATHOLOGY, DIAGNOSIS.

Introduction

Archaeologists, anatomists, radiologists, historians, pathologists, and occasionally the police may all encounter skulls of recently deceased, or occasionally of long-dead individuals during the course of their activities which contain “holes” in unexpected places. If the skull is well preserved, but particularly if found with the rest of the skeleton, then it may well be possible, with a reasonable degree of certainty, to establish the aetiology of the lesion. Should the skull be in poor condition, however, as it may well be, particularly if it is exposed to the elements, or to certain soil conditions (Henderson, 1987), it may be impossible to distinguish between the numerous possibilities outlined in this paper. Accordingly, it may well be wise to err on the side of caution in coming to a diagnosis, particularly if there are no suspicious circumstances to suggest otherwise.

Historically, these lesions are commonly attributed to either syphilis, military trauma or primitive trepanning (synonym; trephining). However, a review of the literature reveals a wide ranging aetiology for holes in the skull, though in the majority of cases these are likely to have resulted either from disease states or from traumatic lesions. In the examination of living individuals, neurosurgeons, trauma surgeons and radiologists as well as biological anthropologists will encounter them relatively frequently in their clinical practice.

There are five aetiological groups of holes in the calvarium as follows.

1. Congenital and developmental defects. This represents one of the most important groups. Examples of “holes” in the skull of congenital origin can often, though not invariably, be recognized because of their characteristic location. All result from a failure of ossification. These lesions are sometimes present in the form of elongated defects associated with suture lines or, relatively commonly, may be bilateral and located in the parietal region, being either parietal fenestrae or foramina. A selection of illustrations of these conditions is included, because it is particularly important that they be distinguished from “holes” resulting from surgical intervention (i.e. trephining) or trauma.

2. Pathological lesions due to disease states such as infection or malignancy, but excluding trauma. While this group probably accounts for the highest proportion of cases encountered with “holes” in the skull, once the skull has been in the ground for any period of time, it is most unlikely that any indication of the cause of the lesion could be ascertained. For this reason, while we have attempted to provide a reasonably complete classification which indicates the various pathological lesions that could account for the type of lesion under discussion, this section is likely to be of academic interest only. Clearly if a lytic lesion is seen radiologically in a living patient, then an appropriate biopsy could be taken and/or other clinical findings be recognized (e.g. absent clavicle in cleidocranial dysostosis), or the primary source of a secondary metastatic deposit be established, and these would in most instances allow a diagnosis to be made.

3. Surgical intervention (i.e. trepanning/trephining).
(4) Traumatic—usually military trauma, e.g. musket ball, bullet, shrapnel injuries, sword/sabre injuries, or due to the effect of other blunt or sharp implements.

(5) Post-mortem holes encountered in palaeopathology; this group represents a major problem in diagnosis, and general guidelines will be given on how it may be possible to distinguish between ante-mortem and post-mortem holes.

While the vast majority of the lesions to be discussed here that may be encountered by the palaeopathologist involve the calvarium, or skull cap, lesions may be encountered which involve all regions of the neurocranium, as well as the facial bones (the viscerocranium) and mandible, depending clearly on their aetiological origin. Thus, for example, while an isolated secondary tumour may commonly be associated with the parietal area, it could involve any other region of the skull or even the mandible. Clearly, in some conditions, there may be multiple lesions involving various parts of the skull. These might result from, for example, multiple traumatic lesions, or from metastatic tumour deposits. While we have mostly considered conditions which produce lesions of the skull or even the mandible. Clearly, in some conditions, there may be multiple lesions involving various parts of the skull. These might result from, for example, multiple traumatic lesions, or from metastatic tumour deposits. While we have mostly considered conditions which produce lesions of the calvarium, in some instances (principally for completeness) we have briefly alluded to other conditions which produce similar lesions in other regions of the skull, such as the viscerocranium, the base of the skull or the mandible.

Radiography undoubtedly plays a large part in establishing the diagnosis in living individuals where skeletal lesions are present, its role in palaeopathology (with very few exceptions when only the skull is available for analysis) is, however, much more limited. Indeed, possibly the most important part of establishing the aetiology of a hole in the skull involves the careful examination of the specimen and a detailed knowledge of the context and site of its discovery. More often than not, there is no well-documented history, and accordingly the skeletal remains will be all that is available to those that are involved in the analysis of this material.

Clearly, if a skull of a young male adult is found at a known battle-site, and displays obvious evidence of trauma induced by a blunt instrument or from an edged weapon, then the chance of obtaining an accurate diagnosis to account for the skull lesion(s) and likely cause of death is high. Should a skull with apparently similar lesions be found in a burial plot, in the absence of a relevant historical context, then a variety of other factors such as, for example, post-mortem trauma, would have to be excluded before such a definitive diagnosis could be made.

In the case of holes in the skull induced by disease, it may well be impossible and, in the majority of cases it is probably unwise (even foolhardy) to make a definitive diagnosis in the absence of appropriate histopathological evidence. Because the latter largely depends on the finding of characteristic histological features in the tissue that was originally contained within the osteolytic lesion, this might only be possible if the skull had been obtained from a recently dead individual, a situation more commonly encountered by a forensic pathologist than a palaeopathologist.

Accordingly, we have only been able to illustrate those conditions from examples in the collections to which we have had access, either where a definite clinical history was available or in those cases which illustrate traumatic lesions when the historical context is known. While every attempt has been made to provide a complete classification of conditions in which holes in the skull may be induced, we have only been able to provide illustrations of those conditions that a palaeopathologist is likely to be able to recognize with some degree of confidence. The examples that are not illustrated therefore largely represent conditions that a palaeopathologist would not in any case be able to diagnose because of the absence of (1) an adequate clinical history, (2) access to histological sections through the lesion, and (3) the findings from radiological analyses carried out in order to establish the clinical diagnosis.

Access to sophisticated methodology, such as scanning electron microscopy, now provides a more detailed means of analysing the margins of a lesion than was formerly possible, and in certain cases may allow the nature of the instrument used to produce a traumatic lesion to be determined (see, for example, Wenham, 1989). Equally, spectrophotometric analysis of skeletal samples is now being used increasingly to confirm (or in some cases establish) the presence of certain disease states, such as osteomalacia. An analysis of the mineral content of bone may be used to assess nutritional deficiencies, determine possible changes in diet, and assess exposure to toxic elements and its consequence for health (Gilbert, 1977; Waldron, 1987a). Another under-used approach would appear to be the histological examination of skeletal remains, particularly for the analysis of long-term bone decomposition (Garland, 1987).

While there are a number of excellent recent monographs available for those with a special interest in the detailed examination of the skeleton (see, for example, Waldron, 1994; Roberts & Manchester, 1995) in addition to the standard works on this topic by Brothwell (1981) and Ortner & Putschar (1985), we believe that illustrations and descriptions of those conditions that are relatively commonly encountered by palaeopathologists serve a particularly useful purpose. In this report, we have additionally attempted to provide a comprehensive catalogue of those conditions that may produce holes in the skull, emphasizing (a) those which may be recognized from their morphological features alone, and (b) those which could not be reasonably diagnosed in the absence of additional information unlikely to be available to the palaeopathologist (see above).
Where the skull only, and not the complete skeleton, is all that is available for analysis, this clearly often substantially increases the problems involved in attempting to establish a proper diagnosis (Waldron, 1987b). However, despite this limitation, it can still be extremely instructive to attempt to produce a differential diagnosis based on the morphological appearance and features as well as the location of the lesion(s) in question. Where appropriate, the diagnostic features are emphasized in the figure legends. In many instances, while this exercise may not allow the establishment of a definitive diagnosis to be made, it may at least allow the investigator to exclude the more unlikely causes of holes in the skull in relation to the specimen under investigation.

While the underlying cause may be obvious in some cases, for the reasons indicated above, in the majority it is not. In cases of trauma, for example, the initial appearance may be modified by surgical repair, healing or due to the effect of chronic infection. In this paper, we attempt to simplify the above issues, by providing a simple working classification for likely causes of lesions of this type and, where possible, illustrate them with examples from skulls in the museum collections of the Department of Anatomy, University of Edinburgh, and from the osteological collection in the Museum of the Royal College of Surgeons of Edinburgh. Our material complements that illustrated in the standard works in this field (see above), and the review of the radiological literature provided by Pendergrass and Lorimier (1936) and more recently by Keats (1988) and Kaplan et al. (1991). The volume by Keats, entitled Atlas of Normal Roentgen Variants that may Simulate Disease is possibly the most comprehensive and invaluable account available on this topic.

### Congenital and Developmental Defects

**Failure of ossification**

**Complete dysostosis.** These are usually present in the form of elongated defects, and commonly result from deficient union of cranial bones. They tend to occur in the midline, since they commonly involve the sagittal suture. They may also be associated with the parietal suture lines. Dysostosis may occur as a result of severe hydrocephalus (which may be encountered as a congenital lesion often associated with spina bifida cystica, or may occur in childhood, secondary to an intra-cranial infection) or be associated with defects of the clavicle in cleidocranial dysostosis.

Cleidocranial dysostosis tends to have an autosomal dominant inheritance (Jarvis & Keats, 1974; Dore et al., 1987), but there are rare cases where it is recessive. In infancy, there is poor or even absent ossification of the parietal bones. There is marked widening of both the sutures and the fontanelles. The anterior fontanelle splays forward between the frontal bones to become a “metopic fontanelle” (Jarvis & Keats, 1974). As the child grows, further ossification of the calvarium occurs but the sutures remain widely patent. The anterior fontanelle will remain patent into adulthood. Patients have a normal intelligence quotient and prognosis. This contrasts with the situation in individuals with severe congenital hydrocephalus, which may be a consequence of intrauterine infection, or the presence of an abnormality of the brain stem, such as the Arnold–Chiari malformation, or aqueduct stenosis, where the normal channel between the third and fourth ventricles of the brain is occluded. In rare cases, cleidocranial dysostosis may be associated with the Arnold–Chiari malformation or syringomyelia.

**Partial dysostosis.** In this condition, the lesion consists of a more localized gap between the cranial bones, and is usually caused by the presence of an intervening meningocele. This lesion therefore tends to be rounded in shape rather than elongated, is commonly located in the midline, and is found particularly in the lower occipital, sagittal or frontal region (Figures 1 & 2).

**Bi-parietal thinning.** This condition is relatively commonly encountered, and is usually said to be congenital in origin (Durward, 1929), but it is also sometimes seen as a manifestation of post-menopausal osteoporosis. The outer table and diploe are lost, with characteristic preservation of the inner table (Steinbach & Obata, 1957). While usually bilateral and symmetrical (Figure 3), this condition may rarely be unilateral (Wilson, 1947). It is also sometimes seen in association with symmetrical depressions of the parietal bone (Figure 3), this relationship being first described by Sir George Humphry (1858: 242–243) in the Orang and in Man. Similarly, extreme thinning of localized regions of the calvarium may occur at other sites. For example, it may be bilateral and symmetrical in the frontal region, or appear as isolated or symmetrical areas of lucency in the occipital region. In these sites, it may be mistaken radiologically for the radiolucency seen in osteoporosis circumscripta of Paget’s disease (Keats, 1988) (see later). Clearly, extensive areas of calvarial thinning are particularly vulnerable to post-mortem damage, and should be recognized as such.

**Anomalous suture line deformities (e.g. drug induced).** In the past, the drug Aminopterin was given to induce abortion in pregnant females. This practice was discontinued when it was found that a fetus which actually survived to term showed severe developmental malformations including sutural line abnormalities. Shaw and Steinbach (1968) described a case where the fetus survived to term and was born with a cranium bifidum (a congenital condition one feature of which is persistence and abnormal widening of the metopic (frontal) suture). Radiographs taken when the individual was
The material illustrated here comes from the Anatomy Museum, University of Edinburgh (Anat.) (for additional information, see Anon, 1891), the Museum of the Royal College of Surgeons of Edinburgh (RCSEd.) and the Ballingall Collection of Military Surgery, Department of Anatomy, University of Edinburgh (Ball.) (for additional information, see Ballingall, 1855). The reference numbers of all specimens are provided.
4 years old showed that a separate centre of ossification had developed within the cranium bifidum dividing the abnormal suture space into two small defects parasagittally. Shaw and Steinbach noted that the “extent and sequence of ossification was somewhat similar to that seen in cleidocranial dysostosis” (1968: 480).

While Aminopterin-induced skull malformations represent an extreme curiosity, and would not now be encountered, other drugs are certainly capable of inducing cranial abnormalities if exposure occurs during pregnancy, and some of these may lead to the development of “congenital holes” of the skull.

Clearly in the majority of cases these lesions are seen in the skull of newborn infants following either stillbirth or neonatal death. The most extreme example of one of these conditions would clearly be anencephalus, or a limited variation of this condition, e.g. meroacrania, which can involve herniation of, usually, the forebrain through any of the sutures but usually the sagittal suture. In cases of meningocoele, it is the dura/arachnoid filled with cerebrospinal fluid that herniates through the sutural deficiency, in contrast to meningoencephalocele/encephalocele where the herniated sac additionally contains disorganized neural/brain tissue.

Since most of these conditions, with the possible exception of sutural deficiencies associated with small meningocoeles, and occipital meningoencephaloceles, are incompatible with prolonged survival, these lesions would not be seen in adult skulls and are therefore only briefly mentioned in this analysis.

An example of survival to adulthood of an individual with this condition is given by Maclagan (1865) and Turner (1866). He describes the skull of a 28 year old female, with a midline occipital lesion which was covered in life by a cystic “tumour” containing clear fluid, about the size of a hen’s egg (Figure 2). It is unclear whether this was a meningocoele or a cutaneous cyst which, over a prolonged period, caused erosion of the skull due to pressure necrosis.

**Abnormal enlargement of parietal foramina**

Parietal foramina are normal structures. They occur in bilateral, symmetrical situations on either side of the sagittal suture, just anterior to the lambdoidal suture. They are present in 60% of adults (Boyd, 1930) and are usually approximately 0.5–1.5 mm in diameter, though they may have a considerably larger diameter (Greig, 1927). They are thought to arise from the persistence of the most lateral part of the primitive parietal notch. The latter is a notch or fissure found in most parietal bones at birth. It is a “very constant cleft in the upper border of the bone in the position of the parietal foramen, formed by a vessel which produces a rounded or oval notch at the outer angle of the cleft” (Paterson & Lovegrove, 1900: 236). These authors noted its presence in 70/85 (82%) specimens examined. They transmit normal emissary veins (of Santorini) between the superior sagittal sinus and the epicranial veins (Currarino, 1976). Boyd (1930), however, states that abnormally large parietal foramina are not usually associated with an enlarged emissary vein.

The predisposition towards the presence of enlarged parietal foramina may be an hereditary transmission (Pepper & Pendergrass, 1936). Sequential radiographic analysis carried out over a period of years has revealed that they often show considerable evidence of progressive ossification (Pendergrass & Pepper, 1939).

**Parietal fenestrae**

These are the most commonly encountered of the congenital skull defects and need to be distinguished from enlarged parietal foramina. Parietal fenestrae (or Catlin marks) are not caused by enlargement of a parietal foramen. They are a separate entity as shown by their occasional occurrence alongside parietal foramina. They tend to be bilateral more often than unilateral, and are equal in size in 60% of bilateral cases. They are oval in shape with a greater mediolateral than anteroposterior diameter, have

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**Figure 1.** Partial dysostosis in region of sagittal suture. Dated between the 4th and 8th centuries AD (Henshall, 1956; Dalland, 1993) this skull shows partial dysostosis. The oval midline lesion measures 24 x 12 mm, and is located within the sagittal suture immediately posterior to the coronal suture. It has a typical symmetry with regular bevelled edges and is most likely to have been caused by an intervening meningocele (overview, (a); close-up, (b)) (Anat. ref: IB 263).

**Figure 2.** Parietal fenestrae associated (by chance) with parietal dysostosis in mid-occipital region. There is a hole in each parietal bone (arrow heads) and in the upper part of the occipital bone (arrow). The two typical parietal fenestrae measure 14 x 7 mm on the left and 17 x 12 mm on the right, equidistant (32 mm) from the midline. They have smooth rounded edges as if closing. An unobliterated suture runs between the aperture in the left parietal and that in the occipital, crossing the lambda at a right angle. The midline occipital lesion measures 26 x 13 mm (Anat. Ref: Os Ga 3.Y 117).

**Figure 3.** Biparietal thinning. External view of the outer surface of the calvarium of an intact skull illuminated through the foramen magnum. Two symmetrical areas of lucency which measure approximately 60 x 60 mm in diameter are clearly displayed in the mid-parietal region where the skull is particularly thin. The lucent areas on this skull are associated with congenital depressions of the calvarium each of which has a slightly elevated peripheral margin. The aetiology of these depressions is unclear, but it has been suggested that they may be due to “faulty fetal packing”, due to long-standing pressure on these sites by the fetal feet or maternal sacrum (Keats, 1988). Other cases where there has been a clear hereditary component have also been described (Shepherd, 1895). A line of lucency just in front of the area of parietal thinning on the left side is due to thinning of the calvarium in the region overlying the sphenoparietal venous sinus, and if seen on radiography, may simulate a fracture (Anat. ref: XXXV.B.2).
smooth edges, and their margins are bevelled becoming smaller towards the inner table. Parietal fenestrae are thought to occur as a result of a defect in parietal bone ossification. This takes place in membrane during the second month of gestation. The cause of the defect is unknown, but it is possible that these lesions may be genetically determined. Parietal fenestrae also tend to get smaller as the individual ages, though it is a matter of some controversy as to whether they ever close completely (Figures 2, 4 & 5). It is important that these lesions are not confused with healing trephine holes or fractures.

Parietal fenestrae are often associated with other conditions such as craniosynostosis and other craniofacial abnormalities. There is a rare autosomal dominant condition in which parietal fenestrae are found with lateral clavicular hypoplasia, macrocephaly and an occipital hair tuft. This is a separate entity from cleidocranial dysostosis (Currrarino, 1976).

Venous connections between intra- and extracranial vessels

Turner described these in the above-mentioned skull (Figure 2) in which there were two paramedian foramina situated in the posterior part of the parietal bones. These foramina were in life covered by a “cribriform” fascia, but the presence of veins passing through the fascia was only a supposition on Turner’s part, based on the cribriformity and surrounding grooves on the inner table of the skull. Turner believed that in this instance the appearance observed was due to a primary increase in the number of veins in this region leading to an enlargement of the normally situated parietal foramina. However, it is now known that the normal parietal foramina convey an emissary vein (of Santorini) whereas parietal fenestrae do not transmit veins. Abnormal communications between intracranial and extracranial veins do occasionally exist, and each is known as a ”sinus pericranii”. They are most commonly congenital in origin although they may be spontaneously or traumatically acquired (Ohta et al., 1975; Witrak et al., 1986).

Craniotabes

In this condition, multiple focal sites of thinning or even, in more extreme cases, complete dysostosis and weakness of the skull bones are present in the infant which are of sufficient degree to allow indentation on pressure with the finger. It occurs in association particularly with rickets, cleidocranial dysostosis and may also be a feature of the newborn skull in congenital syphilis (syphilitic craniotabes, Figure 6).

Alternative (aetiologial) classification

Since all congenital holes are due to failure of ossification it might be better to divide them according to the causes that lead to this failure i.e. on an aetiological basis as follows:

1. Hydrocephalus;
2. Meningocoele or meningoencephalocoele;
3. Emissary veins (causing parietal foraminae, NOT fenestrae);
4. Unknown (e.g. the unexplained causes of parietal fenestrae and cleidocranial dysostosis);
5. Syphilitic craniotabes.

Pathological Lesions

Tumours: primary benign

These lesions only uncommonly produce holes in the skull, and no examples are to be found in either the University or Royal College of Surgeons collections.

Haemangioma. This constitutes about 10% of benign skull tumours and has a male to female ratio of one to three. There are two types—cavernous and capillary. Cavernous haemangiomatas are more common and tend to occur in the frontal or parietal bones. Although both types arise in the diploe, there is a variable degree of erosion of the outer table, and the inner table is typically well preserved. The lesion tends to be circular and have an irregular but well defined margin. Radiologically it gives a honeycombed appearance. The presence of an intact inner table and a lack of hyperostosis helps to differentiate this tumour from a meningioma.

Dermoid and epidermoid. These lesions, which are identical to each other clinically and radiologically, can occur anywhere in the calvarium but are more likely to be supraorbital, in the anterior temporal region or near the vertex. They arise in the diploe and then expand in all directions to involve both tables of the skull (Taveras & Wood, 1964). As they expand, they compress the surrounding bone to produce sclerotic margins. These margins are sharply defined and may be scalloped or lobulated. It is also important to note that since these tumours often become infected there may be an area of osteomyelitis surrounding the lytic lesion.

Chondroma. This tumour is rarely found in the skull and when it is it tends to be related to the synchondroses of the skull base. However, it is not unknown in the calvarium, and may produce lytic lesions with fairly sharply defined margins and adjacent stippled calcification (Minagi & Newton, 1969).

Giant cell tumour. Also extremely rare and related to the endochondral skull base bones. It will cause a sharply demarcated area of bone destruction (McNerney, 1949).
Tumours: primary malignant

A variety of primary malignant tumours affect the skull, though they are usually impossible to distinguish from isolated metastases from primary tumours located in other parts of the body.


Osteogenic sarcoma. This is the most common of the primary malignant tumours of bone (Figure 7) but is
rare in the skull (Vandenberg & Coley, 1950). It occurs in younger individuals (ages 15–25) but may also be seen in older individuals secondary to Paget’s disease. In the skull, it more commonly occurs as a result of Paget’s disease. The frequency of osteogenic sarcoma arising within the osteitis deformans of Paget’s disease is quoted as 10–15%. A large lytic area with poorly defined margins is produced (Geschickter, 1936; Dahlin & Coventry, 1967). The entire thickness of the calvarium is usually destroyed (Garland, 1945). There is spiculation and calvarial thickening around the lesion. A drawing showing multiple lesions of various sizes, up to a maximum of about 1.5–2 inches in diameter is illustrated by Ballingall (1829: 252). This individual was diagnosed as having multiple sarcomatous lesions, and the appearance of the skull probably represents an extreme example of what may be seen in this condition. In the absence of a biopsy of the tumour, however, it would not be possible to establish whether the condition seen in this skull resulted from multiple metastases from a primary tumour located elsewhere in the body.

**Fibrosarcoma.** This may arise from the periosteum, scalp or dura. It causes complete destruction of all tables over a large area. As the process is extremely rapid the margins are irregular (Conley et al., 1967).

**Chordoma.** Arises from notochordal remnant of the clivus leading to destruction of the basiocciput (Haas, 1934). The clivus is the sloping area behind the dorsum sellae, and is uninterrupted in continuity with the clivus of the occipital bone (i.e. the basiocciput) in the adult skull; it supports the upper part of the pons (Warwick & Williams, 1973). In the embryonic period, the most rostral part of the notochord is surrounded by an area of condensed ectomeninx which becomes chondrified—this unpaired plate-like mass is located between the notochord and the brain stem and is termed the parachordal cartilage or basal plate (Hamilton & Mossman, 1972).

**Tumours: secondary benign**

**Meningioma.** Meningiomas usually cause hyperostosis (hypertrophy of bone) of some sort but occasionally a meningioma arising close to bone will cause an erosion without provoking an osteoblastic response (Cushing & Eisenhardt, 1938). The lesion produced will have poorly defined margins and affect the inner more than the outer table. This will thus look similar to a bony metastasis, myeloma or osteomyelitis.

**Glomus jugulare tumour.** Affects the skull base exclusively. The glomus jugulare is a small mass of benign tumour tissue lying in the dome of the bulb of the internal jugular vein (Guild, 1941). If untreated, this tumour may invade the skull base. There is enlargement of the jugular foramen and progressive erosion of the petrous and occipital bones. The entire petrous part of the temporal bone may be destroyed.

**Tumours: secondary malignant**

**Metastatic carcinoma.** Osteolytic lesions occur more commonly than osteoblastic. They may arise from carcinoma of the prostate, breast, bladder, kidney, lung, uterus, gastro-intestinal tract (Figure 8), thyroid, and malignant melanoma. The majority come from breast and lung carcinomas. Metastatic lesions are characteristically multiple with varying size and ill-defined margins. Lesions less than 5 mm in diameter tend to be confined to the diploe (Taveras & Wood, 1964). Radiographically they may simulate multiple
myeloma or, if single, osteomyelitis. Often the edges are bevelled with one table affected more than the other. Metastases may affect any region of the skull but are detected most easily in the calvarium.

Neuroblastoma. The skull is a frequent site of metastasis from this common childhood tumour. Diffuse lesions are produced as in metastatic carcinoma. The tumour is highly vascular and causes periosteal elevation. This leads to radial bone spiculation and eventual calvarial thickening (Carter et al., 1968).

Ewing’s tumour (endothelial myeloma). While rarely arising as a primary in the skull this tumour, a form of bone sarcoma seen in childhood, frequently metastasizes there. The lesion produces bone destruction with ill-defined margins similar in appearance to osteomyelitis (Willis, 1940).

Haemopoietic lesions

Myeloma. Myeloma most often affects males aged 40–60. Any bone may be involved but the skull is a common site. There are multiple sharply defined (“punched out”) lesions which penetrate all tables of the skull. There are usually a large number of lesions spread over much of the calvarium. However, a solitary myeloma may rarely reach a diameter of 2–3 cm before other lesions are seen (DuBoulay, 1965).Appearances in myeloma are similar to lymphoma and metastatic carcinoma.

Lymphoma. This disease tends to give rise to multiple lesions of varying size with ill-defined margins.

Leukaemia. Leukaemia will only produce calvarial lesions in childhood disease and even then this is rare. The lesions are multiple but ill-defined, and not as dramatic as the preceding two conditions.

Locally invasive carcinomas

Nowadays the commonest neoplasms to involve the skull directly are those of the nasopharynx and the paranasal sinuses (Fletcher & Million, 1965). Extensive destruction of the facial bones may result from the growth of a squamous cell carcinoma originating in the frontal air sinuses. This tumour may invade into the surrounding air sinuses, and cause erosion of the maxillary, nasal, ethmoidal and sphenoidal bones (Figure 9). However, before effective treatment became available, any head and neck tumour could eventually spread locally to involve the skull.

Lesions secondary to trauma

Intradiploic cyst. This arises in the newborn or young child as a result of traumatic haemorrhage into the diploe. A round or oval defect in the diploe results and this has a fine sclerotic margin (Epstein & Epstein, 1967).

Leptomeningeal cyst. Following a linear skull fracture there may be a dural tear and a small arachnoid herniation through this into the site of the fracture. The resulting cyst, which contains cerebrospinal fluid, gradually erodes adjacent bone and produces a lytic defect with scalloped margins (Taveras & Wood, 1964). Although far more common in children these cysts can occur following fractures in adults.

Fibrosing osteitis. This condition also follows a fracture. It is formed by the resorption of bone and its replacement with a fibrous scar. It has a similar radiological appearance to the lesion produced by a leptomeningeal cyst (see above).

Pressure necrosis following a sub-periosteal haematoma. A traumatically induced sub-periosteal (sub-pericranial) haematoma may strip the pericranium from the outer table of bone and then cause aseptic resorption of the outer, and subsequently the inner, table (Figures 10 and 11).

Vascular lesions

Sinus pericranii. Described in congenital section.

Pacchionian (or arachnoid) granulations. These arachnoid invaginations produce thinning of the overlying calvarium, and are often, erroneously, diagnosed as being pathological; they produce lucent depressions on
a radiograph, and may therefore simulate a destructive lesion. Usually only the inner table and diploe are affected but sometimes the outer table is also eroded. They are most often found parasagittally but occur commonly in the squamous occipital bone and occasionally in the more lateral parts of the parietal and frontal bones. The degree of erosion produced is very variable, and the example illustrated (Figure 12(a)), shows several isolated deep depressions located on either side of the superior sagittal dural venous sinus produced by arachnoid granulations. The use of transmitted light for viewing such specimens emphasizes the fact that, in most instances, all but the outer table of the skull has been eroded (Figure 12(b)).

Secondary to intracranial disease

(1) Intracerebral glioma (tumour of neuroglial tissue).
(2) Chronic raised intracranial pressure.

Both of the above conditions tend to produce gradual erosion of the calvarium from inside to out, resulting in lesions of variable size, often with poor margination. Clinically, on radiological examination of the head of an adult with chronic raised intracranial pressure (such as in adult hydrocephalus, or secondary to an intracerebral glioma), the irregular mosaic pattern of normal and thinned areas of bone of the skull is said to resemble “beaten silver”, and a similar appearance would be seen on transillumination of the isolated skull. Prominent “digital” markings, particularly in children between the 4th and 10th years do not, in themselves, necessarily reflect increased intracranial pressure (Macaulay, 1951).

Metabolic

Hyperparathyroidism. In this disease the bone changes are ubiquitous. The skull shows wide areas of granular osteoporosis giving the calvarium a woolly appearance (Camp, 1932). Larger areas of osteoporosis may coalesce to form “brown tumours” (also called the osteitis fibrosa cystica of Von Recklinghausen). These

Figure 9. Sinus carcinoma. This skull shows extensive destruction of the facial bones as a result of a squamous cell carcinoma of the frontal air sinuses. This tumour has extended laterally and has eroded into both orbits, and more generally to cause erosion of the upper part of the maxillary, the nasal, ethmoidal and sphenoidal bones. This 63 year old male had an operation to relieve the local pressure effects of this lesion. The latter has distinct margins and there is no obvious attempt at repair. The margins of the left frontal sinus have crescentic marks caused by bone-cutters at operation (Greig, 1912). This individual had acquired syphilis 19 years previously (RCSEd. ref: G.C. 5374).

Figure 10. Absorption of outer table of calvarium. In this adult skull an oval portion of the outer table at the vertex has been destroyed (dimensions 85 mm antero-posterior and 60 mm laterally). The margins are distinct and regular and the surrounding bone is normal. The floor of the lesion is smooth and more dense than normal diploe. Complete perforation has occurred in the regions of the sagittal and coronal sutures within the margins of the lesion. There are no signs of an inflammatory reaction (RCSEd. ref: G.C. 10771).

Figure 11. Circular bregmatic depression. This is the skull of a young adult which shows a depressed area with an irregular but smooth margin measuring 40 mm antero-posterior and 45 mm laterally. The floor is irregular and there is a central perforation through the calvarium in the bregma area measuring 4 mm antero-posterior and 2 mm laterally. The margins of this central perforation are slightly crenated. This skull is thought to show the features resulting from a traumatic, aseptic haematoma which had occurred earlier in life. The indented oval depressed area (arrow heads) probably represents the peripheral margin of the original sub-periosteal haematoma (close-up of lesion as viewed from outside the skull, (a); close-up of lesion as seen from inside the skull, (b)) (RCSEd. ref: G.C. 7036).

Figure 12. Pacchionian (or arachnoid) granulations. Two views of the same skull. This possesses a number of circular/oval depressions on the inner aspect of the calvarium. In this specimen, they measure between 11 and 13 mm in diameter, and display the characteristic features of pacchionian granulations (a). View of the outer surface of the calvarium which has been illuminated from below to emphasize the extreme degree of thinness of the bone overlying these lesions (arrows) (b) (Anat. ref: H.T. 521).
are sharply defined lytic areas. In the healing stages there may be areas of sclerosis in the surrounding osteoporosis.

**Vitamin D deficiency.** Thinning and flattening of the occipital bone produces "craniotabes". Theoretically, this condition may lead to erosions of the skull, although we have yet to find documented evidence of holes being produced by this mechanism. A similar appearance of the infantile skull is also seen in association with congenital syphilis (Figure 6), and in cleidocranial dysostosis.

**Osteomyelitis**

*Pyogenic.* In extreme cases, long standing untreated pyogenic osteomyelitis may lead to very extensive erosion of bony tissues. An example is illustrated in which infection involving the mastoid region (Figure 13) has eroded and destroyed almost the entire occipital/temporal region on one side of the skull. In an example involving the parietal region (Figure 14), extensive destruction of the surrounding tissue is seen.

Osteomyelitis as a result of local spread of infection affects the calvarium far more often than it affects the skull base. The bone has a "moth-eaten" appearance with lytic areas coalescing to form holes with irregular margins. With time, sclerosis may occur around the margins of such lesions, but periosteal new bone formation is almost unknown.

Granulomatous—tuberculous, syphilitic, yaws, leprosy, mycotic, parasitic. Before discussing this topic in any detail, it should be emphasized that it is said to be notoriously difficult to distinguish between the skeletal lesions produced by the advanced stages of syphilis and yaws, and that it is equally difficult to make a definitive diagnosis of leprosy. With these reservations in mind, it is hoped that the following observations may be helpful to those wishing to distinguish between these various conditions.

The availability of an intact post-cranial skeleton is particularly helpful in the case of tuberculosis where tubercular osteitis usually begins to develop in the ends of the long-bones or commonly affects the vertebral column (particularly in the thoracic and lumbar regions). This produces destruction of the bone which

![Figure 13. Septic osteitis (osteomyelitis) due to mastoiditis. Skull of a female aged 75 who developed mastoiditis. The squamous region of the left temporal bone and its petro-mastoid part have been largely destroyed. The left coronoid process and part of the ramus of the mandible which supports it remain, but the condyle, neck and posterior half of the left mandibular ramus have been destroyed. The temporal process of the left zygomatic bone is also destroyed (RCSED. ref: G.C. 2647).](image1)

![Figure 14. Septic osteitis (osteomyelitis). This lesion in the right parietal bone is an example of septic osteitis following a scalp laceration. The scalp infection has extended deeply to involve the subjacent pericranium. There is a large posterior opening (55 mm antero-posterior and 30 mm laterally) and a smaller anterior opening which are separated by a narrow bridge of bone. The margins are typically irregular except at the superior part where a trephine hole has made a clean and crescentic cut. The inner and outer tables are equally affected but the diploe has been excavated between them. The bone along the medial margin of the larger opening shows the osteoporotic changes which often accompany septic osteitis (outside of skull, (a); inside of skull, (b)) (RCSED. ref: G.C. 595).](image2)
often extends to involve the joints, causing bony union, for example, between two or more vertebrae. In fact, vertebral deformity is the most reliable diagnostic feature. Alternative, less likely, possibilities are pyogenic osteitis, typhoid or brucellar spondylitis, vertebral syphilis and mycotic infection (Mantle, 1959) and blastomycosis (Buikstra & Cook, 1978). Vertebral collapse may also follow a compression fracture (Stewart, 1956).

In yaws and syphilis, bony lesions are seen in the advanced stages of both of these conditions. As in many of the other conditions discussed here, the exact diagnosis may be extremely difficult to make on the strength of a skull lesion alone, though the availability of post-cranial elements where characteristic lesions may be present is usually extremely instructive. According to Brothwell (1981), “in the case of the skull, and in marked contrast to bone destruction resulting from leprosy or secondary tumours (metastases), there is usually a widespread vault (and to a lesser extent facial) osteitis, beginning with restricted zones of periostitis (the so-called ‘clustered pits’ of early inflammation) . . .”.

As indicated previously, yaws and syphilis are extremely difficult to distinguish, and this is a particularly contentious area in palaeoarchaeological circles. In yaws, localized gummata may be present, or a more diffuse osteitis. Irregular depressions or circular areas of rarefaction may be seen.

On the head and facial bones, characteristic depressed scars may be found, and in more advanced stages the hard palate may be completely destroyed, and the nasal region generally attacked.

In relation to leprosy, certain characteristic lesions of the skull are described (see Brothwell, 1981, based on Moller-Christensen, 1961, 1978) as follows.

(a) Specific atrophy of the alveolar bone in the region of the upper incisors (with or without resulting loss of teeth)—termed pre-maxillary erosion.
(b) The hard palate may show minor evidence of osteitis or areas may be completely resorbed.
(c) The anterior nasal spine may be absent or considerably reduced, and this is often associated with atrophy of the margins of the anterior nasal (piriform) aperture.
(d) Tooth malformations, particularly of root growth may be seen following leprosy in childhood.

In these two collections, which mainly consist of 18th and 19th century specimens, a high proportion of “holes” in the adult skull are attributed in the various
museum catalogues to syphilis, and this is seen to present in a number of different ways (Figures 15–19), some of which are believed to be quite characteristic of this condition. These figures are described in detail in the Figure legends. For further details, see Hackett (1976; see also Manchester, 1987).

**Dysplasias**

**Neurofibromatosis.** In this condition, there may be either a complete absence of bones (especially the posterior wall of the orbit) or lytic lesions may result from the erosive effect of the neurofibromatosis. Also, as a result of bone dysplasia, in generalized neurofibromatosis there may be round or oval lesions in the region of the lambdoid sutures (Joffe, 1965).

**Fibrous dysplasia.** This condition usually only affects the skull but can be a generalized osseous disease. The three variants of the disease are cystic, sclerotic, and mixed. It is the cystic variety which arises in the diploe and produces thinning of the outer table and a lesser degree of thinning of the inner table. The margins are sharply defined and have a sclerotic rim similar to epidermoid tumours (Leeds & Seaman, 1962). However, these lesions are usually larger than epidermoid tumours.

**Miscellaneous**

**Paget’s disease—osteoporosis circumscripta (also termed osteitis deformans).** This is most commonly encountered in middle-aged and elderly individuals, both sexes being almost equally affected, though its cause is unknown. It is a common condition in Great Britain, apparently even more common in Australia, and extremely rare among the Chinese (Fairbank, 1978). The pelvis is the most commonly involved part of the skeleton, followed closely by the upper ends of the femur and tibia, vertebral bodies, the skull and humerus (Dickson et al., 1945). The calvarium is affected along with the rest of the skeleton. There is
Figure 18. Lesions believed to be due to syphilis. Syphilitic osteitis has caused thickening and sclerosis of the diploe of the frontal bone whereas the outer and inner tables have been eroded (a). Also, in the left parietal bone (b) there are two roughly circular holes of 6 and 12 mm diameter (inner table). The serpiginous margin of the outer table is smooth but the inwardly bevelled diploe is roughened and the margin of the inner table is sharp and irregular. Again, this appearance should not be mistaken for trephination (Bull. Ref: ?B10, 56/16).

Figure 19. Gumma of the dura mater. This is the skull cap of a case that was presented to Professor Turner by Professor Laycock of the Royal Infirmary of Edinburgh. The hole is at the junction of the frontal and parietal bones on the left side. The lesser erosion of the outer table (a) as compared to the greater erosion of the inner (b) indicates that the lesion was caused by erosion of bone due to pressure necrosis from within the skull. Remnants of periosteum externally and dura mater internally can be seen. Internally the lesion has scalloped margins posteriorly and small osteophytes anteriorly, indicating attempts at new bone formation. The dimensions are: internally 54 x 38 mm/externally 16 x 8 mm. The lesion was assumed to be syphilitic in nature ("probably a gumma of the dura mater") at the time, this being one of the commonest causes for such a lesion. The outer surface of the rest of the calvarium is uneven and, in places, quite rough. This is most likely the result of healed gummata (Anat. ref: Os Gh 14.Y 116).
diffuse mottled thickening of the calvarium accompanied by sharply demarcated lytic areas (the osteoporosis circumscripta). These usually affect the frontal or occipital bones and the margins are irregular producing the “geographic skull”. The differential diagnosis includes: (1) fibrous dysplasia, mixed type, (2) mixed lytic/blastic metastatic lesions, and (3) healing stages of primary hyperparathyroidism. (1) and (2) are never as diffuse and widespread as in Paget’s disease, and neither (2) nor (3) produce the calvarial thickening seen in Paget’s disease.

In this condition, the earliest changes consist of irregular osteoporosis, at the same time accompanied by new bone formation. Initially, the former predominates, but later new bone formation overtakes the osteoporosis and the bones become hard and brittle. The microscopic appearance of the bone, with its mosaic of new cement lines, is characteristic. Similarly, only the outer table of the skull is affected, so that the volume of the cranial cavity is not diminished. Malignant change may occur in this condition, osteogenic sarcoma being the commonest form (see relevant section above for characteristic features in skull involvement), but chondrosarcoma and malignant giant cell tumours are also encountered (see above); such lesions may be single or multiple. Thus while Paget’s disease per se does not produce holes in the skull, they may result from disease states that are secondary consequences of this condition.

Histioctysis X. This is a disease group comprising eosinophilic granuloma, Hand-Schüller-Christian disease (chronic idiopathic xanthomatosis; a type of cholesterol lipidosis characterized clinically by a triad of defects in the membranous bones, exophthalmas, and diabetes insipidus) and Letterer-Siwe disease (a form of xanthomatosis marked by great hypertrophy of the microphages in the spleen, bones and other organs but without lipid substances in the cells). It affects children and young adults. The lytic lesions produced during the active phase of this condition have irregular margins with no surrounding eburnation. The margins are often bevelled reflecting differential involvement of the skull tables. Bony sclerosis (eburnation) may, however, be seen at the margins during healing but not during the active phase. During the healing phase, the margins of the lesion may be converted into ivory-like masses which are characteristically also seen in osteitis and osteosclerosis. Eosinophilic granuloma produces solitary lesions whereas the other two conditions produce multiple lesions which may expand to become confluent. The frontal bone is the commonest bone in the body to be affected by these diseases.

Mucocoele. These may produce osteolytic lesions around the paranasal sinuses.

Surgical Intervention: Trepanation/Trephining

Cranial trepanation/trephining is cited as the first ever surgical procedure. To date, approximately 1500 trepanned skulls have been found world-wide (Außerheide, 1985). It was performed by prehistoric, historic and even some modern societies as a forerunner of burr holes.

Trepanning is thought to have started during the European Neolithic Age (8000–3000 BC) (Brothwell & Sandison, 1967). Priorschi (1991) estimates that 6–10% of Neolithic skulls show trepanation. Neolithic trepanned skulls have been found in Great Britain, France, Spain, Portugal, North Africa, Italy, Switzerland, Belgium, Russia, Sweden and Poland (Piggott, 1940). Trepanation was also performed in South America (mainly Peru—see Campillo, 1984) and possibly North America as well as China, Japan and Afghanistan. This “primitive” trepanning has continued to the present time. It was witnessed in Kenya and Polynesia (see Margerett, 1967; Campillo, 1984), and in a Kisi settlement by Meschig & Schadenwaldt (1981). A report by Hershkovitz et al. (1991), from Israel suggests that two cranial lesions found at autopsy were the result of primitive-style trepanning which is continuing, unobserved, in the Middle East. It should be noted that trephining (including self-trephination) has recently gained in popularity in certain “new age” cults, where it is believed that it “releases tension” and increases “awareness”. It is likely that this procedure complements, rather than acts as a substitute for, chronic drug abuse to produce the same net effects in these individuals.

Trepanning/trephining was carried out for the following reasons.

1. Genuinely therapeutic i.e. neurosurgical reasons. It is postulated that trepanning/trephining by prehistoric people was done to relieve intracranial pressure following skull fractures. Although impossible to disprove, there is little evidence of trepanning/trephining in skulls with co-existent lesions which could have been treated surgically. On the other hand, Arabic surgeons of historic times have documented the use of burr holes to evacuate blood or relieve depressed fractures. An 18th/19th century example where this procedure has been undertaken to reduce the intracranial pressure due to an extradural haematoma following a linear fracture of the skull is illustrated (Figure 20). This procedure was also commonly carried out during the 18th and 19th centuries for relief of the symptoms of syphilis (Figure 21).

2. Ritual/magic/religious reasons. Evidence: this procedure appears principally to have been performed on living subjects. Numerous examples are available, however, of post-mortem “trepanning”. On the strength of the detailed examination of certain amulets in the form of roundels made from fragments of human crania from prehistoric and early historic times, it has been suggested that these are not, in the main, the products of roundels made from fragments of human crania from prehistoric and early historic times, it has been suggested that these are not, in the main, the products.
of trepanning the living subject. They are more likely to have been a by-product of ritual cannibalism and/or head hunting (for discussion, see Piggott, 1940). Other examples of trepanation from similar sites were, however, clearly performed on living individuals.

The commonest site is in the left parietal bone. A piece of skull from the left parietal bone is most often removed, although frontal or more rarely occipital bones may be involved, though the basal part of the occipital bone is never touched (Piggott, 1940; Campillo, 1984). Usually only a single hole is made but a skull with up to seven healed holes has been found (Oakley et al., 1959).

This procedure was predominantly carried out on males (Campillo, 1984), though women and children were also operated upon (see Prioreschi, 1991). Zones covered by muscle tended to be avoided (Campillo, 1984). The main methods of trephining include (1) drilling (trephining i.e. with a trephine/drill) with a hard sharp stone to produce a conical orifice with neat edges, (2) scraping with an abrasive stone to produce an ellipsoid orifice and a surrounding abrasive ring, and (3) cutting with an incisive instrument to produce a polygonal, square or circular hole.

In relation to (2) and (3), the term trepanning is more appropriate, as a hole is produced with an instrument other than a drill or trephine. Although trephine holes may have fairly distinctive features, depending on the method used and degree of healing, they may mimic parietal fenestrae or neat sabre wounds. This had led to considerable arguments in the literature (Mallin & Rathbun, 1976; Hoffman, 1979; Rathbun & Mallin, 1979). The different kinds of prehistoric trephination are illustrated diagrammatically in Figure 22.

According to Piggott (1940) "... the proportion of survivals from this operation ... is extremely high, as is evidenced by skulls showing a healthy growth of new bone around the edges of the opening ...". Thus survival must have occurred for at least a week or often for a very much longer period, sufficient for macroscopic evidence of bone healing to occur (see below). Since medical records are non-existent, the reasoning of these primitive surgeons is open to much discussion. We do not, however, wish to enter this debate on possible reasons for undertaking trephination. We are most interested in methods of distinguishing trephined holes from other lesions in the calvarium. It is important to note that trephining was carried out both post-mortem and ante-mortem. Thus a lack of healing around a trephine may be due to death supervening either before or soon after the trephination, or it may have been produced after the death of the individual (i.e. it may be a post-mortem hole). Depending on the state of the specimen, it may not be possible to distinguish between these various possibilities. The presence of "osteitis" (a localized area of porous bone visible to the naked eye or radiologically) indicating inflammation around a non-healed lesion, indicates at least that the trephine was performed ante-mortem. However, it is remotely possible that the trephine could have perforated a localized area of porous bone. For a detailed discussion see Stewart (1958). This tissue is demonstrated in a skull described by Broca (1874). Here, the lesion shows no obvious signs of healing but is surrounded by a circular area of porous bone suggesting an estimated survival of 7 days post-procedure.
Figure 22. Diagrams illustrating different kinds of prehistoric trephination [from Campillo (1984) with permission]. 1. Incomplete trephination by means of a drilling technique; 2. complete trephination by a drilling technique producing a conical orifice; 3. multiple trephinations, commonly used in pre-Colombian American cultures; 4. multiple trephinations to produce a single, large, circular orifice; 5. trephination using either an abrasion technique or bone and saw method; 6. isolated incisive trephination producing a fusiform groove (the “Indian-canoe” form of Latin American authors), possibly produced by scraping; 7. incisive polygonal trephination; 8. incisive or gouged trephination in a circular shape, allowing the removal of a complete disc of bone.
Trauma (Military and Civil)

Many examples of trauma-induced skull lesions are found in the Ballingall collection of military surgery, in the Department of Anatomy of the University of Edinburgh. Traumatic skull lesions may present as a localized lesion such as a gunshot wound or as a more generalized defect such as a skull fracture from “blunt” trauma such as may be induced by a sabre or other type of edged weapon (Figure 23). The same implement may induce a variety of different types of lesions depending on the mechanism of injury e.g. a gunshot wound may give an entrance and/or one or more exit wounds. Potential sequelae of such injuries such as infection or healing may also alter the appearance of the initial bone injury. It is also worth noting that the trauma may be induced post-mortem or, equally possibly, that the individual may not have survived the cranial surgery necessitated by the wound, for example, trephining to relieve a subdural haematoma caused by a depressed fracture, or to relieve the symptoms of syphilis (Figures 20 & 21).

What is of particular importance in this group is whether the trauma/injury caused the immediate or early death of the individual (as in many of the skulls in the Ballingall collection) or whether survival occurred, at least long enough to allow obvious evidence of healing to have taken place. Of equal importance is whether there is a reliable “history” associated with the specimen, and this forms the basis of our classification below. If this is available, then clearly there should be little problem with the diagnosis of the cause of the lesion. If no appropriate history is available, then it may not always be possible to come to a definitive diagnosis. If the latter is the case, it is then necessary to establish the circumstances surrounding the finding of the skull.

It is also clearly of particular importance, where at all possible, to distinguish between the various categories of lesions in order that a “most likely” cause is established taking all the features of the lesion into account, particularly its site, whether associated with a cranial suture or other common site of a congenital lesion (e.g. a parietal fenestra or foramina), and whether its borders show any characteristic features, for example, as seen in the case of primary malignancies or secondary (metastatic) invasive tumours. It is equally relevant in this context to draw attention to the usefulness in selected cases of sophisticated means of analysing the injury site in traumatic lesions. For example, much information has recently been obtained using scanning electron microscopy to analyse fatal blade injuries induced by Anglo-Saxon weapons (Wenham, 1989), and a similar approach is commonly used for the forensic examination of skeletal remains of more recent victims of trauma, some of whom may have been dismembered. In most cases, examination of the trauma site under the x 10 or x 25 magnification of a dissecting microscope is usually adequate to enable the principal diagnostic features to be recognized. In this way, for example, hairline depressed fractures of the cranial vault may be seen (Maples, 1986).

Means of distinguishing ante-mortem from post-mortem injuries

This is an appropriate place to make some general observations on this topic. While it may be possible to recognize certain characteristic features which would enable such a distinction to be made in the skull of a recently dead individual, particularly where the skull is reasonably intact and in good condition, the situation is often far from clear-cut when the skull has for a long period been defleshed and exposed to unfavourable environmental conditions.

As a general rule, evidence of an inflammatory reaction or the process of healing/repair indicates that the individual survived for some time after the traumatic lesion was inflicted. In a recently dead individual, histological examination of the lesion would, under normal circumstances, be available to confirm that this has indeed been the case, with the demonstration of an inflammatory response at the trauma site. If, however, the lesion was induced shortly before death (during what is sometimes termed the “peri-mortem” period) no evidence of an inflammatory response would be seen, despite the fact that the trauma was induced during the ante-mortem period.

When the skull is devoid of its overlying soft tissues, the margins of the lesion need to be examined...
particularly carefully, as the presence of a smooth or rounded border is usually indicative of the fact that sufficient time was available before death to allow some degree of healing to occur. Even 48 h is apparently sufficient to see evidence of proliferation of the osteogenic layer of the periosteum (Ham & Harris, 1956). For the forensic anthropologist or palaeopathologist usually only the calcified tissues remain, and it may take at least a week, and often more, before any perceptible “reactive” change is evident in the bone. If there is any evidence of healing, absorption or infection, then the trauma was undoubtedly ante-mortem.

The topic of blood-staining of the bones in the immediate area of trauma is discussed by Brothwell (1981) in the light of the extensive study carried out on this topic by Elliot Smith and Wood Jones (1910). Despite the fact that at that time chemical tests applied to such stained areas failed to reveal the presence of blood substances, it was suggested that this topic...
justified further detailed analysis. Attention was, however, drawn to the fact that some soils and other deposits produce patches of darker staining in bone through heterogeneous concentration of ferruginous material.

After death, the physical properties of bone change, as bones become harder and more brittle. They are consequently more liable to shatter on impact, and the fragments tend to become smaller as progressive drying of the bone occurs (Gurdjian, 1975). Furthermore, the fracturing characteristics are changed, and the cranial fracture patterns characteristic of fresh bone, such as concentric and radiating fracture lines (Figure 24), and stellate fractures, are not seen.

In individuals with extensive areas of calvarian thinning, post-mortem damage is particularly likely to occur, and would clearly have to be excluded before it was reasonable to suggest that a traumatic lesion to such an area of the skull was the likely cause of death. Damage produced by animals should also be excluded (teeth marks are usually present at the trauma site), and are also almost exclusively induced during the post-mortem period.

In all cases, extreme caution is necessary before a tentative diagnosis of ante-mortem trauma can be given. This is particularly the case when the skulls of young individuals are involved, because of the difficulty encountered in establishing whether a fracture was present along an unfused suture line, or the skull came apart during decomposition. The loss of wormian bones can also be readily mistaken for penetrating wounds to the skull. This equally applies when parietal foramina are incorrectly interpreted. The absence of diploe at the exposed edge of the lesion, however, strongly suggests a natural perforation rather than an induced lesion (for fuller discussion on ante-mortem versus post-mortem trauma, see Maples, 1986).

The aetiology of traumatic lesions may be classified as follows.

**Where history known**

*Ante-mortem injury*

1. Gunshot wounds—e.g. musket ball injuries.
   Entrance wound only (Figure 24).
   Entrance and exit wounds (Figure 25).
   Gunshot wound + infection (Figure 26).
(2) Shrapnel injury—a relatively low velocity “missile” injury such as may have occurred in the case of Fieschi (Figure 27).

(3) Sabre or sword wounds—varying degree of injury e.g. slicing of the outer table of the skull (Figures 28 & 29) to full thickness removal of a circumscribed region of the skull (Figure 23). Sabre injury + healing (Figures 30 & 31). In one example in the University collection, a skull is illustrated in which, following a sabre wound, a large segment of skull cap has been separated, rotated and has subsequently fused to the margin of the wound (Figure 31). In another case, an equally large portion of bone has been separated by a sabre cut, and the displaced plate of bone has fused to the surrounding bone.

(4) Post-mortem injury—Could be sustained from a variety of causes. Skull with bitemporal holes—sometimes seen in tribal skulls when exhibited as trophies. Occasionally, skulls may be seen that have a single “spike” hole in the region of the vertex. This is observed in the case of executed individuals whose heads were often displayed on a pole pour encourager les autres. It should also be noted that damage is relatively commonly sustained after burial or during the excavation procedure.

Where history unknown
In many cases the mechanism of injury is not known. The skull must therefore be assessed using all other information available as discussed in the summary. Causes other than trauma must be considered. It is interesting to note that where a history is known for the skulls in our collection the trauma has often been penetrating in nature. Where blunt trauma causes injury the individual is more likely to survive the insult, particularly if soft tissue rather than skeletal damage has been sustained.

If, however, a depressed fracture is induced, the site of the lesion may be of critical importance, as injury to certain sites (such as the region of the temple, particularly in the proximity of the pterion) is more likely to induce a subdural haemorrhage and/or haematoma, followed by compression of the brain and ultimately...
See p. 216 for legends to figures.
death of the individual if untreated. This equally applies where other vulnerable areas are exposed to blunt trauma (such as regions overlying the dural venous sinuses), when intracranial haemorrhage may result. Blunt injury may present as the following types of skull fractures.

1. Compound skull fracture (Figure 32).
2. Depressed skull fracture (Figure 33; see also Figure 27).
3. Horizontal linear fracture (Figure 34).
4. Multiple fractures+healing (Figure 34).

Summary

To summarize, the factors to consider in trying to determine the nature of a hole in the skull where no history is available include:

- Artefact—loss of wormian bone, degeneration, post-mortem damage.
- Age of individual—if young, likely to be congenital. If older, pathological or traumatic. Clearly, the location and morphological features of a hole are of critical importance in coming to a diagnosis of the most likely aetiology.
- Sex of individual—male more likely to be military trauma or trephining.
- Associated skeletal lesions—e.g. in cleidocranial dysostosis (absence of clavicles). Metabolic bone disorders e.g. Paget’s disease, hyperparathyroidism, rickets, osteomalacia.
- Era of specimen—various diseases prevalent at this time e.g. syphilis. Weaponry existing at this time e.g. shrapnel shells first used during Peninsular War.
- Geographical location—if found on battle site, usually traumatic.
- Location on skull—e.g. edged-weapon injury to calvarium, possibly sabre wound from horseman. Left parietal region, common site for trephining.
- Size and shape—square trephining. Serpiginous margin (i.e. creeping from part to part) e.g. syphilitic osteitis.
- Signs of healing—ante-mortem trephine.
- Depth of lesion—useful for defining type of pathological lesion. Inner table erosion—e.g. in pacchionian granulations, where inner table and diploe are affected. Outer table erosion—e.g. in bi-parietal thinning, where outer table and diploe are affected. Full thickness erosion—e.g. in congenital and developmental defects, in pathological lesions (as a consequence of infection or malignancy), following surgical intervention or military or civil trauma, and of post-mortem origin (see text for details).

Figure 34. Extensive linear skull fracture with evidence of healing. A skull presenting an extensive comminuted fracture of the frontal and upper part of the right parietal bones, with some of the fragments removed by operation. The breach is partly filled up by bony substance, and the edges rounded off. Part of the fractured parietal bone, and a triangular portion of the frontal bone, which seems to have been partially detached and depressed below the level of the surrounding parts, are firmly reunited. The patient, a workman in Portsmouth dockyard, had evidently survived the injury for a length of time, but the circumstances of his death are not known. A large portion of the right parietal bone and a small attached portion of the frontal bone have been displaced laterally to leave a 103 mm long linear aperture in the paramedian region (Anat. ref: Os Ba 8; Ball. ref: B7).
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References


Hackett, C. J. (1976). Diagnostic Criteria of Syphilis, Yaws and Treponarid (Treponematoses), and of some other Diseases in Dry Bones (for Use in Osteoarchaeology). Berlin: Springer-Verlag.


