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Cholesteatoma in a Pre-Columbian American Aborigine from the Gyftakis Site, St. Ignace, Michigan

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Western Michigan University

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CHOLESTEATOMA IN A PRE-COLUMBIAN AMERICAN ABORIGINE
FROM THE GYFTAKIS SITE, ST. IGNACE, MICHIGAN

by

Robert William Hull

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
Degree of Master of Arts
Department of Anthropology

Western Michigan University
Kalamazoo, Michigan
April 1985
A 25 to 30 year old American aborigine male dating from A.D. 170 ± 80 years exhibits bilateral osteolytic lesions of the temporal bones. A differential diagnosis was set up to determine the cause of the pathology. It was determined that the individual had probably been subject to chronic suppurative otitis media complicated by mastoiditis, cholesteatoma, and probable lateral sinus thrombosis of the right temporal bone. Otitis media then developed within the left temporal bone causing some bone destruction, although not to the extent seen in the right temporal. It is likely that a small cholesteatoma had developed or would have developed in the left temporal bone, had the individual survived longer. The ultimate cause of death is believed to be due to meningitis following dural exposure. This is a significant specimen because no earlier case of cholesteatoma in America has been reported.
ACKNOWLEDGEMENTS

I extend my greatest thanks and appreciation to Dr. Robert I. Sundick for his generosity with time, materials, knowledge, and experience. Of all the educators I've known, he has been the most willing to share what he has with those who need it. I would also like to thank all the faculty of the Department of Anthropology at Western Michigan University for sharing their knowledge and enthusiasm with me. I would like to thank Dr. Robert Jack Smith for his patience, understanding, and wisdom, without which I would not have succeeded in my graduate work. Also, to Dr. William Cremin, my sincere gratitude for not only helping me edit my thesis, but for also teaching me how to write with all his two page critiques. Special thanks are extended to Dr. John Gregg of the University of South Dakota for taking the time and effort to meticulously scrutinize every printed word of this project, and for sharing with me his invaluable knowledge and experience. Without Dr. Gregg's assistance, I would always wonder if I had gotten it right. Thanks go to Dr. William Bass of the University of Tennessee for allowing me to x-ray the specimen on his campus. Finally, I would like to thank my parents, Heather and Thomas Hull, and Ms. Stephanie Oie for their love and support throughout this project and in times gone by.

Robert William Hull
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Western Michigan University

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# TABLE OF CONTENTS

| ACKNOWLEDGEMENTS                         | i1          |
| LIST OF TABLES                           | iv          |
| LIST OF FIGURES                          | iv          |

## CHAPTER

I. INTRODUCTION .................................. 1

II. DIFFERENTIAL DIAGNOSIS ...................... 10

III. EMBRYOLOGY ................................ 14

IV. ANATOMY ..................................... 17

V. OTITIS MEDIA ................................ 23

VI. MASTOIDITIS ................................ 27

VII. CHOLESTEATOMA ............................... 29

VIII. DISCUSSION ................................ 34

BIBLIOGRAPHY .................................... 41
LIST OF TABLES

1. Osteolytic Pathology ........................................ 13

LIST OF FIGURES

1. Right Temporal Bone—Medial Aspect ...................... 4
2. Right Temporal bone—Lateral Aspect .................... 5
3. Left Temporal Bone—Medial Aspect ....................... 7
4. Left Temporal Bone—Lateral Aspect ...................... 8
CHAPTER I

INTRODUCTION

Paleopathology has become the multi-disciplinary study of disease in antiquity, particularly those diseases which afflicted our human ancestors. Throughout the vast majority of time in which humans evolved, there are no written records that allow paleopathologists can ascertain the extent of ancient disease. Although artwork, figurines, and coprolites can provide some clues to ancient disease, they are extremely restricted in time and geographical location. Any diagnosis of pathology in ancient populations is further complicated by the fact that, with rare exceptions, only those diseases which leave their mark on the skeletal remains are available for interpretation. The preservation of soft tissue is rare; some examples being intentional preservation, as found in Egyptian mummies and preservation through regional environmental characteristics such as extreme aridity, extreme cold, water of high salt content, and bogs (Wells, 1967; Hare, 1967). A further complication, according to Wells, is that many postmortem changes in exhumed skeletal remains mimic pathological conditions found in living populations. The weight of the overlying earth, for example, may warp bone to such an extent that a pathological process such as hydrocephaly, or a cultural practice such as artificial cranial deformation is mimicked. This is particularly true in wet and acidic soil where the decalcification of
bone often occurs. The chemical action of the soil may also cause pitting and erosion of the bony surface resembling many pathological conditions such as osteoporosis, leprosy, mastoiditis, and others. In addition to the mechanical and chemical action of the soil, living agents such as bacteria, fungi, gnawing animals, plant roots, and careless excavators contribute to the difficulty in making accurate pathological diagnoses from human skeletal remains (Wells 1967).

This thesis deals with the paleopathological diagnosis of an American Indian cranium from the Gyftakis burial site near the town of St. Ignace in the Upper Peninsula of Michigan. The Gyftakis burial site (SIS-6) was excavated in 1973 by Timothy Smith and James Fitting. It is situated on what was once the "Algoma beach terrace that existed between 1500 and 2000 years ago", and is contiguous to the later Marquette Mission site (Smith and Fitting n.d., p.3). The seven burials at the Gyftakis site were found in association with a "distinctive Lake Forest Middle Woodland ceramic assemblage" (Smith and Fitting n.d., p.1). This is corroborated by a radiocarbon date of A.D. 170 ± 80 years obtained for feature #22, which in turn is associated with feature #15, the burial pit.

The specimen reported herein is a male of approximately 25-30 years who exhibits bilateral osteolytic lesions of the temporal bone. The other burials consist of two middle to old age males, one middle to old age female, and three subadults of unknown sex (Sundick n.d.).

The initial examination of the skeletal remains was conducted by Dr. Robert Sundick of Western Michigan University in 1977. At that time it was noted that the only skeletal pathology present in the
post-cranial remains was a significant amount of degenerative joint disease among the older adults. Other pathology noted in the crania were the aforementioned temporal lesions of the young adult male, and what appeared to be a well-healed depressed fracture on the frontal bone of one of the older males.

A preliminary diagnosis of cholesteatoma was made for the temporal bone lesions in the young adult male by Sundick in his unpublished report submitted to Dr. James Fitting. To date, the only published report of the Gyftakis site can be found in Hopewell Archaeology (Brose ed. 1979). There is no mention of the burials within Fitting’s published site description.

Because of the relative rarity of cholesteatomas in prehistoric populations, it was decided to undertake a more indepth study of this particular specimen. The initial diagnosis of cholesteatoma was confirmed by two otolaryngologists, one of whom felt confident in diagnosing a cholesteatoma for the dry bone specimen, the other was not convinced the lesions were necessarily the result of a pathological condition.

There are several possible explanations for the temporal bone lesions of the Gyftakis specimen therefore, descriptions and photographs of the temporal bones may help clarify the problem. Figures 1-4 are photographs of the Gyftakis temporal bones. Figure 1 shows the medial surface of the right temporal bone, the occipital margin to the right. Directly to the left of the occipital margin is the groove for the lateral sinus, the superior half showing one 8mm fistula (possibly an enlarged mastoid foramen, although it seems too
Figure 1. Right Temporal Bone—Medial Aspect. A) 8mm fistula and 3mm fistula in the lateral sinus groove with 12mm area reactive new bone growth. B) 3x14mm fistula which communicates with cavity in mastoid antrum.
Figure 2. Right Temporal Bone—Lateral Aspect. A) 12mm fistula in mastoid antrum. B) Extensive pitting around external auditory meatus.
high) and above it a 3mm fistula eroded into the bone. Between the two fistulas and extending slightly to the right is a 12mm wide area of reactive new bone growth (see figure 1A). To the left of the groove for the lateral sinus is an air cell system within the bone where the petrous joins to the medial surface of temporal bone. Directly above this area is a 3x4mm fistula that communicates with an interior cavity in the region of the mastoid antrum (see figure 1B).

This cavity can also be seen in figure 2, which is a photograph of the outer surface of the right temporal bone. In the center of the photograph is a 12mm fistula into the mastoid antrum, surrounded by an area of extensively pitted bone (see 2A). At the superior margin of the 12mm fistula is an area of reactive new bone growth which indicates that the site of the lesion was being actively remodeled at the time of death. Directly below this lesion lies the external auditory meatus, the opening of which is also extensively pitted (figure 2B). There is also a 4mm fistula eroded through the posterior-superior portion of the meatus (not visible in photograph), and part of the inferior portion as well. The right petrous pyramid (not shown) was not attached to the temporal bone due to the absence of bone between the two parts. This may have been due to postmortem causes, but it is clear that there was active bone resorption in this area as seen in Figure 1. Moreover, there is 10mm long, 3mm deep abscess along the superior surface of the petrous pyramid in the region of the vestibular labyrinth, again indicating some bone resorptive process.

Figure 3 shows the medial surface of the left temporal bone. In the region of the groove for the lateral sinus there does not appear
Figure 4. Left Temporal Bone—Lateral Aspect.  A) 5mm fistula and 10mm fistula in posterior-superior portion and inferior portion of external auditory meatus.
to be a mastoid foramen. However, the bone in the region shows the same pitting, and to a lesser degree some bone remodeling characteristic of the right medial surface. Similar to the right side, there is 5mm long fistula eroded into the wall of the temporal bone, except that it lies directly to the right of the auditory meatus (see Figure 3A). Again, the left petrous was not attached to the temporal wall due to missing bone.

Figure 4 is a photograph of the outer surface of the left temporal bone, which in many respects resembles the right temporal bone. Although there appears to be no bone erosion in the region of the mastoid antrum, the pitted appearance of the bone in the area of the external auditory meatus is identical. Similarly, there is a 5mm fistula in the posterior-superior portion of the meatus and a 10mm fistula on the inferior portion (Figure 4A).
There are several explanations for the kind of bone destruction present in the Gyftakis cranium. Postmortem erosion due to the action of the soil can be eliminated on the basis of the reactive bone growth which was still active at the time of death. However, there are several disease processes which may have been responsible for this type of bone destruction. While chronic suppurative mastoiditis accompanied by cholesteatoma seems to be a likely candidate, a thorough investigation of all the possibilities seems appropriate in attempting to diagnose any pathology for a specimen of this age and condition. A differential diagnosis was deemed necessary, in order to determine if, in fact, the presence of the bilateral temporal lesions could be more readily explained by a condition other than cholesteatoma, which is somewhat rare. Table 1 summarizes the manifestation of a number of different osteolytic diseases and serves to eliminate many of the more common osteolytic disease processes described in the paleopathological literature.

In constructing a table for differential diagnosis, six criteria were used to describe pathology which has been known to cause osteolytic defects. These pathological manifestations were recorded from the examination of other archaeological remains, as is done in the paleopathological literature, and further examined in the modern
clinical medical literature (primarily otolaryngology). Both sources are included in the "Ref. #" column. An "X" in the appropriate category represents that such conditions of the criteria are frequently associated with the pathology named. The absence of an "X" in any category indicates the opposite.

The first category, labelled DESTR 0->IN (meaning there was destruction of bone through both the outer and inner tables), is used to eliminate those pathological conditions which are restricted to the periosteum and do not involve the marrow or diploe of bone.

The second category, labelled N.BON LTD. (meaning a limited degree of reactive bone growth), describes those conditions which are consistent with some limited new bone growth. This is used to eliminate either extensive bone growth as seen in hypertrophic conditions, or no new bone reaction such as is characteristic of leprosy, multiple myeloma, tuberculosis of the skull, histiocytosis X, and other purely lytic conditions.

The third category, labelled TEMP. BONE, designates whether the condition is ever found on the temporal bones. There are many pathological conditions which affect all bones of the cranial vault. These conditions are included in this category, while conditions usually located on the cranium, but rarely or never on the temporal bones, are not. Thus, this category alone eliminates, leprosy, syphilis, aspergillosis, and sarcoidosis.

The fourth category, labelled BILAT SYMM. (meaning bilaterally symmetrical), is one of the most limiting, as it describes the bilaterally symmetrical defects present on the Gyftakis cranium.
Bilateral symmetry is rare in any condition; most cases having either total skeletal involvement, such as vitamin or mineral deficiencies, or are congenital in origin.

The fifth category, labelled AGE 25-30, is much less restrictive, but includes those conditions that are not uncommon in a male of 25-30 years. This will usually eliminate many forms of cancer because of age or sex. Metastatic cancer, for example, is more often osteolytic in females and osteoblastic in males.

The sixth category, labelled PRE-C MICH. (meaning pre-Columbian Michigan), includes all those conditions which could have been present in a pre-Columbian aboriginal population of Michigan approximately 2,000 years ago. Because of the small populations, lack of many domestic animals or agriculture, and the absence of Old World pathogens, many conditions can be eliminated on this basis alone. Examples would be tropical diseases such as yaws, or fungal infections such as brucellosis which have arisen in populations practicing agriculture.
<table>
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<th>Ref. #</th>
<th>IDESTRIN.BON</th>
<th>TEMP.</th>
<th>IBILAT</th>
<th>IAGE</th>
<th>PRE-C</th>
<th>I0-&gt;IN</th>
<th>LTD.</th>
<th>IBONE</th>
<th>ISYMM.</th>
<th>125-30</th>
<th>MICH.</th>
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<td>I</td>
<td>I</td>
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<tr>
<td>Glanders</td>
<td>(1)</td>
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<td>I</td>
<td>I</td>
<td>X</td>
<td>X</td>
<td>I</td>
<td>I</td>
<td>I</td>
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<td>X</td>
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<td>I</td>
<td>I</td>
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<tr>
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<td>I</td>
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<td>X</td>
<td>X</td>
<td>I</td>
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<td>I</td>
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<td>I</td>
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CHAPTER III

EMBRYOLOGY

The embryology and development of the ear and its surrounding bone is made up of seemingly unrelated processes taking place over long periods of time, some processes not being completed until puberty. The earliest external feature of the ear to appear in the human embryo is the otic placode, which appears at 3 weeks post conception as a thickened area of ectoderm on either side of the head. As the embryo develops, the otic placode invaginates to form a pit and later migrates inward and closes to form a cyst. At this stage it is known as the otocyst (or otic capsule) and will eventually become the housing of the inner ear (Mawson 1967).

Also, early in human embryonic development, structures analogous to the gill slits of fish appear on the cephalic end of the developing embryo. These structures are known as the branchial clefts, which arise from the ectoderm and are structurally reinforced by mesodermal tissues known as the branchial arches. The first branchial arch ultimately forms the mandibular process and two of the three ossicles of the middle ear, the malleus and the incus. The depression formed by the first branchial arch develops into the cavities of the nasopharynx, middle ear, and the eustachian tube. The mucosa lining the eustachian tube, middle ear, and the mastoid is derived from an invagination of the primitive pharynx, the mucosa.
growing upward into the embryonic skull from the pharynx (Gregg, personal communication). The first branchial cleft later develops into the external auditory meatus which slowly migrates inward to meet the tympanic membrane at the lateral boundary of the middle ear. The second branchial arch forms the third ossicle, the stapes, the stapedial muscle and the facial nerve (Kobrak 1959).

The otic capsule which surrounds the structures of the inner ear, arises independently and quite differently from the other bones of the skull. Starting from at least 14 ossification centers in the cartilaginous shell, the ossification process quickly spreads to its full dimensions by the fifth fetal month and fuses without sutures. The periosteal bone of the petrous later develops in layers around the capsule, eventually becoming very cancellous in nature, and developing air cells around the otic capsule (Anson and Donaldson 1967).

By the first half of fetal life, the middle ear, ossicles, and inner ear have been surrounded by the membranes of the developing temporal bone, and ossification centers appear in the petrous, squamous, and tympanic parts of the bone. The four parts of the temporal bone develop from scattered ossification centers within the membranous and cartilaginous precursors to bone within the developing fetus. The petrous and mastoid both develop out of the cartilaginous arches continuous with the otic capsule. The squamous develops out of a membrane which lies laterally to the middle ear cavity housing the ossicles. The tympanic part of temporal bone also develops from a membrane near the now detached ossicles derived from the first and
second branchial arches (Hamilton 1976).

In a neonate, only three of the four parts of the temporal bone can be readily distinguished: the squamous, the petrous, and part of the tympanic. The tympanic part at birth consists only of an incomplete ring of bone which slowly becomes fused with the squamous and grows medially to form the anterior-inferior portion of the bony wall of the external auditory meatus. The mastoid process does not begin to develop significantly until the second postnatal year, although in some cases pneumatization does begin at birth (Gregg, personal communication). As it develops, the bone between the cavity of the tympanic antrum and the exterior surface of the mastoid thickens, while inside bone is being resorbed to form the mastoid air cells. This process, barring any serious ear infections in early childhood, is usually completed by puberty (Hamilton 1976).
CHAPTER IV
ANATOMY

The ear can be divided into three discrete units: the external ear, the middle ear, and the inner ear. The external ear is functionally designed to transmit sound waves from the outside environment to the tympanic membrane or eardrum. It consists of the auricle on the exterior surface of the head and the external auditory meatus which conveys sound through a short (25 mm) cartilage and bone tube to the tympanic membrane.

The middle ear begins laterally at the tympanic membrane and includes the tympanic cavity, the tympanic antrum, and the eustachian tube. The tympanic cavity is located within the temporal bone and is bounded laterally by the tympanic membrane and medially by the bony labyrinth of the inner ear (specifically the vestibule). The tympanic cavity is an irregularly shaped air space which houses three small connected bones (ossicles), which intensify and transmit the vibration of the tympanic membrane across the air space of the middle ear to the vestibule of the inner ear. The eustachian tube is a tube about 37 mm long in adults and extends from the tympanic cavity downward to the pharynx, creating an air passage between the otherwise sealed middle ear and the pharynx. In this way the eustachian tube functions to keep the mucous membranes lining the middle ear and associated structures relatively dry and thus free
from infection. It also serves as a means of equalizing pressure differences between the air within the middle ear and the outside environment. This air system or pneumatization, is augmented by the tympanic antrum (also called mastoid antrum) and a system of intercommunicating air cells within the squamous, petrous, and mastoid parts of the temporal bone.

The tympanic antrum is an air chamber about 10 cubic millimeters in volume, which lies laterally, slightly superior and posterior to the tympanic cavity, communicating with it via a small opening (known as the aditus) in the posterior-superior portion (epitympanic recess) of the tympanic cavity. The tympanic antrum communicates with the mastoid air cells, which are abundant air pockets lined by mucous membranes, continuous with the tympanic cavity and lying within the mastoid part of the temporal bone (Anson and Donaldson 1967). A thin plate of bone (tegmens tympani) separates the tympanic cavity and antrum of the middle ear from the cranial cavity by forming the roof of the two connected chambers. Another thin plate of bone forms the floor of the tympanic cavity and separates it from the bulb of the jugular vein. The anterior wall of the tympanic cavity is formed by another thin plate of bone which also serves as the bony wall of the carotid canal. The posterior wall extends from the inferior portion of the tympanic cavity to the superior portion of the tympanic antrum. In the region directly inferior to the aditus on the posterior wall is a projection of bone which houses the stapedius muscle and is known as the pyramid. The posterior wall of the antrum also contains air cells within the bone.
between the antrum and lateral wall of the lateral sinus. The lateral
wall of the antrum is opposite the outer surface of the squamous part
of the temporal in the region of the suprameatal triangle (the area
surrounding and just above the external auditory meatus to the
supramastoid crest). Medially, the cavity of the antrum ends as the
bony canal of the posterior semicircular canal (Hamilton 1976).

The inner ear is housed within the petrous part of the temporal
bone and contains the specialized organs of hearing and balance. It
is comprised of the membranous labyrinth, the osseous labyrinth, and
the otic capsule. The membranous labyrinth is a sealed system of
fluid-filled channels and pockets inside, but somewhat smaller than,
the osseous labyrinth which forms around it. The membranous labyrinth
is surrounded by perilymphatic fluid within the hard dense bone of
the osseous labyrinth, both of which are housed within the bony otic
capsule (Anson and Donaldson 1967). The labyrinth consists of two
sections: a non-auditory section in the superior portion of the otic
capsule, and the auditory labyrinth in the inferior part of the otic
capsule. The superior portion of the otic capsule contains the organ
of balance, consisting for the most part of three fluid-filled tubes
at right angles to one another, known as the semicircular canals. The
inferior portion of the otic capsule contains the spiral, cone-shaped
organ of hearing known as the cochlea. The central portion of the
osseous labyrinth forms the medial wall of the tympanic cavity—the
vestibule. Along the lateral wall of the vestibule is an opening
known as the fenestra vestibuli (oval window) into which the
footplate of the stapes rests (Mawson 1967). The stapes conveys the
vibrations of the tympanic membrane and the ossicular chain into the fluid filled cochlea where they are ultimately translated into sensory information. Comprising the largest feature of the medial wall of the tympanic cavity is a projection derived from the first turn of the osseous labyrinth surrounding the cochlea known as the promontory (Hamilton 1967). Posterior and inferior to the promontory is another membrane covered opening in the vestibule known as the fenestra cochleae (round window), which may provide protection against unusually loud noise by expanding in response to excessive vibration of the fluid within the cochlea (Schuknecht 1974).

The temporal bone which houses the ear can be divided into four parts: the squamous part, the mastoid part, the petrous part, and the tympanic part. The squamous part comprises the largest portion of the temporal and extends superiorly from the external auditory meatus to the inferior margin of the parietal and articulates along its anterior margin with the sphenoid. The outer table is smooth and functions as the site for the attachment of the temporal muscle. Near the base of the outer table, the zygomatic process projects laterally and anteriorly towards the face. Directly inferior to the base of the zygomatic process, lies the mandibular fossa. The medial aspect of the squamous part presents the sulcus for the middle meningeal artery, the surface of which is pitted with channels leading into the diploe. (Anson and Donaldson 1967). The inner table of the squamous also forms part of the floor and lateral wall of the middle cranial fossa, and together with the petrous it forms the tegmen tympani—the roof of the middle ear cavity. Deep within the squamous near the
origin of the zygomatic process, air cells extend above and anterior to the external auditory meatus (Hamilton 1967).

The petrous bone extends medially from its junction with the temporal bone and articulates at its inferior margin with the mastoid part. Roughly pyramidal in shape, its base fuses with the medial wall of the squamous at the petrosquamosal fissure and extends medially to house the structures of the inner ear (Anson and Donaldson 1967). Like the mastoid and squamous, the petrous also contains air cells which are situated below the posterior semicircular canal and communicate with the air cells of the tympanic antrum, mastoid, and squamous. While laterally forming part of the walls, floor, and roof of the middle ear, the medial end of the petrous forms the bony part of the eustachian tube, and inferiorly it is perforated by the carotid canal. Lateral to the carotid canal, the styloid process extends inferiorly from its base near the junction of the petrous and tympanic part (Hamilton 1976).

The mastoid part of the temporal bone occupies the area immediately behind the external auditory meatus. It articulates with the occipital bone along its posterior border, and with the parietal bone along its superior border. It makes up part of the lateral wall of the posterior cranial fossa. In adults, the inferior border of the outer table of the mastoid is dominated by an inferiorly projecting cone of bone known as the mastoid process. This projection serves as an attachment for the muscles of the shoulder girdle and neck (Hamilton 1976). The inner table of the mastoid presents a deep groove along its occipital margin. This is the groove for the sigmoid
venous sinus of the dura matter of the posterior cranial fossa (Anson and Donaldson 1967).

The tympanic part of the temporal bone is that part which surrounds the external auditory meatus. Roughly "C" shaped, it makes up the anterior and inferior and part of the posterior walls of the external auditory meatus. Medially it presents a feature called the tympanic sulcus; this is the groove in which the tympanic membrane rests. It is a thin plate of bone and shows much variability in its degree of ossification in adults (Anson and Donaldson 1976).
CHAPTER V

OTITIS MEDIA

Otitis media most often arises as an extension of an upper respiratory infection via the eustachian tube, but may also spread from pathogens in the blood, or from trauma to the tympanic membrane. Generally, otitis media can be divided into four types: (1) acute suppurative otitis media, (2) chronic suppurative otitis media, (3) secretory otitis media, and (4) chronic adhesive otitis media (Glorig and Gerwin 1972).

Acute suppurative otitis media was formerly a common disease of early childhood, most cases being diagnosed at the age of 1.5-4.5 years and resolved by about 7-9 years (Gregg, personal communication). The upper respiratory system—the throat and nasal passages—communicate with the middle ear, mastoids, lacrimal passages, and the sinuses. Infections can spread from inhaled organisms in the nasal passages to any tissues of the upper respiratory system and the lungs. Infections of the middle ear usually begin in the nasal passages and spread upward through the eustachian tube where the infectious process damages the ciliary action within the tube causing obstruction. When the tubes are obstructed there is often pressure within the ear and fluid buildup (Nester, Roberts, Pearsall, and McCarthy 1978). In infants, acute suppurative otitis media is sometimes unassociated with upper
respiratory infections, but is disseminated from the oral cavity. The short and straight eustachian tube in the infant, teething, and vomiting are thought to be the influential agents in the disease process. Perforation of the tympanic membrane, a complication of otitis media, is also a common source of middle ear infection in all ages, the pathogens migrating inward from the external auditory meatus. Severe acute suppurative otitis media may lead to an abscess within the mastoid cells and cause osteomyelitis (Mawson 1967; Gregg, personal communication). Today, the pathogen responsible for the majority of the cases of all types of otitis media is *S. pneumoniae*. Other pathogens include *H. influenzae*, *Streptococcus pyogenes*, and *Staph. Aureus*. (Nester et al. 1978). Respiratory viruses (rhinoviruses, adenoviruses, and enteroviruses) are usually precursors to a secondary bacterial invasion (Gregg, personal communication). In the pre-antibiotic era, scarlet fever, measles, whooping cough, and diphtheria contributed to the greatest incidence of acute suppurative otitis media (Mawson 1967).

Chronic suppurative otitis media can develop from a severe necrotizing acute otitis media, or from ineffective middle ear aeration. While the process takes much longer (often into adulthood) than acute suppurative otitis media, the damage to the middle ear and hearing can be irreversible. If a previous bout of acute suppurative otitis media is not resolved the infection may spread throughout the mucous membranes of the middle ear cleft, producing the granulation tissue characteristic of severe mucoperiosteal inflammation. Small underdeveloped mastoids and poor pneumatization within the spaces of
the temporal bone tend to increase the frequency and duration of chronic suppurative otitis media. This type of infection was also commonly seen in epidemics of scarlet fever, diphtheria, and measles in the pre-antibiotic era (Glorig and Gerwin 1972).

Acute secretory otitis media is most often the result of a eustachian tubal obstruction that may be due to viral infection. Rather than the pus characteristic of suppurative otitis media, the effusion is serous in nature. Edema of the mucous membranes may cause the lumen of the eustachian tube to be blocked within the tympanic cavity, the effusion following thereafter. The fluid within the ear cannot be drained and becomes viscous, leading to hearing loss in the ear affected. There is no organic damage in this instance and the disease may resolve itself without medical intervention. Other causes for secretory otitis media include allergies and endocrine disorders such as suppressed ovulation, obesity, hypoactive metabolic states, and endocrine changes in pregnancy (Glorig and Gershwin 1972).

Chronic adhesive otitis media is a process whereby the inflammation of the mucous membranes of the middle ear leads to scarring of the tissue. This is followed by the laying down of dense fibrous tissue by fibroblasts over the inflamed tissue. Adhesions may form between contiguous structures within the middle ear cleft resulting in ossicular fixation that culminates in a conductive hearing loss in the ear affected. The original inflammation may have been the result of suppurative otitis media, physical trauma, or from an unresolved secretory otitis media. In either case, complications of chronic adhesive otitis media include fibrosis of the tympanic
membrane, tympanosclerosis, ankylosis of the ossicles, and new bone formation in areas not previously ossified (Mawson 1967). The onset of secretory otitis media is usually in childhood, but may occur in adults as well. There also appears to be a hereditary factor involved in the contraction of the disease, as is also the case with the other types of otitis media, although the mechanism of inheritance is unknown (Glorig and Gerswin 1972). It has been shown, however, that hereditary factors play a large role in the size and distribution of air cells within the temporal bone and that this pneumatization process is directly related to an individual's susceptibility to otitis media (Daimant and Daimant 1977).
CHAPTER VI

MASTOIDITIS

One may define a complication of otitis media as being the spread of inflammation of the mucous membrane beyond the middle ear cleft. Typically, this involves the infection of bone. If it occurs within the bony air cell walls of the mastoid, it is known as mastoiditis. There are two phases of mastoiditis: chronic and acute, the difference being due to the amount and size of air cells within the mastoid. This pneumatization process varies widely from individual to individual. Five categories can be drawn from the variation in pneumatization: (1) pneumatic (normal, well developed air cell system), (2) diploic (no air cell system), (3) sclerotic (dense, hard bone replacing air cell system), (4) mixed (combinations of the preceding types), or (5) atypical (unusually large or patterned air cell systems) (Gregg and Steele 1978). Generally, those individuals with little or no pneumatization are more likely to be subject to chronic mastoiditis, while those with normal or extensive pneumatization are more likely to develop the acute form of mastoiditis (Mawson 1967). As stated previously, the mastoid is not fully developed at birth. Even in infancy, the bone within the mastoid process does not contain air cells but is diploic in nature. In approximately 20% of the population, the mastoid process remains diploic throughout life, never developing
air cells (Turner 1968). In these individuals, chronic mastoiditis may cause the bone of the acellular mastoid to erode by the osteolytic action of granulation tissue (Mawson 1967).

In the acute form of mastoiditis there is a "coalescence of air cells due to hyperaemic osteoporosis and pressure necrosis of the bony cell walls, with the formation of an empyema" (Mawson 1967: 331). The inflammatory process is first evidenced by an enlargement of the blood vessels within the haversian canals. The lining of the canals fills with blood serum and pus, followed by leukocytes appearing in increasing numbers. Finally osteoclasts invade the haversian canals and begin to erode the walls of the air cells. If the process continues untreated it may spread throughout the temporal bone's communicating air cell system. The process may then erode through the roof of the middle ear cleft from the antrum into the middle cranial fossa, or posteriorly through the canal for the sigmoid sinus. In chronic mastoiditis with suppuration, the erosion of bone may be augmented by the presence of a cholesteatoma, which may likewise spread throughout the temporal bone causing extensive bone resorption and intercranial complications. In either case, a mastoid infection may spread to the dura matter of the brain causing abscesses, meningitis, or otitic hydrocephalus (Turner 1968).
Cholesteatoma is a frequently seen complication of otitis media.

By definition, cholesteatoma is:

stratified squamous epithelium trapped and growing in foreign sites within the temporal bone, resulting in the production of a progressively expanding tumor mass consisting of new growth of epithelium, various stages of degenerating epithelium, abundant keratin, and is usually associated with cholesterol and chronic inflammatory cells (Cody 1977: 6).

Keratinizing stratified squamous epithelium is normally found as a thin layer of epidermis comprising human skin. As it grows, it constantly sheds thin layers of keratin. If it is confined to the spaces within the middle ear (or even the frontal sinus) it is known as cholesteatoma (Paparella, Shumrick, Meyerhoff, and Seid 1980).

The pathogenesis of cholesteatoma has been a subject of continuing controversy since it was first described by Cruveilhier in 1829 as "tumeurs perlees" and termed the pearl-like growths "cholesteatomie" (Hopp and Montgomery 1984). The term cholesteatoma, in fact, is a misnomer, as the growths are not composed primarily of cholesterol as the name suggests, but of keratin. The term keratoma is used by many otologists, but the misnomer still persists. Similarly, many theories on the pathogenesis of cholesteatoma have been proposed, rejected, and revived. Today, the controversy continues, but three theories have the widest acceptance among
researchers of the problem: (1) the migration theory, (2) the metaplasia theory, and (3) the congenital theory.

The migration theory focuses on the migration of stratified squamous epithelium from the skin lining the external auditory canal into the middle ear, usually via a posterior-superior perforation of the tympanic membrane (Paparella et al. 1980; Gregg, personal communication). When this occurs it gives rise to what is termed a secondary acquired cholesteatoma (Cody 1977). Another form of the migration theory, suggested by Bezold in 1890, states that a dysfunctional eustachian tube may cause a vacuum within the middle ear space to retract a part of the tympanic membrane (Schrapnell’s membrane) into the epitympanic recess, forming what is known as an attic retraction pocket. To date, this theory has been the most widely accepted, and cholesteatomas of this type are commonly known as a primary acquired cholesteatoma (Derlacki and Clemis 1967).

The metaplasia theory, first proposed by Wendt in 1873, and revived by Bendek in 1963, suggests that through inflammation the mucosa of the middle ear undergoes a change from the normal "flattened endothelium-like cells", which are essentially of "respiratory epithelial character", to the stratified keratinizing squamous epithelium characteristic of cholesteatoma (Friedmann 1977 pp.12). Experimental studies have shown that a vitamin A deficiency can cause squamous metaplasia to take place in animals, but it has not been proven to occur within the human middle ear (Friedmann 1977). In fact, most otologists today do not believe the metaplasia theory to be a likely one for the pathogenesis of middle ear
The congenital theory of cholesteatoma pathogenesis, unlike the preceding theories, does not depend upon an infectious or inflammatory agent to precede the formation of the cholesteatoma. Most otologists believe there are two types of cholesteatoma, a congenital type and the acquired type mentioned previously. The congenital form is thought to arise from epithelial cell rests which become trapped within the developing temporal bone before birth. This type of cholesteatoma is most frequently found among males (80% male compared to 20% female). This type of cholesteatoma is also very rare. Of a series of 483 operations for aural cholesteatoma performed at the Mayo Clinic between 1963 and 1969, only 2% of the cholesteatoma were of the congenital type (Cody 1977). Moreover, in a review of medical literature conducted by Curtis (1979), only two cases had been previously reported. An additional case reported by Curtis (1979) is particularly interesting in that it involved a 20-year-old male with bilateral aural cholesteatoma, the mother of whom had undergone a bilateral mastoidectomy. As Curtis points out, "this raises the possibility of bilateral hereditary congenital cholesteatoma" (Curtis 1979).

Typically, congenital cholesteatoma remains symptomless for years. It is usually first discovered when a patient experiences facial paralysis due to pressure upon the facial nerve in the middle ear, or upon routine ear examination when the cholesteatomous mass is detected as a pearl-like object behind an intact tympanic membrane.

The precise mechanism with which cholesteatoma promotes bone
resorption is also a subject of continuing controversy. At one time it was assumed that cholesteatoma stimulated bone resorption through the pressure of its expanding mass upon the bone which surrounds it. In the last decade, however, several researchers have proposed new mechanisms of bone resorption. It has been demonstrated that:

sterile granulation tissue, which is histologically similar to the subepithelial chronic inflammatory tissue found in cholesteatoma, is capable of chemically activating bone resorption in vitro (Gantz, Clancey, and Abramson 1977: 168).

Furthermore, it has been reported that prostaglandin "is one of the chemical mediators responsible for producing demineralization of bone" (Gantz, Clancey, and Abramson 1977: 168).

It has also been observed that mononucleated osteoclasts, typically found in great abundance in areas of bone resorption, are rarely seen in the area of cholesteatoma (Gantz et al. and Abramson et al. in Bretlau, Jorgensen, Sorenson, and Dabelsteen 1982). Other mechanisms may be involved in the bone resorption process, such as the "lymphocytes and microphages associated with chronic inflammatory lesions" which may be stimulated into releasing an osteoclastic stimulating factor, or producing collagenase and degrading collagen and bone (Horton et al. in Bernstein, Hausmann, and Wright 1977: 158).

It is becoming increasingly clear that the keratinized squamous epithelium of the cholesteatoma mass is not the primary factor in bone resorption in the middle ear. Rather, it is the chronic inflammatory process or granulation tissue between the bone and the cholesteatoma sac itself which is responsible for bone resorption.
(Abramson and Huang 1977). Precisely which factors are involved in the breakdown of the bone matrix and demineralization is still unknown. It is known, however, that bone resorption can take place in the middle ear with or without cholesteatoma; albeit the rate of bone resorption increases dramatically in the presence of cholesteatoma, where it greatly contributes to the inflammatory process by continually shedding keratin and dead cells into confined space, becoming an ideal culture for bacterial growth and infection (Abramson and Huang 1977).
CHAPTER VIII

DISCUSSION

The most likely explanation for the type of bone destruction present in the Gyftakis cranium is a middle ear infection, further complicated by mastoiditis or cholesteatoma. As dry bone specimens are not reported in the modern medical literature, it would be useful for diagnostic purposes to review the paleopathological literature dealing with this type of disease.

Gregg, Steele, and Bass (1982) present a case of probable osteomyelitis of the temporal bone which seems similar to the type of bony destruction seen in the Gyftakis cranium. The specimen is the skull of an American Indian male of approximately 40-45 years. Their description and interpretation of the pathology is as follows:

An extensive localized osteolytic defect involves the left mastoid. Within it there is destruction of bone, minimal new bone formation, and exposure of the intracranial space. There are no other skeletal abnormalities. Our impression: acute suppurative mastoiditis with osteomyelitis of temporal bone and dural exposure. Less likely considerations: a) acute trauma with secondary osteomyelitis, b) neoplastic invasion of bone, c) acute soft tissue infection with spread into temporal bone, and d) granulomatous infection involving the mastoid (Gregg, Steele, and Bass 1982: 245).

The term osteomyelitis has both a general usage and a specific usage. The general usage applies to an inflammation of bone and can include periostitis, an inflammation of the periosteum, and osteitis, simply a general inflammation of bone. The more specific usage of
osteomyelitis is used to describe an inflammation of bone which includes the marrow. Today and in the recent past, 90% of the cases of this type of osteomyelitis are due to the infectious microorganism *Staphyloccus aureus*. The remaining 10% of the cases are due to streptococci, pneumococci, meningococci, and infrequently salmonella or colon bacilli (Steinbock 1976). According to Ronald Hare, such microorganisms are capable of surviving among small scattered populations such as one would expect to have encountered in the Upper Peninsula of Michigan 2,000 years ago. He further adds that *Streptococcus pyogenes* and *Diplococcus pneumoniae* are "generally responsible for acute infections of the mastoid following an extension of an infection in the throat to middle ear", and that "pre-Columbian skulls have been found in America showing evidence of this type of infection" (Hare 1967: 123).

An example of this type of evidence, presented by Tiche, Coulthard, Wachter, Thies and Harries (1981), is a report of 1,296 temporal bones of prehistoric Arizona Indians, 17.3% of which are considered to have been subject to some form of infection during their development. This is determined by the presence of 148 or 11.3% of the mastoids having a diploic mastoid air cell system, 56 or 4.3% of the mastoids being of a mixed type, and 20 or 1.7% being sclerotic. In instances where both temporal bones from an individual are present, 6% show evidence of infection unilaterally, and 13% show evidence of bilateral infection (Tiche et al. 1981).

In a similar study by Gregg, Steele, and Holzhueter (1965), 417 temporal bones from South Dakota Indian burials are examined. Of
these 417 temporal bones, 3.4% or 14 show mastoid air cell systems that are diploic, 37.6% or 157 are of the mixed type, and 6.2% or 26 are sclerotic. Although it was not possible to determine how many individuals have altered mastoid air cell systems bilaterally, both bilateral and unilateral alterations are present. There is no evidence of "cholesteatoma, cancer, antemortum surgery upon the bone, or other bony disease" present in the x-rays of any of the temporal bones (Gregg et al. 1965:59). They conclude, based upon the hypothesis that infections within the temporal bone alter the development of the mastoid air cell system, that "about 50% of the people represented by the skulls examined must have had a significant amount of middle ear disease during the period of mastoid development" (Gregg et al. 1965:60).

Mastoiditis is a relatively common example of bone destructive middle ear disease to be found in the paleopathological literature. Mastoiditis is characterized by a type of "abscess formation with destruction of the mastoid cells" (McKenzie and Brothwell 1967 p.464). In prehistory, before surgical intervention, it is unlikely that such an abscess would heal untreated, but if such were the case an absence of air cells in the mastoid region would be characteristic. A chronic infection arising from the middle ear and mastoid antrum, in the absence of mastoid air cells, is "associated with the formation of a cholesteatoma, an expanding cyst of epithelium" (McKenzie and Brothwell 1967: 465.)

An unusual case of mastoiditis is presented by McKenzie and Brothwell (1967). It is an early Dynastic Egyptian skull from Tarkhan which shows an osteolytic defect in the external auditory meatal wall.
surrounded by pitted bone which "might well be the sinus of a mastoid abscess", and directly above the lesion is a "trephine hole" in the parietal of the same side, indicating that possibly the trephination was an attempt at dealing with the disease (McKenzie and Brothwell 1967: 467).

Other examples of temporal bone lesions described by McKenzie and Brothwell include the well known Rhodesian and Boskop skulls of southern Africa. The Rhodesian skull is that of an Upper Pleistocene man and exhibits two lesions on the left temporal bone; a small circular hole through the squama directly above the external auditory meatus and a similar lesion occurring on the mastoid process directly posterior to the external auditory meatus. The Boskop skull is assigned to the late Stone Age and exhibits (like the Tarkhan skull) a hole in the roof of the external auditory meatus, which communicates with a large cavity that extends through to the inner table. According to Singer (1967), the pathology evident on the Boskop skull:

has undoubtedly been produced by a cholesteatoma which, as a complication of chronic middle ear infection, has the peculiar physiochemical property of eroding bone, and is frequently followed by intracranial complications (Singer in McKenzie and Brothwell 1967: 469).

McKenzie and Brothwell agree that "the evidence does suggest possibly a cholesteatoma", but they erroneously believe that cholesteatoma is found only in the presence of an "acellular mastoid", which the Boskop skull did not have (McKenzie and Brothwell 1967: 469). They describe nine other known cases of temporal lesions involving the ear region from various paleopathological sources, but are very reluctant
to venture any diagnosis or support the diagnoses of others.

From the preceding examples of middle ear disease and its complications in paleopathology, and from information gleaned from the medical literature presented in earlier chapters, it is now possible to propose a diagnosis for the pathology exhibited by the Gyftakis cranium. X-rays of the temporal bones clearly show that the right temporal bone has a mixed type of mastoid air cell system and a large cavity in the mastoid antrum. The left temporal bone shows normal pneumatization and no apparent osteolytic cavities. However, it is clear from gross examination that the infective process had spread to the left temporal bone and that the bone resorptive process had begun. This may have occurred in the absence of a cholesteatoma, or the cholesteatoma may not have been developed enough to have been seen in the x-rays. In any event, it is likely that if the individual had survived longer a cholesteatoma would have been evident in the x-rays of the left temporal bone as well. Although bone destruction in otitis media can occur with or without cholesteatoma, the extent of bone destruction present in the Gyftakis right temporal bone in the region of the mastoid antrum would tend to implicate cholesteatoma. The origin of the cholesteatoma may be infectious, as in the primary and secondary acquired forms, or it may be congenital. Bilateral cholesteatoma is rare today, most cases being of a congenital origin. However, as can be seen from Gregg et al. (1965) and Tiche et al. (1981), bilateral ear infections were prevalent among Native American populations and continue to be so to this day (Gregg, personal communication). In addition, the Gyftakis right
temporal bone shows the "mixed" type of mastoid air cell development, indicating chronic suppurative otitis media and mastoiditis (Gregg, personal communication). If the Gyftakis temporal bone lesions were due to congenital cholesteatoma, it is unlikely that the right temporal bone would show such poor pneumatization, as congenital cholesteatoma typically does not begin to manifest itself until early adulthood and later, after the pneumatization process has been completed. It is much more probable, given the rarity of congenital cholesteatoma, the mixed mastoid air cell system of the right temporal bone of the Gyftakis specimen, and the prevalence of bilateral mastoid infections among both modern and ancient Native American populations, that the cholesteatoma has an infectious origin. This type of cholesteatoma therefore, is the one proposed to be responsible for the osteolytic lesions of the right and left external auditory meatus, the right mastoid antrum, the right petrous pyramid, and possibly destroying the connecting bone in the region of the petrosquamosal junction bilaterally.

Other pathological considerations include a probable lateral thrombosis situated in the region of the groove for the lateral sinus on the medial surface of the right temporal bone, and probable meningitis; the ultimate cause of death in the individual (Gregg, personal communication).

One additional feature of the Gyftakis specimen, not mentioned previously, is the presence of two supernumerary central maxillary incisors. Associated with this is an unusually large incisive foramen in the hard palate, and a "possible inborn midline maxillary defect.
anteriorly" (Gregg, personal communication). Cases of supernumerary teeth have been linked with "multiple polyposis of the large intestine, osteomas of bone, multiple epidermoid or sebaceous cysts, and dermoid tumor" (Foley and del Rio 1970: 61). Whether the "epidermoid cysts" the authors refer to could be related to aural cholesteatoma or not is unclear. However, defects of the palate have long been associated with an increased incidence of otitis media due to associated problems with eustachian tube function, but whether or not the presence of the supernumerary teeth and "possible midline defect" can be related to the severity of the temporal bone destruction of the Gyftakis cranium is uncertain.

In conclusion, this thesis deals with the paleopathological diagnosis of a 25-30 year old American aboriginal male. The specimen is approximately 2,000 years old and was recovered from the Gyftakis site near St Ignace, Michigan. The specimen presents bilateral temporal bone lesions. The diagnosis for the osteolytic lesions is chronic suppurative otitis media complicated by mastoiditis and cholesteatoma of the right temporal bone, the infectious and osteolytic process spreading to the left temporal bone, possibly including a small cholesteatoma. Both external auditory canals were involved in the osteolytic processes, as well as the right petrous pyramid, and possibly the bone in the region of the petrosqamosal junction. It is also likely that the medial surface of the right temporal bone in the region of the groove for the lateral sinus was the site of a lateral sinus thrombosis and that meningitis was ultimately the cause of death in the individual.
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