

University of Nebraska Medical Center DigitalCommons@UNMC

MD Theses

Special Collections

5-1-1936

Suppuration of the petrous pyramid

Earl C. Slaughter University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search PubMed for current research.

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Part of the Medical Education Commons

Recommended Citation

Slaughter, Earl C., "Suppuration of the petrous pyramid" (1936). *MD Theses*. 467. https://digitalcommons.unmc.edu/mdtheses/467

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

SUPPURATION OF THE PETROUS PYRAMID

*8*7.

Senior Thesis

University of Nebraska College of Medicine

1936

Earl C. Slaughter

TABLE OF CONTENTS

2¹⁰

		rage
1.	Introduction	1
2.	Anatomy	4
3.	Pathology	14
4.	Symptomatology	20
5.	Diagnosis	39
6.	Conclusions	42
7.	Bibliography	47

480816

<u>5</u>7

For many years otologists have concerned themselves with the problems presented by otitic meningitis. In the course of studies it became apparent tha, in the interval of time which elapses in most cases between the otitic and the mastoid lesion and the therapy applicable to them, on the one hand, and the terminal lesion in the meninges, on the other, there ensues a period during which the patient is almost free from symptoms. This penultimate period would ordinarily pass unnoticed. Only after the conclusion of the case, in the post-mortem scrutin of symptoms, pathologic data and operative findings, did the lesser signs of that interval assume significance. Finally, once a relationship was established between the pennultimate phase of the infection and the terminal meningitis, and diagnostic aid was developed by means of x-rays of the petrosal pyramid during life and before the onset of the terminal meningitis, diagnosis of the lesion in the petrous pyramid became more exact and a distinct clinical entity was developed, called suppuration of the petrous pyramid, which is the topic of this paper.

The writer is fully aware of the fact that petrous pyramid suppuration is a complication of an otitic lesion, and the otitic lesion leads to a mastoiditis and then, after an interval of time, the petrous tip suppuration may or may not develop.

In this paper no attempt will be made to discuss the treatment of cases of petrous tip suppuration. The paper will consist, in the main, of the anatomy, pathology, symptomatology and a short discussion of the diagnosis of this type of lesion.

Before proceeding with the details of this paper, it is necessary to enunciate certain fundamentals to clarify some misconceptions of this

entity. Considerable confusion exists regarding the so-called Gradenigo syndrome, the pathologic changes underlying those cases which present trifacial pain and abducens paralysis, sometimes accompanied, but more often unaccompanied by changes in the cell content of the cerebrospinal fluid. In the discussion which is to follow, the writer is not dealing with the so-called Gradenigo syndrome, except incidentally. Most otologists hold with Vogel (32) that the series of events which are usually characterized by the term Gradenigo syndrome are not characteristic of a specific lesion and do not form a distinct clinical entity. The uncritical acceptance of the idea that a distinct clinical entity formed the basis of the Gradenigo syndrome is one of the reasons for the confusion which exists with regard to the real significance of suppuration of the petrous pyramid.

Gradenigo (33), in his original studies, brought forward no histologic evidence to support his belief that the triad of symptoms he described are diagnostic of any one organic pathologic condition. Vogel (32) sums up the argument against the triad as representative of a given pathologic condition by stating that "when a symptom complex is beclouded by other factors or varies, it no longer is a symptom complex characteristic of a certain condition. Otogenic paralysis of the abducens is not diagnostic of affection of the petrosal tip, and in itself does not furnish indications for extensive operative procedures on the pyramidal tip".

Suppurations of the petrous pyramid are of two varieties: (a) frank suppuration of the pyramid, more particularly its tip, and (b) osteomyelitis of the pyramid. In this discussion reference will be made only to (a). While osteomyelitis of the petrous pyramid ultimately leads to

the endo-cranial structures, its route of advance is not as specific, it does not form as marked a clinical entity in its development and the treatment applicable to petrous tip suppurations can not be used. It is of rarer occurrence than the lesion giving frank suppuration in the petrous pyramid, and it happens to occur in diploetic rather than pneumatized bone.

It is on the works of Wittmack (24) and his theory of pneumatization that the explanation of the mechanics of invasion of the petrosal pyramid, of the symptomatology which follows invasion of the peri-labyrinthine air cells and of the causation of chronicity in middle ear infections which, occuring later than early infancy, have their onset in an acute middle ear infection in a fully pneumatized temporal bone, is based.

The writings of Profant, (17) Eagleton (18), Kopetzky and Almour (27), Friesner, Druss (16), and Voss (2) have stimulated a widespread interest in the subject of supporation of the petrous pyramid during the past few years. As far back as 1904, Mouret (39), carried out systematic anatomic studies on the petrous pyramid. At that time he evidenced his understanding of infection of the petrous tip. He stated that "if the cellular arrangement favors an infection of the mastoid process in the presence of suppuration of the middle ear and facilitates the retention of pus in that part of the temporal bone, it is easy to understand how the pus can be extended into the perilabyrinthine cells and cause necrosis of the petrous pyramid".

Also, during the past few years considerable interest has been aroused as regards certain anatomic and pathologic characteristics of the petrous bone. It has been through the efforts of many writers that a restudy of the anatomy of the petrous bone has thrown much light on the pathological possibilities.

In a study of fifty temporal bones, twenty-five being studied microscopic and twenty-five macroscopic or gross. in which an effort was made to determine the variation in the distribution of bone marrow and pneumatic spaces in the apex. In four of the seven there were paratubal cells. Of the twenty-five microscopic studies bones, ten or 40% had pneumatic spaces in the apex. In nine out of the ten paratubal cells were found. Taking the total of the fifty bones, there were seventeen, or 34% with pneumatic spaces in the apex. In both bones, that is, right and left sides respectively, pneumatic spaces extended above and below the labyrinth from the antrum region to the apex.

Pneumatic spaces were frequently seen, about the canals and vestibule, although not extending to the apex. Marrow was found in the petrous apex in forty-seven, or 94% of the bones. Marrow seems constant in the region. Marrow spaces also noted along the posterior border of the petrosa behind the internal auditory meatus in nine, or 18%, of the total. A small amount of marrow was found in a few instances in other parts of the sections, such as the bone lateral to the tympanic cavity. The main types of bone marrow found were the red and fatty and a combination of the two. The fatty marrow predominated, especially in the petrous apex. It was evident that marrow alone could exist in the petrous apex, but when pneumatic spaces were present there, marrow also was present. Examination of the carotid wall showed dehiscences in several instances. Anatomic picture in the right and left bone of the same patient may be similar or it may vary. Also, interesting observations made by this writer were the distances from the basal part of the cochlea and the carotid artery; he found it to vary from less than one to four mm., being one to three mm. in most of the bones. This distance increased, however, just below the tegmen tympani at the level of the canal for the tensor tympani and the tympanic opening of the eustachian tube, because the cochlea had receded at this higher level and the carotid canal had coursed forward. Here the distance between the cochlea and the carotid canal varied from about four to ten mm. giving an average of 6.2 mm.

These observations bring out certain interesting anatomic and practical points and further support statements made in the literature. There seems to be no reason why a given bone may not present any one of the number of variation. Thus, one bone may have pneumatic spaces in the antrum

region alone; another may have spaces about the vestibule and canals as well, and a third may, in addition, have spaces in the petrous tip. The pneumatic spaces may vary in size from those that are small to those that give the petrosa a cellular appearance.

Myerson, Rubin and Gilbert (I) studied a series of two hundred temporal bones. These bones were obtained from the skulls of one hundred persons ranging in age from seven months of fetal life to eighty-nine years.

Pneumatization of the petrous apex was encountered in eleven percent of the specimens. Pneumatization does not appear to occur as frequently as has been supposed, especially in children. Up to and including seven and one half years, there were no pneumatized apexes.

According to these writers, the cell-groupings was simple. In this series also there was a greater incidence of pneumatization in the skull of colored people than in those of the white race.

Normally, the tympanic cavity is transformed immediately after birth from a space filled with embryonal connective tissue into one containing air. During the process of development, which begins soon after birth, the infantile mastoid process, a spongy bone containing marrow between its trabeculae, is subjected to pneumatizing influences of the tympanic mucosa. In brief, the process of pneumatization consists of an ingrowth of the subepithelial embryonal connective tissue into the marrow spaces which communicate with the air containing antrum. This connective tissue displaces the marrow originally contained between the bony trabeculae. With increasing age of the individual, the embryonal tissue begins to undergo a certain amount of contraction and fibrosis so that it becomes contracted down toward the bony walls. In this process it draws with it

the epithelial lining, first from the antrum into the neighboring cells, and then from the latter into other cells adjoining them, and so on. As the subepithelial tissue contracts still further, it eventually reaches its adult structure as a thin layer of connective tissue stroma closely applied to the trabeculae. Covering this is the epithelial lining. Thus a series of air containing chambers is created from formerly marrow containing spaces, and these communicate with the middle ear through the antrum. (5) (19)

Examination of a well pneumatic temporal bone discloses the fact that pneumatic cells are by no means limited to the mastoid portion of the bone. They are seen to occupy the squama and the zygoma also. They are found in the floor of the middle ear, under the tegmen and more extensively around the mouth of the eustachian tube. It thus becomes evident that wherever there are marrow spaces which are in contact with the tympanic mucosa, the latter can exert its pneumatizing influence and convert them into air containing chambers, which will then communicate directly with the middle ear.

From an embryonic standpoint, the petrous bone in reality consists of two distinct types of bone. One, the otic, or labyrinthine capsule, houses the membranous labyrinth and conforms to its contour and windings. It, in turn consists of three distinct layers of bone. The innermost, the one immediately bounding the membronous labyrinth, is termed the endosteal layer and, as its name implies, is derived from the connective tissue endosteum which every where lines the perilymphatic space. The intermediate, or endo-chondral layer, is developed from cartilage and shows what is peculiar to this layer, a retention of islands and strands of

cartilage throughout life. These two layers are compact bone and have reached their full size at birth. The outer or periosteal layer is connective tissue bone derived from the periosteum. That portion of it which forms the inner tympanic wall comes in contact with the tympanic mucosa and, in its developmental stage, may be subjected to the pneumatizing power of that tissue. It therefore happens that very often the periosteal layer shows definite tracts of pneumatic spaces which surround the other layers. Eagleton (14), in his studies of the cadaver, in three out of nine cases found tracts of cells situated in the perilabyrinth in the periosteal layer which led from the region of the superior semicircular canal directly to the petrous tip.

The remainder of the petrous bone, excluding the otic capsule, is normally a spongy bone containing marrow spaces. However, if subjected to the pneumatizing influence of the tympanic mucosa, either by way of the peritubal cells or via the peri-labyrinthine cells, it can also become converted into a bone consisting of air containing spaces. Various observers have reported in the literature findings of large pneumatic spaces in this portion of the petrous bone.

Other anatomic factors of importance in the comprehension of the lesion are the relationships of the various soft tissue structures which come in contact with or pass through the temporal bone. These are as follows: The membranous labyrinth; The fifth, sixth, seventh, eighth, ninth, tenth and eleventh nerves, the carotid artery; the eustachian tube; the petrosal nerves. These will be considered in turn as given. (3)

The Membranous Labyrinth: This structure is housed in the otitic capsule, which is always seen as compact bone. No reports have yet appeared to show that pneumatization ever occurs in the inner two layers of this bony capsule, consequently it is protected from invasion at all points except natural openings into it, vix., the oval and round windows, the endo-lymphatic and cochlear aqueducts and the internal auditory meatus. An involvement of the perilabyrinthine cells or of those in the petrosal tip may therefore cause an irritation of the membranous labyrinth, but not an inflammatory disease, unless erosion occurs into one of the preformed channels.

The Fifth (Triggminal) Nerve: The relationship of this nerve to the petrous portion of the temporal bone is of the utmost importance in understanding the symptomatology of the lesion. The fifth nerve, both motor and sensory roots, after arising from the pons, passes forward and, through an oval opening in the dura above the internal auditory meatus, reaches the superior border of the petrous bone. It then runs between the bone and the dura, along the superior surface of the petrosa, to the apex of the pyramid where the sensory roots take the form of a large semilunar ganglion, known as the Gasserian ganglion. This lies in a depression on the upper surface of the petrous apex, which, with the overlying dura to which the ganglion is firmly adherent, forms a pocket for its reception. Underneath the ganglion lies the great superficial petrogal nerve. From this ganglion three large branches are given off, the ophthalmic, the superior maxillary and the inferior maxillary.

It is with the ophthalmic branch of the fifth nerve that we are most concerned. Within the caverum Meckli, the ophthalmic branch has a longer course than the other two divisions. In this area it is firmly bound down and can be separated only with difficulty from the overlying dura and from the superior surface of the petrosa to which it is attached. In addition, it is closely adherent to the cavernous sinus. $(/\mathcal{B})$ The second and third branches of the fifth nerve, on the other hand, are not bound down in their course. In the ganglion the fibers rest on the carotid artery and the branches are not adherent to the dura. Consequently, any disturbance of the position of the Gasserian ganglion will cause tension to be exerted on the ophthalmic division first, while the other two branches, because of their flexibility, can accommodate themselves for a time to their new position.

The Sixth Nerve: (Abducens) The dissections of this nerve by Dorello, Vail (34) and Wheeler (35) and lately by Eagleton (18) have given us an accurate idea of its relationship to the petrous bone. After leaving the lower border of the pons, the nerve runs upward and outward to pierce the dura over the sphenoid bone. It then turns forward and passes between the apex of the petrous bone and the posterior clinoid process of the sphenoid. The space between these two bony structures is formed into a canal by the interposition of a very strong fibrous bundle known as Grubers ligament, of the sphenoidal ligament. This ligament forms, with the upper margins of the petrous bone, a three cornered space, in which lie the superior petrosal sinus and the abducens nerve. This canal has been termed Dorello's canal.

Eagleton's (18) dissections of this area show difference in the length of the nerve in this canal in different specimens. In addition, the nerve is sometimes found tightly constricted and immoblized at this point, whereas in other specimens it is fairly loose and mobile. Eagleton has determined also that Dorello's canal varies as regards position, shape and size, all of these factors being determined by the structure of the bone basii, cranii.

The Seventh (Facial) Nerve: This enters the internal auditory meatus with the eighth nerve, lying at first to the inner side of the latter and above it1 At the depth of the meatus it enters the fallopean canal, running outward to reach the inner tympanic wall, where it forms reddish swelling known as the geniculate ganglion. At this point the large and small superficial petrosal nerves are given off, the former to connect with the sphenopalatine ganglion and the latter with the otic ganglion. It also gives off, at this point, the external superficial petrosal nerve, which communicates with the sympathetic filaments, accompanying the middle meningeal artery. The remainder of the course of the seventh nerve is within the fallopian canal, until it emerges from the stylo mastoid foramen, where it is distributed to the muscles of the face. The chorda tympani nerve is intimately associated with the facial from a quarter of an inch above the sylomastoid foramen backward into the medulla. (19)

The Eighth (Acoustic) Nerve: This nerve enters the internal auditory meatus and divides into its cochlear and vestibular portions, to be distributed respectively to the spiral and Scarpa's ganglion.

The Ninth (Glossopharyngeal) Nerve: This nerve leaves the skull at the central portion of the jugular foramen and is enclosed in a separate sheath of dura and arachnoid. It occupies a position anterior to the eleventh and twelfth nerves. In its passage through the jugular foramen it forms a groove on the lower portion of the petrous portion of the temporal bone. It is the sensory nerve of the pharynx, fauces and tonsil; it is the motor nerve of the pharyngeal muscles and a special sensory nerve of taste to the posterior third of the tongue. Within the jugular foramen it presents two ganglionic enlargements. The upper is called the jugular ganglion, and the inferior is the petrosal ganglion. The latter sends communicating branches to the vagus and the sympathetic. It also gives rise to the Jacobsen's nerve, which enters a bony canal in the under surface of the petrosa, and then the middle ear, where it forms the tympanic plexus. From here branches go to the round and oval windows and the lining of the eustachian tube and tympanum. The tympanic plexus, sends communicating branches also to the carotid plexus, the greater petrosal nerve, and a branch from the facial, the lesser superficial petrosal nerve.

The Tenth (Vagus)Nerve: This is both a motor and a sensory nerve. It emerges from the skull through the jugular foramen accompanied by the spinal accessory, the two occupying a position posterior to the glossopharyngeal, from which they are separated by a membranous septum. It has a wide distribution, supplying the larynx, heart, lungs, esophagus, and stomach. It receives communicating filaments from the seventh, ninth and eleventh nerves.

The Eleventh (Spinal Accessory) Nerve: In the area of this nerve both its spinal and accessory portion accompany the vagus in its passage

through the jugular foramen. It supplies motor filaments to the trapezius muscle and to the sterno mastoid. Its accessory portion joins the vagus.

The Petrosal Nerves: As above described, these arise from the facial and glossopharyngeal nerves. The former gives rise to the great superficial petrosal, the latter to the small superficial petrosal. The great superficial joins with the deep petrosal from the carotid plexus and both run in grooves situated on the superior surface of the petrous bone. They eventually join to form the vidian nerve, which enters the sphenopalatine ganglion, and so communicates with the superior maxillary branch of the fifth nerve. The lesser superficial petrosal passes then through the petrous bone to enter the otic ganglion. The latter is situated immediately below the foramen oval and lies on the inner surface of the inferior maxillary nerve. Internally it is in relationship with the cartilaginous portion of the eustachian tube.

The Internal Carotid Artery: This enters the carotid canal in the petrous bone, runs upward for a short distance, then curves forward and inward, and again ascends as it enters the cranial cavity. At first, it lies below and in front of the cochlea and middle ear, then behind and internal to the eustachian tube. Further on it is separated from the Gasserian ganglion by a thin plate of bone, which is often absent, and replaced by fibrous tissue.

From the cases reported in the literature, from cases observed by men in this field and from macroscopic and microscopic examination, there is unquestionably a distinct pathologic entity which has been termed suppuration of the petrosal tip.

The various avenues of infection are:

1. From the antrum or epitympanic space, above or below the superior semi-circular canal, following the posterosuperior surface of the petrosa into the pyramidal tip.

2. From the peritubal cells into the pyramidal tip.

3. From the peritubal cells directly into the carotid canal or through dehiscences in the anterior tympanic wall into the carotid canal and then rupturing into the cavum Meckeli.

Most of the observation in regard to the avenues of infection are macroscopic and tend to favor the peritubal cells and the carotid canal as the avenues of invasion. However, the microscopic studies of cases of petrosal tip suppuration show the pathway of infection to be along the perilabyrinthine cells originating in the antrum and the epitympanic space. Wheeler (15) Druss (16) and Friesner (16) all have been able to demonstrate that a definite connection exists between the peritubal cells and the petrosal tip. Wheeler injected fluid into the opening of one of the pertubal cells and this fluid filled the pyramidal tip and leaked out into the middle cranial fossa.

According to Druss (31) infections of the middle ear and antrum have particular affinity for certain groups of cell: these are in order of their frequency: 1. Mastoid process, 2. Fossa sigmoida, 3. Tegmen

tympani, 4. Petrosa, including the labyrinth, 5. Posterior limb of the external semi-circular canal and carotid canal. While the predilection for the cells of the petrous pyramid is fourth on the list, nevertheless a petrositis is far more prevalent than was previously believed. This has been demonstrated both clinically and histologically. While comparatively little work has been done on the gross anatomy, still less has been done on the histology of the apex of the petrous pyramid.

Microscopic evidence, however, seems to disprove the peritubal cells as the avenue of invasion and established the perilabyrinthine route as the pathway of infection. Lange (36), in a microscopic study of two cases of pyramidal tip suppuration, found identical pathologic conditions in both instances. In the vicinity of the petrous tip was a large abscess bordering on the dura. From this area the serial sections demonstrated the tract of infection to proceed along the cells above and behind the superior semi-circular canal, which led directly to the mesial antral wall. The peritubal cells in the two cases were uninvolved and showed no connection with the suppurative focus in the petrous tip. Lange is of the opinion that petrosal tip suppurations have their origin in the antrum and extend along the tract described, and that in those instances wherein the peritubal cells were noted to be involved, the tubal area represented the avenue of escape of the purulency. Kopetzky (29) is inclined to accept this view as a logical one and has given clinical proof of it, which will be discussed later.

In considering petrositis, Friesner, Druss, Rosenwasser and Rose (25), think it is of importance to include pathologic changes which occur

not only in the apex but in the perilabyrinthine structures as well. Thus, cases have been reported in the literature in which the condition was termed perilabyrinthitis and deep extra dural abscess, when, in reality it should be considered as petrositis. It also must not be forgotten that by no means in all cases does the lesion in the pyramid extend to the apex, nor are the symptoms dependent on an extension of the lesions to the apex.

In twenty-four cases in which the temporal bone was sectioned serially, a histologic diagnosis of petrositis was made. In twenty of these sections were made to the tip. Of the twenty-four cases of petrositis the pyramid was pneumatic in four cases (16%), mixed in eighteen (75%) and diploic in two (8%). It must be borne in mind that in the determination of the type of anatomic structure of the temporal bone, no definite criteria have been established and the personal interpretation of the observer is an important factor. Of course, as said before, age is an important fact also.

In the cases of Friesner and Druss, (16) the first case showed t the extension of the infection from the antrum to the cells in the perilabyrinthine area along the posterior margin of the superior semi-circular canal and thence to the pyramidal tip. The second case likewise showed this route of infection; but in this instance an egress for the pus was established in the region of the internal auditory meatus, so that the petrous tip showed inflammatory involvement to a lesser extent than in the first case and also several cases reported by others which are contained in this article.

It seems that the pathologic picture in all cases studied postmortem show an extension of the purulent focus in the middle ear into the petrous tip through one of the channels above mentioned. Wilkinson's (37) first case showed a cariors process in the petrosal apex which had eroded both the anterior and posterior surfaces of the pyramid. A large cavity was present in the pyramid, filled with granulation tissue and pus. The Gasserian ganglion was edematous. His second case showed a large cavity in the petrous apex, filled with cheesy pus and detritus. An erosion into the middle cranial fossa occured which caused the Gasserian ganglion to be surrounded by granulation tissue and to be edematous. The route of invasion in this case was through the supratubal cells. Friesner and Druss record similar findings of purulency within the petrous pyramid.

The above findings are in cases wherein a purulent process within the petrous pyramid extended into the cranial cavity and resulted in death. In all of these cases it is evident that an original focus within the middle ear eventually spread to involve not only the mastoid process but also the petrosal cellular tissue and eventually resulted in exitus of the patients, due to a rupture into the endocranium. From a clinical observation of nine cases of Kopetzky (29) petrosal tip suppuration, five of which have recovered and are still living, he is prepared to add to the post mortem findings several additional factors which can be observed during the progress of the case from a single otitic suppuration to its final stage in the petrosal tip. These findings are:

1. All of his cases had an extensively pneumatized mastoid process.

At the primary operation the cells extended well forward into the zygona. In the region of the antrum and in the area posterior to the superior semicircular canal, cellular elements were present.

2. As the suppuration in the mastoid process and middle ear clears up the suppurative process spreads into the perilabyrinthine tracts toward the pyramid.

3. After a period during which the middle ear remains dry, there suddenly reappears a profuse aural discharge as a source of which the mastoid wound can be definitely ruled out, for it appears healthy and contains no pus. This finding leads one to believe that the pus within the petrous pyramid finds an avenue of escape by rupturing through the cells around the eustachian tube and then out through the middle ear.

In all of Kopetzky's (3) cases of suppuration of the petrous pyramid, he has been impressed by the recurrence of a discharge from the middle ear after the mastoid wound had completely healed and after the middle ear had been dry for a time. In the cases upon which he has operated for petrous tip suppuration and which have recovered, two still present a chronic aural discharge. The radical cavity is completely epidermitized, there is no rhinopharyngeal suppuration, inflation does not blow any discharge into the cavity from the tube, and yet a discharge can be seen coming from the region of the tubal ostium at the site of entrance into the pyramidal tip. In one of his cases, in which cleared up without operation, a persistent discharge is present with a completely healed mastoid process. In the section on symptomatology the clinical significance of this will be discussed further. The latter is presented

as an additional factor in petrous tip suppuration which has a close relationship to the interpretation of protracted aural discharge in the presence of a pneumatized mastoid process. Kopetzky (29) states that with a protracted acute middle ear infection after operation, one must think of a pyramidal tip suppuration.

The symptomatology of suppuration of the petrous tip can be divided into four periods (28); the period of eye pain and aural discharge; the period of low grade sepsis; the period of quiesence; and the terminal stage. As a rule, only the fourth stage appears as an entity; but in order to develop the clinical picture so as to describe a sequence of events, the grouping of the symptoms as described is deemed advisable. Later on, the entire symptomatology will be correlated. In this section an effort will be made to explain each symptom, give its clinical significance and analyze it anatomic and pathologic cause.

It can be said here that in going over the cases in the literature, most of the reported cases of petrous tip suppuration occur in patients whose mastoid process showed extensive pneumatization. Before the primary operation on the mastoid process, extensive ramification of the cellular elements was always noted on radiographic examination. At operation, this observation was verified. The cells themselves, or the spaces created by their coalescence due to the disease, were found to occupy large areas of the zygoma, squama and occipital bone and to surround the base of the petrosa completely. The region of the socalled solid angle contained cellular elements or showed areas of necrosis. These findings are significant for demonstrating that the process of pneumatization has extended beyond the limits of the mastoid process, they suggest the possibility that the petrous pyramid is pneumatic. The presence of extensive pneumatization in the temporal bone should help

one to interpret later signs and symptoms, if and when they appear. (3)

Friesner, Druss, Rosenwasser and Rosen (25) have said, contrary to the opinion of many, that there are no symptoms which are invariably associated with this condition. They say that the most significant symptom is pain. Furthermore, provided other etiologic factors have been excluded, the presence of pain is much more important than its localization. They have found pain referred to the temple, the supra-orbital region, the eye, the teeth, the face and the ear. Rarely, pain from petrositis is referred to the posterior part of the skull. The pain may be intermittent, but more frequently it is continuous with marked exacerbations. Pain from this condition may occur during the first week of otitis or may begin in from one to a number of weeks following a mastoidectomy. Generally speaking, after an adequate operation for suppurative mastoiditis, the later the onset of pain the graver is its significance. Perhaps pain which comes on late is a manifestation of meningeal irritation or other intracranial complications of insidious osteitis of the petrous pyramid. In all of the cases reported by the former writers, the cessation of pain marked the beginning of recovery. They further add that the generally accepted belief that serious disease in the petrous portion of the temporal bone is always associated with persistent or recurring discharge from the tympanum is also erroneous. In the presence of pain a scant discharge is of much more grave import than a profuse discharge. Almour (38) reports a case and says: "Just as all of our cases have shown the symptoms characteristic of a petrous suppuration. this case also presented the deep seated eye pain and the continued

otorrhea which could not be accounted for by any lesion within the mastoid process itself". The prompt recovery obtained following the surgical procedure which he has advocated for the relief of this condition is further evidence that a deep-seated suppuration within the petrous apex can be attacked surgically before and invasion of the endocranium occurs and its subsequent trend of events.

EYE PAIN: This is, in the majority of instances, the first symptom to make its appearance. Its location and character are so typical that it is almost diagnostic of petrosal tip suppuration in itself where the anatomic structure already described has been found.

The pain is on the side of the lesion. It is limited to the region about the eye and is felt within the orbit itself. It is described as a deep-seated ocular pain and, at the onset, is nocturnal in character. During the day the patient is more or less comfortable; but as evening comes on, the pain becomes more and more intense. The patient describes it as being just above the eye and through the eyeball.

The peculiar type of pain is highly significant of a petrosal tip suppuration. It is the result of an irritation of the ophthalmic branch of the trigeminal nerve, which, as has been pointed out in the first section of this paper, is firmly bound down in its course from the ganglion to and through the cavernous sinus. This branch, altogether sensory in function, supplies the eyeball, the lacrimal gland and the skin of the nose, upper eyelid, forehead and scalp.

The presence of retro-orbital pain early in the disease is explained by Eagleton (14) as due to an inflammatory reaction of the dura

overlying the diseased portion of the petrous tip and the petrosphenoid articulation. In an analysis of the clinical picture presented by his two cases, Lange (36) gave prominence to the deep seated eye pain. In eight cases reported by Kopetzky and Almour (3) this typical eye pain was the initial symptom.

The presence of post-operative pain after surgery on the mastoid process is to be expected. Where, however, this pain assumes definite characteristics, it is usually significant of some complication. A dull aching pain felt in the side of the head and persisting for a time after operative interferences, is very significant of lateral sinus thrombosis. (40) Pain in the nape of the neck or in the occiput makes one suspicious of an impending meningitis if it follows mastoidectomy. Similarly, deep seated eye pain is a distinct symptom of a lesion in the middle cranial fossa which is irritating either the ophthalmic branch directly or its root in the semi-lunar ganglion. A posterior fossa lesion cannot, for anatomic reasons, involve the region of the Gasserian ganglion. The tentorium so completely separates the middle from the posterior fossa that a localized purulent focus within the latter cannot possibly, even with its surrounding inflammatory reaction, involve an isolated portion of the fifth nerve, which is situated wholly within the middle cranial fossa.

Other branches of the fifth nerve besides the first may be involved if the inflammatory reaction is widespread. Pain will then be felt all along the area supplied by the second and third branches. This pain is not diagnostic, however, as it can be associated with cases of uncomplicated middle ear abscess, and mastoiditis. Any irritation of the

23

geniculate ganglion of the facial nerve may cause pain to be referred to the area supplied by the superior maxillary branch of the trigeminal. This occurs through the communication established between the geniculate ganglion and the second trigeminal branch of the great superficial petrosal nerve. The mandibular branch of the fifth nerve, through its connection with the otic ganglion, receives sensory fibers from the small superficial petrosal nerve. This may be irritated either at its origin in the tympanic plexus or in its passage through the petrosa to reach the otic ganglion. Irritation of Jacobsen's nerve or the tympanic plexus may cause pain to be referred along the distribution of the superior maxillary nerve through the great deep petrosal, which joins with the great superficial petrosal.

It is therefore clear that pain in the face and teeth can occur with a suppurative lesion anywhere in the middle ear or mastoid process. This pain will be relieved, however, as soon as the source of irritation in the middle ear or mastoid is removed. Pain felt in the region of the orbit, due to irritation of the ophthalmic branch, of the fifth nerve, must be caused by a lesion in direct proximity to this branch, for it has no connection with other sensory nerves in the petrosa.

When surgical removal of the purulent focus in the mastoid process and middle ear does not result in a cessation of the pain distributed over the areas supplied by the second and third branches of the fifth nerve, the persistence of the pain should be viewed as suspicious of a petrosal tip suppuration. Spasmodic pain is more apt to be associated with an idiopathic lesion of the Gasserian ganglion itself, like tic

suppuration, one is more likely to get a constant headache than a spasmodic pain.

THE AURAL DISCHARGE: It has been the experience of most otologists that simple mastoidectomy which has established adequate postauricular drainage will cause the middle ear to cease discharging within a week or two after operation. In most cases of petrosal tip suppuration either the middle ear continues to discharge until the lesion in the petrous tip is identified and eradicated, or else after a period during which the ear was dry, a profuse discharge suddenly reappeared, at the same time as, or shortly before, the onset of the eye paih. (30)

In the first section of this paper the significance of petrous tip suppuration was discussed as the cause of a type of chronic otorrhea in patients showing a well pneumatized mastoid process. An acute middle ear suppuration which has a coalescent mastoiditis as a complication will, in most cases, undergo resolution after the mastoiditis has been eradicated. The otorrhea may persist after simple mastoidectomy on a well pneumatized mastoid process, if the acute lesion in the middle ear was of the type known as "acute necrotic otitis". This variety occurs in scarlet fever and in the course of a debilitating disease such as diabetes. When it is present, the greater portion of the mucosa of the tympanum undergoes necrosis, exposing the underlying periosteum and bone to the suppurative focus. A rarefying ostitis, or caries, results, which is evidenced clinically by a marginal perforation and bare bone. In the reparative stage of the lesion the squamous epithelium of the external auditory canal plays a part in the attempted cure of the patho-

logic state, and an ingrowth of epidermis occurs which eventually results in a secondary cholesteatoma. Every otologist is familiar with the rapid appearance of a cholesteatoma in certain cases of this type wherein a thorough simple mastoidectomy has been performed. The squamous epithelium in its ingrowth into the middle ear finds a large preformed cavity in the mastoid process, into which it grows rapidly, eventually forming a large cholesteatoma. This then, is the other cause of a continued otorrhea following acute suppurative lesions in the presence of a well pneumatized mastoid process.

In a series of nine cases, the largest number encountered in the writer's somewhat futile survey of the literature, Kopetzky and Almour could not account for the continued otorrhea by any of the foregoing factors. (3) Kopetzky (28) considers the reappearance of otorrhea in conjunction with the eye pains significant factor in the symptomatology of petrosal tip suppuration. He states when an acute middle ear suppuration is protracted after operation, one must think of a suppuration of the petrosal tip. Eagleton's (14) cases, as well as others reported in the literature, all show this recurrence or continuance of a discharge from the tympanic cavity.

As already stated, the recurrence of the otorrhea on the basis of the observation made by Lange (36) is explained. In most instances, the pus in the petrous tip drains into the middle ear through the channel created by the peritubal cells. In some cases, the pus drains out through the avenue of invasion, which is located in the region of the inner antral or inner tympanic wall.

OTHER SYMPTOMS AND SIGNS OCCURRING DURING THIS PERIOD: It has been the experience of most writers that the eye pain and the aural discharge constitute the salient findings which are invariably present early in the course of a petrosal tip suppuration. There have been other signs which occasionally present themselves in the early stage of the lesion. They are included at this point merely as corroborative, but not of diagnostic data.

FACIAL WEAKNESS: Some cases show a distinct interference with the function of the seventh nerve early in the course of the disease. In some cases this symptom appeared prior to the onset of the eye pain. The disturbance in function was of the infra-nuclear type; at no time is there a complete loss of facial movements on the affected side. The duration of the facial weakness is short. The facial palsy can therefore be described as transient in nature, it occurs in the course of the disease but seems to clear up as the disease progresses. (32)

The facial nerve is affected in the spread of the infection from the inner antral wall through the perilabyrinthine cells. Where this tract of cells runs in a retro-labyrinthine direction, that is, behind the superior semi-circular canal, the infection will not encounter the facial nerve through its passage through the petrous bone. Where the infection spreads into the petrous tip through the peritubal cells, the facial will not be involved at an early date. It is only where the tract of cells encounters the facial canal in its passage through the petrosa, that an infection traveling along this tract may involve the facial nerve. A transient paralysis occuring in the post-operative period of an acute

mastoiditis and accompanied by deep seated eye pain and a profuse aural discharge, is therefore significant of the advance of an infection into the petrous tip through the tract of cells leading from the mesial wall of the antrum. In a majority of cases the facial weakness is accompanied by vertigo and nystagmus.

VERTIGO AND NYSTAGMUS: These are also produced by the perilabyrinthitis and are also transient in nature. That they are not the result of a lesion located within the perilabyrinthine or endo-lymphatic space, can be readily determined by the functional examination of the labyrinth. This elicits normal responses to both rotary and caloric stimulation. The Weber test shows a laterialization of sound to the diseased side.

The vertigo is not in any particular direction; the patients state for the most part that they are dizzy. Neither does the nystagmus have a distinct character or direction. Sometimes the direction shifts within a few hours. None of the patients assume the forced position seen in suppurative labyrinthitis. The vertigo and nystagmus disappears usually at the same time that the facial palsy disappears.

<u>VOMITING</u>: A very few of the patients have occasional vomiting spells during this period of the disease. The vomiting is not projectile and does not have any definite relationship to meals.

THE PERIOD OF LOW GRADE SEPSIS: The temperature in most cases of petrous tip suppuration is that of a low grade sepsis. In a survey of the literature the temperature is low in the morning, between ninety-nine and one hundred. Toward the late afternoon it would rise, in most instances, to 101 to 102. Post-operative temperature following mastoidectomy may have

many causes. Consequently, no significance can be attached to it unless other symptoms are also present to help identify the source of the fever. When a low grade sepsis continues after mastoidectomy, accompanied by eye pain and aural discharge, it is to be viewed as very strong corroborative evidence of a petrosal tip suppuration. The presence of fever indicates an inflammatory disease; the presence of eye pain indicates irritation of the ophthalmic nerve, the two together indicate an inflammatory lesion in the vicinity of the ophthalmic nerve.

<u>TEMPERATURE RANGE</u>: It may be said that an aural suppuration in itself can produce fever. This is very true; but with the additional factors of extensive pneumatization in the apex, the eye pain and perhaps vertigo and nystagmus, the fever assumes added significance and points to the diagnosis of a suppurative focus within the apex of the petrous pyramid. Eagleton considers the low grade sepsis an equally important diagnostic factor as the eye pain. He says, "Pain in the first branch of the fifth nerve, limited to the region behind the eye, is significant of irritation of the dura over the petrous apex, the presence of continuous sepsis, signifies caries of the petrous apex. (14).

It has been found that the fever stays at a low level until a meningitis appears or until the pus in the tip has been drained. In the former event a sharp rise in the temperature is noted; and the fever then continues at the high level characteristic of meningitis. Where drainage has been established, either spontaneously or by operative measures, the fever subsides and the temperature gradually returns to normal. (10)

THE PERIOD OF QUIESENCE: In all cases there appears an interval which is free from all pain of diagnostic import. This period of quiesence varies in duration from five to fifteen days. Before proceeding further, it must be repeated that the pain to which is referred, is the deep seated eye pain, associated with a low grade sepsis. As previously shown, the presence of trigeminal neuralgia alone or of pain not limited to the first branch of the trigeminal nerve in no way serves as a diagnostic symptom of petrous tip suppuration. Therefore, the presence and subsequent disappearance of pain in the areas supplied by the second and third trigeminal branches does not create what is designted as the period of quiesence. Only the presence of deep seated eye pain, in the company of low grade sepsis, and the subsequent abatment of this pain is referred to.

DURATION OF THE PERIOD OF QUIESENCE: From the standpoint of the patient's safety, this period is the most dangerous one, since it may lead both the patient and the surgeon to conclude that the lesion is clearing up. On the contrary, in the majority of cases, this period coincided with the invasion of the endocranium. In a very few instances it may signify spontaneous evacuation of the purulency in the tip through the middle ear.

When it is understood, as has been previously shown, that the eye pain is due to traction exerted on the ophthalmic branch because of the inflammatory swelling of the dura in the region of the petrous tip, it will also be understood that the relief of this inflammation will result in a cessation of the pain.

It is a well known fact that the pain in an acute coatescent mastoiditis is considerably lessened when the pus in the mastoid process ruptures through the cortex and forms a subperiosteal abscess. This is exactly what occurs in cases of petrous tip suppuration. The localized collection of pus in the apex at first causes an edema of the overlying dura with a consequent traction on the ophthalmic nerve, which is in proximity to it. As the lesion progresses, if sufficient drainage is not established through the peritubal cells or through the tract of invasion, the upper surface of the apex becomes eroded, either directly under the Gasserian ganglion or through the thin bony portion which separates it from the carotid artery. These are the two likely places for erosion, as the posterior wall of the apex is anatomically much thicker than the upper and outer, and consequently offers a greater resistance to the carious process.

The question now arises as to whether or not the direct involvement of the Gasserian ganglion by the purulent focus will result in the continuation or intensification of the pain. From the report of Turner and Reynolds (40) and that of Eagleton (41), it is doubtful whether inflammatory infiltration of the Gasserian ganglion itself will produce pain. Turner and Reynolds' case was one of cayernous sinus thrombosis wherein an inflammator infiltration of the ophthalmic nerve was demonstrated post mortem and yet the patient had no pain behind the eye. Eagleton's case, one of cayernous sinus thrombosis with a purulent infiltration of the semi-lunar ganglion, had no trigeminal pain.

If the patient's life is to be saved and a meningitis averted,

operative measures must be instituted prior to the stage of quiesence. In other words, the pus in the tip must be evacuated before it has ruptured into the subdural space. Therefore, the stage of quiesence is not to be utilized as a diagnostic aid in determining the presence of petrous tip suppuration.

THE TERMINAL PERIOD: The terminal period presents, in the main, the clinical picture of an acute purulent leptomeningitis. One notes a gradual onset of cervical rigidity, the appearance of photophobia, high temperature, Kernig, and Brudzinski reflexes, severe generalized headache projectile vomiting and a purulent spinal fluid. (28)

Whether or not any of the cranial nerves in the proximity of the petrosa will be involved depends upon the point of rupture in the apex. If the rupture occurs in the vicinity of the posterior fossa, the terminal stage will show involvement of the seventh, ninth, tenth, eleventh and twelfth nerves.

In none of the cases reported by Kopetzky and Almour (3) was the abducens nerve involved. This seems incongrous at first, because of the deeply rooted impression that a lesion of the petrous apex produces the so-called Gradenig syndrome (33). In the fore part of this paper the reason why the Gradenig syndrome, as such, is not diagnostic of petrous tip suppuration was pointed out, and it has been conversely shown that not all cases of petrous tip suppuration produce a paralysis of the sixth nerve.

Lange's (36) two cases had no abduces palsy, Eagleton's (14) cases had no sixth nerve palsy. On the other hand, many cases are on

record wherein an abducens paralysis has been found associated with a suppurative lesion in the petrous pyramid. Because of the fact that sixth nerve palsy is not a constant symptom in the sense that retro-orbital pain, low grade sepsis and persistent otorrhea are constant, it is not looked upon as a necessary factor in the establishment of a diagnosis of petrous tip suppuration. For one thing, the position of the abducens below the tentorium renders it more easily involved by lesions located in the posterior cranial fossa. In addition Eagleton's dissections, which have been alluded to in the section on anatomy, show that a number of factors influence the susceptibility of this nerve to lesion which might disturb its function in the vicinity of the petrous tip. The difference in length of Dorello's canal, the extent to which the petrosphenoid articulation is closed, variations in the size of the nerve itself and its freedom in the canal, all help to determine whether or not the nerve will be involved by a suppurative lesion in the petrosal tip.

When it is considered that most cases presenting the so-called Gradenigo syndrome go on to complete recovery, whereas cases of petrosal tip suppuration either terminate fatally or result in a chronic otorrhea if no attempt is made to eradicate the lesion surgically, it is inconceivable that the two conditions should be considered identical. Since cases have been with petrous tip suppuration without involvement of the abducens, it is illogical to look for the outward manifestation of external rectus palsy as a symptom diagnostic of petrosal tip suppuration.

It has been Brickers (II) experience that abducens palsy in the course of an otitic suppuration is most often associated with a mild type of meningeal inflammation. In other words, while a paralysis of the ex-

ternal rectus may occur in association with a purulent focus in the pyramidal apex, its presence in such cases is the exception rather than the rule. In fact, the presence of abducens palsy is more apt to point against than to a diagnosis of petrosal tip suppuration.

Perkins, (26) in 1910, reported ninety-four cases showing Gradenigo's syndrome with only eleven deaths, 12%. It may be interesting to add that the treatment at this time consisted of a simple or radical mastoidectomy. This may show that perhaps drainage of the tip is not always necessary to preserve life.

Roberts (8) reported four cases of proven petrous tip suppuration in which Gradenigo syndrome was present. Vogel (32) and Wheeler (35) believe that the so-called Gradenigo syndrome is necessary to make a diagnosis of petrous tip suppuration.

It can be seen that the modern concept of petrous tip suppuration is a matter of gradual development which began with the classic description of the symptom complex by Gradenigo in 1904 and again in 1907. (33) A review of the literature of this subject reveals the fact that the symptoms and signs now associated with this condition were known to pioneer otologists. The number of articles which have appeared in recent literature evidences the increasing attention which the subject has received during the past few years. Unfortunately, numerous divergent views have been expressed with regard to the symptomatology of the pathology, the prognosis and treatment. With regards to the symptomatology, the greatest divergence seems to be centered around the so-called Gradenigo syndrome.

LABORATORY DATA: The information which the laboratory furnishes in these cases is neglible. Only the x-ray can be used to decided advantage.

BACTERIOLOGIC FLORA: There are many reports in the literature which would tend to associate petrosal tip suppuration with the pneumococcus type III. The third case of Friesner and Druss (16) showed a pneumococcus type III, while the first case of Druss showed a streptococcus hemolyticus.

In nine cases reported by Kopetzky and Almour, (3) six of which were studied from the outset, of the lesion, and in two of which they had access to the earlier records, none showed a mucosus infection. Two showed streptococcus viridans, one a streptococcus non-hemolyticus. Two had a streptococcus hemolyticus which was not subcultured and the remaining three had a streptococcus pyogenes infection. It is the opinion of most writers that the organism has no etiologic relationship to the lesion in question. A streptococcus mucous infection occuring in a patient who has no pneumatic structure in his petrosa cannot possibly produce a petrosal tip suppuration. This holds true for other bacterial agents. In other words, the lesion in the petrosal tip depends upon the type of anatomic structure rather than upon the type of bacterial invader.

The tendency to consider that suppuration of the petrous tip is due to the pneumococcus type III, springs from the fact that in both cases the disease runs a protracted, almost symptomless course until fatal intracranial lesions supervene. However, the cases of a petrosal tip suppuration are carefully studied, it is at once seen that definitely

diagnostic symptoms make their appearance as soon as the tip is invaded by the suppurative focus.

In the case of an otitic infection caused by the pneumococcus III, the insidious advance of the lesion and the complete lack of prodromal signs are due to the inability of the bodily mechanisms to set up successful inflammatory barriers against this lesion. (6)

<u>BLOOD</u>: The red cells and hemoglobin are not affected to any extent by this lesion. Most cases show a mild secondary anemia such as is usually associated with any prolonged sepsis. The white count, which would be expected, under the circumstances present in these cases, to manifest a severe infection by a shifting to the left in the v.Schilling count, shows but a slight increase in the staff forms, and a moderate leucocytosis. Only toward the terminal stage, when the endocranium is already invaded and a purulent meningitis established, was there a sudden rise in the staff cells and a marked leucocytosis. (7)

For practical purposes, therefore, examination of the blood yields but little evidence beyond the fact that some infection is still present which is causing an increased leucocytosis. It gives no data concerning the severity of the infection until the terminal stage.

SPINAL FLUID: All cases show normal chemical, bacteriologic and cytologic findings prior to the terminal stage, when a purulent meningitis appears and spinal fluid findings are then those of a typical case of meningitis. The spinal fluid pressure as recorded by the manometer, is normal.

EYE GROUNDS: The eye grounds in all cases in the literature are normal.

<u>DIFFERENTIAL DIAGNOSIS</u>: Suppuration of the petrous tip must be differentiated from nasal accessory sinus disease, lateral sinus thrombosis, cavernous sinus thrombosis, thrombosis of the superior petrosal sinus, acute labyrinthitis and a reinfection of the mastoid process.

Nasal accessory sinus disease, when the frontal and sphenoid sinuses are involved, may very often simulate a petrous tip suppuration in the presence of an aural discharge. Both will give pain in the region of the eye. A sphenoiditis particularly may, by a neighboring edema of the cavernous sinus, give deep seated eye pain. In the absence of aural discharge, involvement of the petrous tip can be immediately excluded. Where an aural discharge is present, however probing the sphenoid sinus, posterior rhinoscopic examination and x-ray of the sphenoid will exclude this lesion. The same means are employed for ruling out a frontal sinusitis.

A cavernous sinus thrombosis is not a common complication of an aural suppuration. Where it occurs, it is the result of an extension of a thrombosis in the sigmoid sinus into the cavernous sinus. It is characterized by a septic temperature, positive blood culture, chemosis of the conjunctiva, Crowe-Beck sign, third, fourth and sixth nerve paralysis. In the presence of these findings, pain behind the eye can be ruled out as designating a petrosal tip suppuration.

Thronbosis of the superior petrosal sinus also results from the extension of a thrombosis located in the sigmoid sinus. In its extension this also may give rise to an inflammatory reaction in the neighborhood of the Gasserian ganglion and the sixth nerve. Its presence should be

expected in a case with eye pain when a sepsis continues after removal of the thrombus in the lateral sinus and blood culture is positive. In this lesion the discharge from the middle ear is not the characteristic described for petrous tip suppuration. Occasionally an atypical clinical picture of sinus thrombosis gives fifth nerve pain with absence of the classical picture of sinus sepsis. Here the assembled data from the roentgenogram of the tip helps the differentiation and locates the lesion in the pyramid.

In the presence of vertigo and nystagmus, an acute labyrinthitis must be ruled out. This is easily done by the functional test of the labyrinth. These tests yield normal findings in a case of petrosal tip suppuration, whereas, in acute labyrinthitis, findings varying from a hyperirritability to a complete loss of response will be found. Further more, the patient suffering from acute labyrinthitis assumes the forced position noted in such cases.

A petrosal pyramid suppuration is differentiated by the following factors from a reinfection of the mastoid wound, which a renewed discharge from the middle ear suggests. It shows no clinical manifestations of a renewed inflammatory process in the mastoid wound. There is no swelling of the incision, no increase in wound discharge. After the canal has been wiped dry, pressure over the recently healed scar does not cause pus to pour into the middle ear, showing that the source of the pus must be at a point remote from the mastoid wound. There is no marked increase in temperature such as usually accompanies a reinfection. There is no pain referrable to the wound and no undue tenderness beyond what one ordinarily elicits on pressure over a fresh scar.

A patient has an acute nasopharyngeal infection and then, in due course of time, develops an abscess in the ear. After an interval of time, an acute coalescent mastoiditis develops. (3) Radiographic examination at this time reveals an extensively pneumatized mastoid process which has undergone destruction. At operation a large mastoid cavity is uncovered with diseased cells located not only in the mastoid process but in the zygoma, squamosa, peri-bulbar area and the region surrounding the semi-circulars canals.

After a period of normal convalesence, during which the temperature returns to normal, the middle ear ceases to drain and the mastoid wound heals normally, the patient begins to complain of pain in the region of the eye on the side of the mastoid lesion. About the same time, there occurs a sudden profuse discharge from the middle ear, but none from the mastoid wound, which by this time may have healed entirely. The patient begins to run a low grade temperature. The pain in the eye is complained of mostly at night, and sometimes the patient has occasional dizziness and vomiting spells. A transient facial weakness may be present.

Examination at this time reveals a profuse otorrhea coming through a central perforation in the ear drum. There is a slight nystagmus which has no fixed direction. The fundi are normal. The blood count shows only a moderate leucocytosis with a slight increase in staff cells. Roentgenographic examination of the petrous pyramid at this time reveals distinct pathologic changes in the region of the pyramidal apex. If

the patient is operated, at this time, mpus will be evacuated from the petrosal tip, usually with subsequent recovery.

If the lesion is permitted to advance, the eye pain suddenly subsides and completely disappears for a time. The patient feels well and only the fever remains. After a short period of time a fatal meningitis supervenes.

The variation in this picture is not numerous. The case may heal spontaneously by the establishment of adequate drainage from the tip into the middle ear, in which case a chronic otorrhea will result.

Tayor (20) says that the most useful information in determining petrous pyramid suppuration is obtained by means of a base plate of the head (inferosuperior projectin). From this view, pneumatization of the petrosa can be determined, changes in aeration are evident and pathologic changes can be visualized. Where roentgen findings are observed in a case presenting suppuration, operative interference is indicated. In addition, cases presenting a protracted aural discharge, following simple mastoidectomy on a pneumatic bone, should have the petrosae radiographed to determine whether they are the cause of the chronicity. It is advisable to radiograph the petrosae as a routine procedure in every case of acute aural infection. Coates (22) and Ersner and Myers (23) are heartily in accord with this view. Kopetzky advocates x-ray studies of the petrosae in all cases of acute aural disease. (27)

It is the accepted opinion of the leading otologists of the country that all cases presenting the above picture should be operated upon and adequate drainage established for the pus in the petrous

pyramid. If operative measures are instituted early enough, many of these cases will be saved the fatal meningitis which "is the usual outcome of a petroal tip suppuration". (3)

CONCLUSIONS

CONCLUSIONS

I. THE ANATOMY OF THE PETROUS BONE MAY VARY CONSIDERABLY AS RE-GARDS PNEUMATAXITION.

2. MOST PETROSAE ARE PNEUMATIC, ESPECIALLY THOSE IN WHICH SUPP-URATION TAKES PLACE.

3. THERE IS NO CONNECTION BETWEEN THE SO CALLED GRADENIGO SYN-DROME AND SUPPURATIONS OF THE PETROUS APEX.

4. INTRA-CRANIAL COMPLICATIONS MAY OCCUR BY EXTENSION FROM THE ANTRAL REGION OR FROM AN INFECTED PNEUMATIC SPACE IN THE PETROSA.
5. AT THE PRESENT TIME, MOST CASES OF PETROUS TIP SUPPURATION ARE UNDIAGNOSED, AND USUALLY TERMINATE IN A FATAL MENINGITIS.

BIBLIOGRAPHY

BIBLIOGRAPHY

I. MYERSON, M. C.; RUBIN,; and GILBERT, J. C. ANATOMIC STUDIES OF THE PETROUS PORTIN OF THE TEMPORAL BONE. ARCH. OTOLARYNG. 201: 195. (August). 1934.

2. HAGENS, E. W.; ANATOMY AND PATHOLOGY OF THE PETROUS BONE BASED ON A STUDY OF FIFTY TEMPORAL BONES? ARCH. OTOLARYNG. (MAY) 1934.

3. KOPETZKY, SAMUEL J,; and ALMOUR, R.; THE SUPPURATION OF THE PETROUS PYRAMID,; PATHOLOGY, SYMPTOMATOLOGY and SURIGICAL TREAT-MENT. ANNALS of OTOL., RHIN., and LARYNG., 39:996, 1930. 40:157, 396 and 992, 1931.

4. GREENFIELD, S. D., ETIOLOGY AND PATHOLOGY OF PARALYSIS OF THE ABDUCENS NERVE ASSOCIATED WITH SINUS THROMBOPHLEBITIS, ARCH. OTOLARYNG., 19:336. (MARCH). 1934.

5. AREY, L. B., DEVELOPMENTAL ANATOMY. W. B. SAUNDERS COMPANY NEW YORK. MAY 1925.

6. KOPETZKY, S. J.; OTOLOGIC SURGERY, NEW YORK P. HOIKEN. SECOND EDITION., 1929.

7. FOWLER, E. P., Jr.; SUPPURATION OF THE PETROUS TIP. J. A. M. A. IO2:1651 (MAY) 1934.

8. ROBERTS, E. R.; INFECTION OF THE PETROUS APEX, LARYNGOSCOPE 44:274, 1934. 9. SUNDE, E. A: , INFECTION OF THE PETROUS BONE. RATIONALE OF TREATMENT AND REPORT OF A CASE. ARCH. OTOL., RHIN., and LARNYG., 19:436 (APRIL) 1934.

IO. GREENFIELD, S. D. CONSERVATIVE TREATMENT OF PETROSITIS;
REPORT OF TWO CASES WITH RECOVERY WITHOUT OPERATION, ARCH.
OTOL., RHIN., and LARYGN., 20:172 (AUGUST) 1934.
II. BRICKER, S.; SUPPURATION OF THE PETROUS PYRAMID. LARYNGOSCOPE. 44:284, 1934.

12. SMITH C. H.: RECURRENT MASTOIDITIS WITH PETROSITIS., TEMPOROSPHENOIDAL ABSCESS, LARGE EPIDURAL ABSCESS AND RE-COVERY., LARYNGOSCOPE. 44:221., 1934.

I3. MYERSON, M.C., RUBIN, H. W., and GILBERT, J. G.: IMPROVED
OPERATIVE TECHNIC FOR SUPPURATION OF THE PETROUS APEX. ARCH.
OTOL., RHIN., and LARYNG., 19:699 (JUNE) 1934.
I4. EAGLETON, W. P.: LOCALIZED BULBAR CISTERNA (PONTILE)
MENINGITIS, FACIAL PAIN AND SIXTH NERVE PARALYSIS AND THEIR
RELATION TO CARIES OF THE PETROUS APEX. ARCH. OF SURGERY. VOL.

20., pp. 386, 1930.

15. WHEELER, J. M., PARALYSIS OF THE SIXTH NERVE ASSOCIATED WITH OTITIS MEDIA. TRANS. SEC. OPHTH. J. A. M. A. 1918, pp. 51. 16. FRIESNER, I., DRUSS, J. G.: OSTEITIS OF THE PETROUS PYRAMID OF THE TEMPORAL BONE. ARCH. OTOLARYNGOLOGY, VOL. 12, pp.342, 1930. I7. PROFANT, H. J.: GRADENIGOS SYNDROME,: WITH CONSIDERATION OF PETROSITIS, ARCH. OTOLARYNG. I33:347. (MARCH) 1931.
I8. EAGLETON, W. P. : UNLOCKING THE PETROUS PYPAMID FOR LOCALIZED BULBAR (PONTILE) MENINGITIS SECONDARY TO SUPPURATION OF THE PETROUS APEX. ARCH. OTOLARYNG. I3:386.(MARCH). 1931.
I9. GRAY HENRY,: ANATOMY OF THE HUMAN BODY. TWENTY SECOND EDITION; LEA AND FEBIGER. NEW YORK.

20. TAYLOR, H. K., SUPPURATION OF THE PETROSAL PYRAMID., ROENTGENOLOGIC PROBLEMS., ARCH. OTOLARYNG. 18:458 (OCTOBER) 1930.

21. PERKINS, C. E.,: SIXTH NERVE PARALYSIS AND OTITIS MEDIA PURULENTA., ANNALS OTOL. RHIN., and LARYNG. 19:692. 1910. 22. Coates, G. M.; SUPPURATION OF THE PETROSAL PYRAMID, ROENTGENOLOGIC PROBLEMS., ARCH. OTOLARYNG. 18:458 (OCTOBER) 1934.

23. COATES, G. M.; ERSNER, M., and MYERS, D.; ROENTGEN CHANGES IN THE PETROUS PORTION OF THE TEMPORAL BONE . ARCH. OTOLARYNG. 20:615 (NAVEMBER) 1934.

24. WHITTMACK, K.: UBER DIE NORMALE UND PATHOLOGESCHE PNEUMAT-IZATION DES SCHAFENBEINS IHRER BEZIEHUNGER ZER DEN. MILLELOHREN KRANKENGEN. JENA, 1918. 25. FRIESNER, I,; DRUSS, J. G., ROSENWASSWE, H.: and ROSEN, S.; SUPPURATION OF THE PETROUS PYRAMID. ARCH. OTOLARYNG. 22:6 (DECEMBER) 1935.

26. PERKINS, C. F.; GRADENIGO SYNDROME, ANNALS OTOLARYNG. 35:348, 1926.

27. KOPETZKY, S., and ALMOUR, R.; SUPPURATION OF THE PETROUS PYRAMID, ANNALS OTOLARYNG. 39:996. 1930.

28. KOPETZKY, S. J., SYMPTOMATOLOGY AND DIAGNOSIS OF SUPPURATION OF THE PETROUS TIP. LARYNGOSCOPE, JUNE 1931, VOL. 41. 29. KOPETZKY, S. J.: PROBLEMS CONNECTED WITH EMPYEMA OF THE

PETROUS TIP. ARCH. OTOLARYNG. JULY 1933.

30. LILLIE, H. I.; SUPPURATION OF THE PETROUS PYRAMID, REPORT OF TWO CASES, ARCH. OTOLARYNG, MAY 1931.

31. DRUSS, JOSEPH G.; PATHOLOGY OF PETROSITIS, LARYNGOSCOPE, VOL. 41., 1931.

32. VOGEL, H., DER SOGENANNTE GRADENIGO SYMPTOMENKOMPLEX. INTERNAT. CENTRALBL. OHRENHEILK., VOL. 18, pp. 293, 1920 and 1921 and VOL. 19, 1922.

33. GRADENIGO. UBER DIE PARALYSE DES N. ABDUCESN BEI OTITIS. ARCH.F. OHRENHEILK.; VOL. 74. 1907.

34. VAIL, H. H.; ANATOMICAL STUDIES OF DORELLO'S CANAL. LARYNGOSCOPE, VOL. 32, 1922.

35. WHEELER, J. M., PARALYSIS OF THE SIXTH CRANIAL NERVE ASSOCIATED WITH OTITIS MEDIA. TRANS. SEC. OPHTH. A. M. A. 1918.

36. LANGE, W., ZUR PATHOLOGIE TIEFGELENER EPIDURALER ABSCESS LABY-UNTHENLZUNDUNG. BEITR. SUR ANAT. PHYSIOL., PATH., etc., DES OHRES etc., VOL. 2, 1929. 37. WILKINSON, G. B.; A CASE OF PARALYSIS OF THE EXTERNAL RECTUS MUSCLE DUE TO THE PRESENCE OF AN ABSCESS IN THE PETROUS APEX. DEATH FROM BASAL MENINGITIS. J. LARYNGOL. AND OTOL. AND RHIN. VOL. 24. 1914.

38. ALMOUR, R.; EMPYEMA OF THE PETROUS APEX.: OPERATION, RE-COVERY. LARYNGOSCOPE VOL. 41, 1914.

39. MOURET, J.; NOUVELLE RECHERCHES SUR LES CELLULES PETRUSES,
REV. HEBD. DE LARYNGOL. 25. 753. (DECEMBER) 1934.
40. TURNER AND REYNOLDS; NASAL MUCOUS POLYPS., INTRANASAL
OPERATION ON THE ETHMOIDAL AIR CELLS., PURULENT MENINGITIS.
JOUR. LARYNGOL. AND OTOL. VOL. 41. 1926.
41. PHILLIPS, W. C.; DISEASES OF THE EAR, NOSE AND THROAT.
FIRST EDITION., F. A. DAVIS, PHILADELPHIA. 1911.
42. EAGLETON. W. P.: CAVERNOUS SINUR THROMBOPHLEBITIS. NEW
YORK. MACMILLIAN AND COMPANY. 1926. pp. 153.