Rare Temporal Bone Pathology of the Singa Calvaria From Sudan

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ABSTRACT Evidence has recently accumulated that the Singa calvaria from Sudan probably dates from Oxygen Isotope Stage 6 (>130 ka). Morphological studies have indicated a mixture of archaic and more modern human traits, but such analyses are complicated by the possibility that the vault is pathologically deformed, although the exact etiology has not been established. Now computed tomography (CT) has revealed that the right temporal bone lacks the structures of the bony labyrinth. The most likely cause of this rare pathological condition appears to be labyrinthine ossification, in which newly deposited bone obliterates the inner ear spaces following an infectious disease or occlusion of the labyrinthine blood supply. A possible cause of vascular compromise could have been the presence of an expanding acoustic neuroma in the internal acoustic meatus, which is suggested by a significantly wider right meatus compared with the left side. Interestingly, labyrinthine ossification is also consistent with the controversial diagnosis that an anemia caused the characteristic diploic widening at the parietal bosses, because prime etiological factors of ossification are among the common complications of some of these blood diseases. CT examination of the vault and a review of the literature suggest that a blood disorder may well have caused the unusual parietal morphology. Given the nature of these pathological conditions, the Singa individual must have experienced a period of considerable disability. The morphological evidence from the normal bony labyrinth on the left side and from the CT evaluation of the vault is consistent with the interpretation of Singa as a late archaic hominid or an early representative of *Homo sapiens* drawn from a population which might be directly ancestral to modern humans. Am J Phys Anthropol 107:41–50, 1998.

Since its discovery in 1924, the Singa calvaria has been rather neglected in studies of recent human evolution. This has occurred partly because of doubts about its geological age and partly because its unusual morphology has proved difficult to interpret. The specimen was discovered at the base of the seasonally exposed bed of the Blue Nile, in the district of Singa, about 320 km south east (upstream) of Khartoum. It was enclosed in a block of limestone calcrete (Woodward, 1938; Arkell et al., 1951). Putatively associated fauna and artefacts were also collected at both Singa and at the

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comparable site of Abu Hugar, some 15 km to the south (Arkell et al., 1951). Bate (in Arkell et al., 1951) described the faunal material, arguing that it perhaps dated to the early Upper Pleistocene. The only absolute age determination which was available was a radiocarbon date on a crocodile tooth from Abu Hugar of 17,300 ± 200 B.P., although it was recognized that there might have been contamination from younger carbon (Whiteman, 1971). The artefacts from Singa and Abu Hugar have been variously interpreted as nondiagnostic (Marks, 1968), Middle Palaeolithic (Lacaille in Arkell et al., 1951; McBurney, 1977), or perhaps even Acheulian (Bräuer, 1984) in affinities.

The calvaria itself has also been subjected to various interpretations. Woodward (1938) regarded it as a brachycephalic variant of the late Pleistocene "Boskop" race of southern Africa, while Rightmire (1984) and Clark (1988) regarded it as a late Pleistocene *Homo sapiens*. However, Tobias (1968), Brothwell (1974), and Stringer (1979) emphasized its archaic characters, with Tobias noting anterior resemblances to Broken Hill, Brothwell pointing out "neandertaloid" features, and Stringer arguing for metrical similarities in shape to the Jebel Irhoud crania (Fig. 1). Multivariate analyses by Bräuer (1984) demonstrated the unique parietal morphology of Singa.

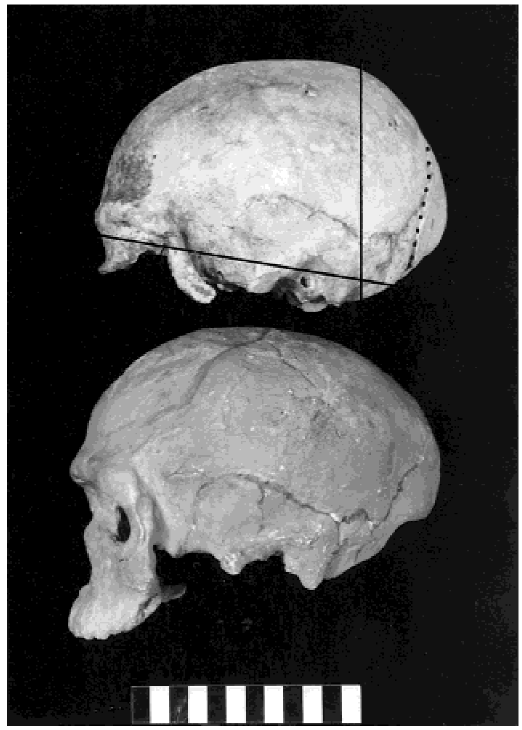

Fig. 1. Left lateral view of the Singa calvaria (**top**) in comparison with a cast of the Jebel Irhoud 1 cranium (**bottom**). The transverse and coronal planes of the CT scans of Singa, given in Figs. 2 and 3, are indicated by solid lines and the fracture posterior to the parietal bosses by the dotted line. Scale, 12 cm.

Brothwell (1974) first raised the question of possible pathology, arguing that its unusual breadth and parietal expansion was the result of compensatory lateral growth following premature synostosis of the sagittal suture. Stringer (1979) agreed that the parietal region was unusual and that the bregma-lambda chord was especially short. Reevaluation after preparation of the specimen revealed unusual unilateral expansion of the sphenoid sinus and that the marked vault thickness at the parietal bosses is associated with an expanded diploic layer almost four times the width of the compact bone (Stringer et al., 1985). However, an attempt to identify the etiology was not successful. In particular, no conclusive evidence was found for the hypothesis that the vault shape and diploic thickening were the result of anemia, an opinion reiterated in Stuart-Macadam (1992). On the other hand, to Webb (1990) the morphology suggests that the Singa individual suffered from some form of blood disease.

More recently, attempts have been made to date the Singa calvaria using the putatively associated fauna and calcareous matrices. Electron spin resonance (ESR) dates were carried out on a supposedly associated horse and bovid tooth from Singa, suggesting an oxygen isotope stage 5–6 age (Grün and Stringer, 1991), while further ESR and uranium series dates, including some on the matrix around and within the calvaria, suggested an age in excess of 133 ka (McDermott et al., 1996). Moreover, chemical analyses of matrix on the calvaria and fauna supported their association, while modelling of uranium history clearly indicated a closer approximation to linear uptake (LU). This suggests that the LU ESR age estimates on the associated teeth in the range 140–160 ka are likely to be most accurate. Thus, the Singa hominid probably does date from oxygen isotope stage 6 and therefore predates all well-dated specimens of *Homo sapiens sensu stricto*. It thus represents a rare sample from a population which might approximate the ancestral one for all modern humans. Knowledge of its morphology and the extent to which this might have been modified by possible pathological change is therefore of considerable importance for human evolutionary studies.

In the context of the ongoing morphological study of Singa, the internal structures of its temporal bones were investigated using computed tomography (CT). The principal aim was to image the bony labyrinth, which houses the sense organs of hearing and balance and has been shown to provide valuable information in studies of hominid locomotor behavior and phylogeny (Spoor et al., 1994; Hublin et al., 1996). Rather unexpectedly, the CT scans revealed the absence of the structures of the right bony labyrinth. This paper describes this unusual pathological condition not found previously in any fossil hominid and discusses the possible etiology. In a wider context, the question arises whether this temporal bone pathology can be linked with, and thus increase an understanding of, the other areas of abnormal morphology. Identifying a single underlying disorder would help in attempts to eliminate pathological characteristics from analyses of Singa's phylogenetic affinities. Apart from evaluating the pathology of the right temporal bone, this paper also briefly considers the normal labyrinthine morphology on the contralateral side.

METHODS

The method of visualizing the internal morphology of the temporal bone by CT follows the procedures described in Spoor and Zonneveld (1994, 1995). The CT scans were made with a Philips Tomoscan 350 scanner (University College Hospital, London, August 1991) with an exposure of 480 mA at 120 kVp tube voltage and a slice thickness of 1.5 mm. By replacing the standard soft tissue beam hardening correction based on a plexiglas calibration with a special aluminium calibration, an extended Hounsfield scale (CT number scale) was created to avoid overflow artefacts caused by the high density of the mineralized bone and the attached matrix (Spoor and Zonneveld, 1994). Scans with a field of view of 240 × 240 mm (matrix 256 × 256) were made in the transverse (nasion-biporionic), sagittal, and coronal planes as indicated in Figure 1 and demonstrated in Figure 2a. Zoom reconstructions of the temporal bone regions were made with a field of view of 80 × 80 mm (Fig. 2b,c). The scans were made contiguously, with an additional overlapping (slice increment 0.75 mm) transverse scan at the level of the lateral semicircular canal and an overlapping sagittal scan at the level of the common crus. Measurements to the nearest tenth of a millimeter of the left labyrinth were taken from the CT scans following the method and definitions described in Spoor and Zonneveld (1995). Measurements of the lateromedial length and the inferosuperior and anteroposterior diameters of the internal acoustic meati were taken from transverse and coronal scans.

The endocranial surface morphology of the petrous pyramids was investigated using an operating microscope with a coaxial light source and a dental mirror.

DESCRIPTION

The left temporal bone is normally developed in all aspects. Most of its internal spaces are filled with matrix of a higher density than the surrounding bone (Fig. 2c). In the bony labyrinth, the superior parts of the anterior and posterior semicircular canals are air-filled, and an area of reduced density in the center of the vestibule suggests the presence of a bone fragment (Fig. 2c).

The labyrinthine measurements that have been shown to be important functionally and phylogenetically in Middle and Late Pleistocene hominids are the radii of curvature of the semicircular canals and the sagittal labyrinthine index (SLI), which describes the proportion of the posterior canal situated inferior to the plane of the lateral canal (Spoor and Zonneveld, 1994; Hublin et al., 1996). The radii of curvature of the anterior, posterior, and lateral canals of the

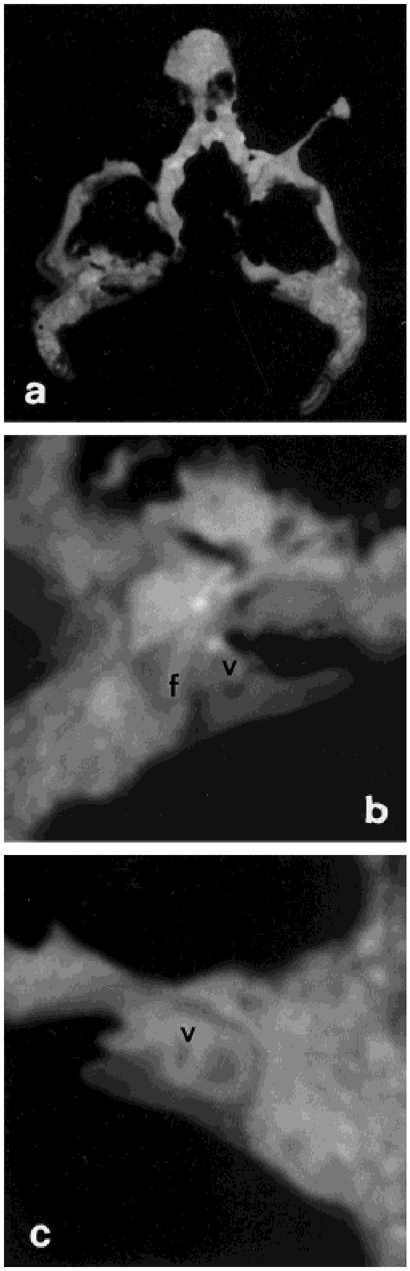

Fig. 2. **a:** Transverse overview CT scan at the level indicated in Fig. 1. **b,c:** Zoom reconstruction of the right (b) and left (c) temporal bone areas, showing the vestibule (v) and the lateral semicircular canal of the normal labyrinth on the left and the irregularly shaped vestibule (v) as well as the intact second part of the facial canal (f) on the right side.

left Singa labyrinth are 3.0, 3.1, and 2.3 mm, respectively, and its SLI is 46%.

Externally the right temporal bone is similar in morphology to the left one, including the overall shape of the petrous part and delicate details such as the endocranial aperture of the vestibular aqueduct. Its internal morphology is marked by the absence of the structures of the bony labyrinth. An irregular space, partly filled with matrix, takes the position of the vestibule (Fig. 2b). Moreover, a faint spot of slightly higher density than the surrounding bone marks the area of the cochlea, and an air-filled structure could possibly represent a remnant of the superiormost part of the anterior semicircular canal. In contrast, the tympanic cavity and all three segments of the facial canal are normally developed (Fig. 2b).

CT scans reveal that the middle part of the right internal acoustic meatus has a considerably larger vertical diameter than the left side, whereas both pori are approximately equal in size. Measurements of the length and the inferosuperior and anteroposterior diameters, halfway between the porus and fundus, are 12.5, 6.4, and 4.4 mm, and 12.0, 4.2, and 4.0 mm for the right and left meati, respectively. Direct observation of the largely matrix-free right meatus confirms that the larger dimensions are not due to erosion. Its fundus is normal in morphology and shows a well-developed falciform crest. The actual spiral part of the tractus spiralis foraminosus is covered by a remnant of matrix, but all other foramina can be observed.

DISCUSSION

The paleopathology of hominid fossils is of importance beyond a basic interest in the expression of disease in skeletal remains. In the case of Singa, appreciating the full pattern of pathologies and the impact on the morphology is crucial in assessing its phylogenetic affinities. Moreover, it may help in understanding the paleoepidemiology of the disease involved, and the nature of the symptoms may reflect on social aspects of early hominid behavior.

A first consideration is the differential diagnosis of the absence of labyrinthine structures in Singa's right petrous pyramid.

Possible causes include postmortem damage owing to the fossilization process, developmental deformity of the otic capsule, and bony obliteration of the labyrinthine lumen after a normal development (e.g., for recent reviews of inner ear anomalies see Phelps and Lloyd, 1990; Ballenger, 1991; Som and Curtin, 1996). The former option can be discarded because the undistorted preservation of the petrous pyramid, in particular of fragile structures such as the facial canal and the falciform crest, clearly demonstrates that this area is free of postmortem damage capable of obscuring the semicircular canals and the cochlea.

The presence of an irregular central space instead of a normal labyrinth could be interpreted as a congenital, so-called common cavity deformity, a persistent otocyst resulting from arrested development (Jackler et al., 1987; Kavanagh and Magill, 1989). However, this is unlikely, as the specimen does not show the hypoplastic petrous pyramid and internal acoustic meatus that characterize such cases (Valvassori et al., 1969; Phelps, 1974; Isenberg and Tubergen, 1979; Kavanagh and Magill, 1989). Also, in the area where the lateral semicircular canal would normally be located, the medial tympanic wall shows a prominence (Fig. 2b), indicative of full development of the otic capsule. In contrast, this area is flat in cases of congenital dysplasia of the common cavity type because the lateral canal never developed (Curtin, 1988; Weissman and Kamerer, 1993). Likewise, the normal morphology of the fundus of the internal acoustic meatus also points at full development of the labyrinthine structures.

Labyrinthine ossification, in which newly deposited bone obliterates some or all of the inner ear spaces, appears to be the most likely explanation of the abnormal morphology seen in the Singa petrous pyramid. Many etiological factors have been reported, such as fractures, advanced otosclerosis, and compromised vascularization, but most often it is secondary to chronic infection of the membranous labyrinth (labyrinthitis ossificans) (see reviews in Suga and Lindsay, 1977; Swartz et al., 1985; DeSouza et al., 1991; Muren and Bredberg, 1997). Infections reach the labyrinth from the tympanic cavity (chronic otitis media), the meninges (meningitis), or occasionally via the bloodstream (septicaemia). Complete ossification of the labyrinth may take many years (Paparella and Sugiura, 1967) but has also been observed within a period of a year (Hoffman et al., 1979). Even in cases of total ossification, the internal acoustic meatus and the cochlear and vestibular aqueducts, structures directly connected with the labyrinth, are not affected (Sugiura and Paparella, 1967).

In the Singa specimen, ossification appears to have proceeded to all labyrinthine spaces except the vestibule and perhaps the most superior part of the anterior semicircular canal. Paparella and Sugiura (1967) observed that newly formed bone in the cochlea can be distinguished histologically from endochondral bone, but in the semicircular canals it completely blends with bone of the otic capsule. If we keep in mind that any contrast between the otic capsule and newly formed bone is likely to be diminished by the fossilization process, this perhaps explains why in the CT scans of the Singa specimen only the cochlear area shows a vague density difference, whereas no outlines of the semicircular canals are visible. The cause of the ossification in the Singa specimen is difficult to establish with certainty, because most etiological factors, especially the primary infections leading to labyrinthitis, are not reflected in the morphology which is fossilized. This is the second reported case of pathology of the otic capsule in a hominid fossil. Previously, CT scans revealed a likely case of cochlear otospongiosis in the Forbes Quarry Neanderthal from Gibraltar (Zonneveld and Wind, 1985).

A striking feature of the right petrous pyramid of the Singa specimen, other than the obliterated labyrinth, is the 2.2 mm or 52% greater vertical diameter of its internal acoustic meatus when compared with the left side. Several studies have investigated the bilateral variation of the meatus dimensions in modern humans, using radiological or casting methods (Ebenius, 1934; Camp and Cilley, 1939; Graf, 1952; Valvassori and Pierce, 1964; Amjad et al., 1969; Papangelou, 1972). Of the total of 686 pairs of temporal bones thus assessed, only 1.6%

TABLE 1. Bilateral differences between the vertical (inferosuperior) diameter of the internal acoustic meatus in modern humans, listing the percentage (%) of the sample with a specified difference

Source	Number of pairs	Difference	Incidence in %
Ebenius (1934)	100	Over 2.0 mm	1
Camp and Cilley (1939)	250	2.0 mm or more	2
Graf (1952)	100	Over 1.5 mm	0
Valvassori and Pierce (1964)	100	2.0 mm or more	0
Amjad et al. (1969)	15	Over 1.0 mm	0
Papangelou (1972)	121	2.0 mm or more	4
Total	686	Over 2.0 mm	1.6

showed a bilateral difference in vertical diameter of more than 2 mm (Table 1). This incidence led Valvassori (1969) to consider a meatus abnormal if it shows a widening of 2 mm or more when compared with the same segment on the opposite side, a criterion subsequently cited in review works such as Schuknecht and Gulya (1986) and Phelps and Lloyd (1990). The difference in vertical diameter of 2.2 mm in the Singa calvaria falls in this category and deserves further consideration.

Abnormal widening is usually associated with expanding, space-occupying lesions inside the meatus, such as facial nerve neuromas, arachnoid cysts, and, by far the most common, acoustic neuromas (Phelps and Lloyd, 1990). Dolan et al. (1978) demonstrate that exceptions occur by presenting nine cases where widening did not correspond with any space-occupying lesion of the meatus. However, these cases were found by reviewing an unspecified body of archival case material and could easily represent the 1.6% widening also found in anatomical studies.

If the widening is confined to the middle part of the internal acoustic meatus, as seen in the Singa specimen, it most likely indicates a small intrameatal acoustic neuroma (primary intracanalicular eighth nerve schwannoma: Goodhill, 1979). The effects of such an expanding lesion include compression not only of the nerves but also of the labyrinthine artery, the principle blood supply to the inner ear. This results in degenerative changes of the membranous labyrinth (Perez De Moura, 1967; Belal, 1979), and both clinical cases and experimental animal studies indicate that arterial occlusion results in bony obliteration of the labyrinth (Belal, 1979; Kimura and Perlman, 1958; Paparella and Sugiura, 1967). Hence, an expanding intrameatal lesion in the Singa specimen, as suggested by the increased inferosuperior diameter, is a possible candidate for the cause of the observed labyrinthine ossification through the mechanism of vascular compromise. However, a nonpathological origin of the bilateral difference in meatal morphology cannot be completely excluded, given its occasional occurrence in modern humans (Dolan et al., 1978)

Neither diploic thickening of the parietals nor expansion of the sphenoid sinus, both previously described as unusual morphology of Singa (Stringer et al., 1985), are known to result directly from any of the possible causes of labyrinthine ossification or from an expanding lesion in the internal acoustic meatus. However, a potential common underlying cause for two of these, diploic expansion and labyrinthine ossification, is a hereditary or acquired blood disorder. Anemias are the best known cause of an increase of the diploic layer (Diggs, 1967; Moseley, 1974; Konotey-Ahulu, 1991; Ortner and Putschar, 1985; Serjeant, 1992; Stoker, 1997), and sensorineural hearing loss has a well-known association with sickle cell anemia (Cole and Jahrsdoerfer, 1988; Donegan et al., 1982; Serjeant, 1992; Odetoyinbo and Adekile, 1987). The likely mechanism underlying this relationship is that common complications of sickle cell disease, vasoocclusion, meningitis, or septicaemia, result in inner ear hemorrhage, avascular necrosis, and labyrinthitis (Cole and Jahrsdoerfer, 1988; Donegan et al., 1982; Serjeant, 1992; Odetoyinbo and Adekile, 1987). As discussed previously these are among the prime etiological factors of labyrinthine ossification. Degenerative changes to the cochlea and hearing loss have also been associated with acquired, mainly iron

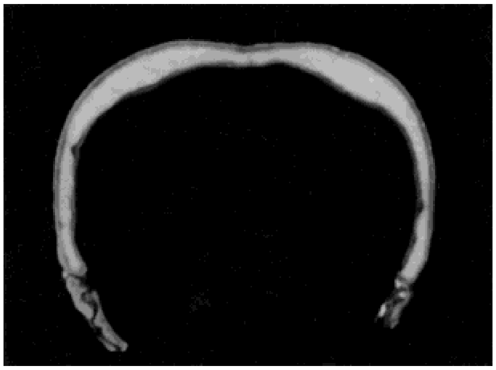

Fig. 3. Coronal CT scans of the Singa calvaria at the level of maximum parietal bossing, as indicated in Fig. 1. Note that the scan plane is not entirely perpendicular to the external surface, but this does not affect the relative proportions of the tables.

deficiency anemia (Yassin and Taha, 1965; Morrison and Booth, 1970; Morrison, 1978; Sun et al., 1992a,b). Furthermore, bacterial or viral infections, capable of invoking labyrinthitis, may also cause hemolytic anemias or an exacerbation of a previously existing chronic anemia (Dacie, 1995).

The finding that the newly discovered temporal bone pathology is consistent with anemia asks for a reexamination of the evidence that led Stringer et al. (1985) to conclude that there is little support for this diagnosis. The study defined seven criteria describing bony change in anemia that are visible in radiographs (further commented on in Stuart-Macadam, 1987) and reported that the Singa calvaria exhibits only one of these: diploic thickening of the parietals. The three other criteria pertaining to the vault, thinning of the outer table, hair-on-end trabecular appearance, and diploic texture changes, could not be detected. These observations must have been based on inspection of the fracture surface posterior to the parietal bosses (Fig. 1), because radiographs of Singa are inherently incapable of revealing details of the vault morphology owing to the complete saturation of the diploic layer by matrix of a higher density than the fossilized bone (Figs. 2, 3). Webb (1990) assessed this fracture, and reported that both the inner and outer table are thin, each with a thickness of less than 1 mm.

With CT, it is now possible to visualize the vault structure at the center of the parietal bosses, and a coronal scan suggests a similar vault morphology as seen at the fracture (Fig. 3). The outer table does not appear to be markedly thinner than the inner table, and the thickness of neither table is specifically reduced at the centers of the bosses where the diploic layer is most expanded. The thickened diploë is imaged as a virtually homogenous layer, suggesting a fine trabecular pattern. However, establishing the presence in the matrix-saturated diploic layer of texture changes or radially oriented bone spicules, associated with the hair-on-end appearance in radiographs, is beyond the limitation of the spatial resolution of the CT scans. Nevertheless, Singa clearly does not show the severe hair-on-end morphology illustrated in the literature (e.g., Diggs, 1967; Moseley, 1974; Serjeant, 1992; Stoker, 1997) because it lacks the strongly eroded outer table with honeycombed compartments of subperiosteal new bone giving this radiological appearance (Ortner and Putschar, 1985). Webb (1990) suggested that, in addition to the two parietal bosses, Singa also shows expansion of the frontal, thus implying that the well-rounded shape of the forehead is pathological in origin. However, this is not supported by the evidence from CT scans, which do reveal a well-developed frontal sinus but no obvious widening of the frontal diploic layer.

Reviews of skeletal involvement in various types of blood disorders (Diggs, 1967; Konotey-Ahulu, 1991; Moseley, 1974; Ortner and Putschar, 1985; Serjeant, 1992; Stoker, 1997) suggest that the radiographic criteria used by Stringer et al. (1985) to assess Singa pertain predominantly to the most severe manifestations of anemia. Indeed, Singa clearly does not show the severe cranial changes, such as of strong erosion of the outer vault table and inhibited development of the air sinuses, that mark thalassemias. On the other hand, it is more consistent with the pattern observed for sickle cell disease, hereditary spherocytosis, and iron deficiency anemia, in which cranial changes are uncommon and restricted to widening of the diploic layer with possible thinning of the outer table and only occasionally the hair-on-end radiographic appearance. Diploic widening and bossing that is restricted to the parietals, as seen in Singa, is the most common pattern in these forms of anemia. Diploic expansion without obliteration of the outer table or the hair-on-end appearance has also been reported in Peruvian crania in the Hrdlička collection (Ponec and Resnick, 1984), in Willandra Lake Hominid 50 (Webb, 1990), and in some of the Kanjera hominids (Plummer and Potts, 1995). The possibility raised in the latter study that remodelling may have brought about an outer table of normal thickness after the demand for hematopoietic tissue had declined with adulthood deserves further investigation, for example by scanning electron microscopy or histological examination of the Kanjera specimens and the Singa calvaria.

The suggestion of Webb (1990) that the diploic expansion seen in Singa is indicative of hyperplastic marrow associated with some form of blood disease seems to be a plausible one, given that its parietal vault morphology appears distinctly different from that in other fossil hominids (Bräuer, 1984; Stringer, 1979; Stringer et al., 1985) and modern humans, in a way that is broadly consistent with the manifestation of some modern-day anemias. Differentiating various types of anemia in dry bone specimens is problematic (Ortner and Putschar, 1985), and in the case of Singa such attempts would be particularly meaningless when considering the age of the specimen in relation to the possible age of hereditary anemias. For example, estimates of the origin of the sickle cell gene vary from just 2,000 years ago (Kurnit, 1979; Wiesenfeld, 1967) up to a range of 70,000–150,000 years ago (Solomon and Bodmer, 1979). Singa thus likely predates the origin of sickle cell disease in its current forms, and there is a real possibility that it suffered from a blood disorder causing an increased demand for hemopoietic marrow space not known today. Future progress in interpreting histological features of diploë modified by hyperplastic marrow or perhaps analysis of ancient biomolecules could lead to a more definite diagnosis. Moreover, current systematic quantitative assessment of the vault structure in extant and fossil hominids on the basis of CT scans (Garcia, 1995) will lead to a more secure differentiation between pathologies and extremes of normal anatomical variation.

Extensive labyrinthine ossification as seen in Singa is rare, and the minimum time reported for this level to be reached is a year (Hoffman et al, 1979). It is therefore testimony to the fact that the Singa individual survived the original insult affecting the labyrinth, whether this was vascular compromise, labyrinthitis following an infectious disease, or some other cause, regardless of a possible association with anemia. The consequence was complete and permanent unilateral loss of auditory and vestibular function, which results in deafness, spontaneous nystagmus, vertigo, postural imbalance, and ataxia (Curthoys and Halmagyi, 1996; Swartz et al., 1985; Schwarz and Tomlinson, 1994). The static symptoms gradually recover over a period varying from a few days to a few months in a process known as vestibular compensation (Curthoys and Halmagyi, 1996; Schwarz and Tomlinson, 1994). An expanding lesion in the internal acoustic meatus would have put pressure not only on the labyrinthine vessels and the vestibulocochlear nerve but also on the facial nerve. However, this rarely results in gross facial dysfunction (Phelps and Lloyd, 1990).

It follows from the inferred pathological history that the Singa individual must have gone through a period characterized at the very least by severely impaired body coordination and at the worst by a life-threatening infection such as bacterial meningitis or septicaemia. In addition, there was the considerable handicap of permanent unilateral hearing loss and possibly any symptoms associated with a blood disorder. Given these circumstances, it is debatable to what extent the survival of the Singa individual would have required care by others.

The morphology of Singa's well-preserved left bony labyrinth resembles that of both modern humans and *Homo erectus*. It lacks the derived features of the Neanderthal labyrinth, most notably a relatively small posterior semicircular canal with a markedly inferior position relative to the plane of the lateral canal (Hublin et al, 1996). Hence, the labyrinth does not support the suggestion of Brothwell (1974) that the Singa calvaria shows neanderthaloid influences. On the other hand, the evidence from both the labyrinth and from CT examination of the vault is consistent with the interpretation of most recent studies, which take into account the pathological parietal shape and regard Singa as either a late archaic hominid or an early and primitive representative of *Homo sapiens* (Stringer, 1979; Stringer et al, 1985; McDermott et al., 1996). These studies highlighted resemblances between Singa and the Jebel Irhoud crania (Fig. 1), which may be of comparable age (Grün and Stringer, 1991). Both may be drawn from a population ancestral to modern humans, which lived in Northern Africa some 150,000 years ago.

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LITERATURE CITED

Amjad AH, Scheer AA, and Rosenthal J (1969) Human internal auditory canal. Arch. Otolaryngol. *89:*709–714.

Arkell AJ, Bate DMA, Wells LH, and Lacaille AD (1951) Fossil Mammals of Africa *2:*1–50.

Ballenger JJ (1991) Diseases of the Nose, Throat, Ear, Head and Neck, 14th ed. Philadelphia: Lea and Febiger.

Belal A (1979) The effects of vascular occlusion on the human inner ear. J. Laryngol. Otol. *93:*955–968.

Bräuer G (1984) A craniological approach to the origin of anatomically modern *Homo sapiens* in Africa and implications for the appearance of modern Europeans. In F Smith and F Spencer (eds.): The Origins of Modern Humans. New York: Alan Liss, pp. 327–410.

Brothwell DR (1974) The Upper Pleistocene Singa skull: A problem in palaeontological interpretation. In W Bernhard and A Kandler (eds.): Bevoelkerungsbiologie. Stuttgart, Carl Fischer, pp. 534–545.

Camp JD, and Cilley EIL (1939) The significance of asymmetry of the pori acustici as an aid in diagnosis of eighth nerve tumors. Am. J. Roentgenol. *41:*713–718.

Clark JD (1988) The Middle Stone Age of East Africa and the beginnings of regional identity. J. World Prehist. *2:*235–305.

Cole RR and Jahrsdoerfer RA (1988) Sudden hearing loss: An update. Am. J. Otol. *9:*211–215.

Curthoys IS, and Halmagyi GM (1996) How does the brain compensate for vestibular lesions? In RW Baloh and GM Halmagyi (eds.): Disorders of the Vestibular System. New York: Oxford University Press, pp. 145–154.

Curtin HD (1988) Congenital malformations of the ear. Otolaryngol. Clin. N. Am. *21:*317–336.

Dacie J (1995) The Haemolitic Anaemias, Vol. 4. Edinburgh: Churchill Livingstone.

DeSouza C, Paparella MM, Schachern P, and Yoon TE (1991) Pathology of labyrinthine ossification. J. Laryngol. Otol. *105:*621–624.

Diggs LW (1967) Bone and joint lesions in sickle-cell disease. Clin. Orthop. *52:*119–143.

Dolan KD, Babin RW, and Jacoby CG (1978) Asymmetry of the internal auditory canals without acoustic neuroma. Ann. Otol. Rhinol. Laryngol. *87:*815–820.

Donegan JO, Lobel JS, and Gluckman JL (1982) Otolaryngologic manifestations of sickle cell disease. Am. J. Otolaryngol, *3:*141–144.

Ebenius B (1934) Results of examination of the petrous bone in auditory nerve tumors. Acta Radiol. *15:*284–290.

Garcia RA (1995) Application de la tomographie informatisée et de l'imagerie virtuelle a l'analyse quantitative de la structure des parois crâniennes. Mémoire de DEA, Bordeaux, Université de Bordeaux 1.

Goodhill V (1979) Ear: Diseases, Deafness, and Dizziness. Hagerstown: Harper and Row.

Graf K (1952) Geschwuelste des Ohres und des Kleinhirnbrueckenwinkels. Stuttgart: Thieme.

Grün R, and Stringer CB (1991) Electron spin resonance dating and the evolution of modern humans. Archaeometry *33:*153–199.

Hoffman RA, Brookler KH, and Bergeron RT (1979) Radiologic diagnosis of labyrinthitis ossificans. Ann. Otol. Rhinol. Laryngol. *88:*253–257.

Hublin J-J, Spoor F, Braun M, Zonneveld F, and Condemi S (1996) A late Neanderthal from Arcy-sur-Cure associated with Upper Palaeolithic artefacts. Nature *381:*224–226.

Isenberg SF, and Tubergen LB (1979) Unilateral complete aplasia of the inner ear with associated tracheoesophageal fistula: report of a case. Otolaryngol. Head Neck Surg. *87:*435–439.

Jackler RK, Luxford WM, and House WF (1987) Congenital malformations of the inner ear, a classification based on embryogenesis. Laryngoscope Suppl. *40:* 2–14.

Kavanagh KT, and Magill HL (1989) Michel dysplasia: Common cavity deformity. Pediatr. Radiol. *19:*343–345.

Kimura R, and Perlman HB (1958) Arterial obstruction of the labyrinth. Ann. Otol. Rhinol. Laryngol. *67:*5–40.

Konotey-Ahulu FID (1991) The Sickle Cell Disease Patient. London: MacMillan.

Kurnit DM (1979) Evolution of sickle cell variant. Lancet *1:* 104.

Marks A (1968) The Khormusan: An Upper Pleistocene industry in Sudanese Nubia. In F Wendorf (ed.): The Prehistory of Nubia. Dallas: Fort Burgwin Research Center and Southern Methodist University, pp. 315–319.

McBurney CBM (1977) Archaeology and the *Homo sapiens sapiens* Problem in Northern Africa. Amsterdam: Netherlands Museum for Anthropology and Prehistory.

McDermott F, Stringer C, Grün R, Williams GT, Din VK, and Hawkesworth CJ (1996) New Late-Pleistocene uranium-thorium and ESR dates for the Singa hominid (Sudan) J. Hum. Evol. *31:*507–516.

Morrison AW (1978) Acute deafness. Br. J. Hosp. Med. *19:*237–249.

Morrison AW, and Booth JB (1970) Sudden deafness: An otological emergency. Br. J. Hosp. Med. *4:*287–298.

Moseley JE (1974) Skeletal changes in the anemias. Semin. Roentgenol. *9:*169–184.

Muren C, and Bredberg G (1997) Post meningitic labyrinthine ossification primarily affecting the semicircular canals. Eur. Radiol. *7:*208–213.

Odetoyinbo O, and Adekile A (1987) Sensorineural hearing loss in children with sickle cell anemia. Ann. Otol. Rhinol. Laryngol. *96:*258–260.

Ortner DJ, and Putschar WGJ (1985) Identification of Pathological Conditions in Human Skeletal Remains. Washington, DC: Smithsonian Institution Press.

Papangelou L (1972) Study of the human internal auditory canal. Laryngoscope *82:*617–624.

Paparella MM, and Sugiura S (1967) The pathology of suppurative labyrinthitis. Ann. Otol. Rhinol. Laryngol. *76:*554–586.

Perez De Moura LF (1967) Inner ear pathology in acoustic neurinoma. Arch. Otolaryngol. *85:*125–133.

Phelps PD (1974) Congenital lesions of the inner ear, demonstrated by tomography. Arch. Otolaryngol. *100:* 11–18.

Phelps PD, and Lloyd GAS (1990) Diagnostic Imaging of the Ear. 2nd ed. London: Springer Verlag.

Plummer T, and Potts R (1995) Hominid fossil sample from Kanjera, Kenya: Description, provenance, and implications of new and earlier discoveries. Am. J. Phys. Anthropol. *96:*7–23.

Ponec DJ, and Resnick D (1984) On the etiology and pathogenesis of porotic hyperostosis of the skull. Invest. Radiol. *19:*313–317.

Rightmire GP (1984) *Homo sapiens* in Sub-Saharan Africa. In F Smith and F Spencer (eds.): The Origins of Modern Humans. New York: Alan Liss, pp. 295–325.

Schuknecht HF, and Gulya AJ (1986) Anatomy of the Temporal Bone with Surgical Implications. Philadelphia: Lea and Febiger.

Schwarz DWF, and Tomlinson RD (1994) Physiology of the vestibular system. In RK Jackler and DE Brackmann (eds.): Neurotology. St. Louis: Mosby, pp. 59–98.

Serjeant GR (1992) Sickle Cell Disease, 2nd ed. Oxford: Oxford University Press.

Solomon E, and Bodmer WF (1979) Evolution of sickle cell variant. Lancet *i:*923.

Som PM, and Curtin HD (1996) Head and Neck Imaging, 3rd ed. St. Louis: Mosby.

Spoor CF, and Zonneveld FW (1994) The bony labyrinth in *Homo erectus*, a preliminary report. Cour. Forschungsinst. Senckenberg *171:*251–256.

Spoor CF, and Zonneveld FW (1995) Morphometry of the primate bony labyrinth: A new method based on high-resolution computed tomography. J. Anat. *186:* 271–286.

Spoor F, Wood B, and Zonneveld F (1994) Implications of early hominid labyrinthine morphology for the evolution of human bipedal locomotion. Nature *369:*645–648.

Stoker DJ (1997) Myeloproliferative and similar diseases. In RG Grainger and DJ Allison (eds.): Diagnostic Radiology, 3rd ed. New York: Churchill Livingstone, pp. 1691–1712.

Stringer CB (1979) A re-evaluation of the fossil human calvaria from Singa, Sudan. Bull. Brit. Mus. Nat. Hist. (Geol.) *32:*77–83.

Stringer CB, Cornish L, and Stuart-Macadam P. (1985) Preparation and further study of the Singa skull from Sudan. Bull. Brit. Mus. Nat. Hist. (Geol.) *38:*347–358.

Stuart-Macadam P (1987) A radiographic study of porotic hyperostosis. Am. J. Phys. Anthropol. *74:*511–520.

Stuart-Macadam P (1992) Cranial thickening and anemia: Reply to Dr Webb. Am. J. Phys. Anthropol. *88:*109–110.

Suga F, and Lindsay JR (1977) Labyrinthitis ossificans. Ann. Otol. Rhinol. Laryngol. *86:*17–29.

Sugiura S, and Paparella MM (1967) The pathology of labyrinthine ossification. Laryngoscope *77:*1974–1989.

Sun AH, Wang ZM, Xiao SZ, Li ZJ, Ding JC, Li JY, and Kong LS (1992a) Idiopathic sudden hearing loss and disturbance of iron metabolism. ORL *54:*66–70.

Sun AH, Wang ZM, Xiao SZ, Li ZJ, Zhenge Z, and Li JY (1992b) Sudden sensironeural hearing loss induced by experimental iron deficiency in rats. ORL *54:*246–250.

Swartz JD, Mandell DM, Faerber EN, Popky GL, Ardito JM, Steinberg SB, and Rojer CL (1985) Labyrinthine ossification: Etiologies and CT findings. Radiology *157:*395–398.

Tobias PV (1968) Middle and early Upper Pleistocene members of the genus *Homo* in Africa. In G Kurth (ed.): Evolution und hominisation. Stuttgart: Gustav Fischer, pp. 176–194.

Valvassori GE (1969) The abnormal internal auditory canal: The diagnosis of acoustic neuroma. Radiology *92:* 449–459.

Valvassori GE, and Pierce RH (1964) The normal internal auditory canal. Am. J. Roentgenol. *92:*1232–1241.

Valvassori GE, Naunton RF, and Lindsay JR (1969) Inner ear anomalies: clinical and histopathological considerations. Ann. Otol. Rhinol. Laryngol. *78:*929–938.

Webb S (1990) Cranial thickening in an Australian hominid as a possible palaeoepidemiological indicator. Am. J. Phys. Anthropol. *82:*403–411.

Weissman JL, and Kamerer DB (1993) Labyrinthitis ossificans. Am. J. Otolaryngol. *14:*363–365.

Whiteman AJ (1971) The Geology of the Sudan Republic. Oxford: Clarendon Press.

Wiesenfeld SL (1967) Sickle-cell trait in human biological and cultural evolution. Science *157:*1134–1140

Woodward AS (1938) A fossil skull of an ancestral Bushman from the Anglo-Egyptian Sudan. Antiquity *12:*193–195.

Yassin A, and Taha M (1965) Hearing disorders in secondary anaemia. J. Laryngol. Otol. *79:*917–920.

Zonneveld FW, and Wind J (1985) High-resolution computed tomography of fossil hominid skulls: A new method and some results. In PV Tobias (ed.): Hominid Evolution, Past, Present and Future. New York: Alan Liss, pp. 427–436.