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Ludwig's angina

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Foreword

Practical minded readers might doubt the advisability of the selection of such a condition as Ludwig's angina as a suitable topic for a senior thesis. Two points influenced the author in the selection of this topic: first, he felt that a simple review of such a disease would form a satisfactory groundwork for the study of other more frequent and complex affections of the neck; secondly, he found that literature concerning this study was readily available for consideration because this clinical entity has been recognized only during the past century.

This thesis can not, in any sense, be said to be all inclusive. However, an honest attempt has been made to present the known facts, as well as the authoritative opinion regarding essential points over which there is a divergence of opinion.

The author wishes to extend his most sincere thanks to Dr. N. Fredrick Hicken for his willing aid and constructive criticism in the writing of this thesis.

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LUDWIG'S ANGINA

Chapter I

Definition and History

Definition and History.

The clinical condition which we now term Ludwig's Angina, has been described from earliest times by medical teachers, students, and writers. The name "Ludwig's Angina" was first applied by Cramer of Langenau, in the year 1839. Cramer of Langenau was a contemporary of Ludwig's and he felt that Ludwig's leadership in this field was so great that the disease should be named for him.

Ludwig was the first author to define the condition as a distinct clinical entity. Because he was one of the outstanding recognized authorities, and because of his works in medicine, the term "Ludwig's Angina" has remained as the non-descriptive definition since the time of origin. (The definition of angina as given by Ludwig will appear later in this chapter.)

It appears that the study of angina is better understood when taken in the chronological order. H. S. Muckleston (58) has made a complete review of the history and observations on this disease and from him the following historical facts have been gleaned.

"The outstanding symptom is suffocation or threatened suffocation; hence are derived the names applied

to this class of disease by the Greek and Latin writers, Cynache and Angina, respectively.

"The etymology of the former word is interesting. It has two roots of origin in Greek, the translations of which are 'dog' and 'choking'. The first root has a parallel in the modern colloquialism 'dogquinsy'.

"It may be noted here that the word 'cynache' was taken over bodily from the Greek by the Latin writers for use in treatises, and later passes into the Old French as Squinancie, and thence into the English as squinancy, a form now obsolete, and finally reaches us as quinsy.

"The Latin term in everyday use was 'Angina', which is itself of Greek origin. The comedian Plauths put a fretful 'aside' into the mouth of one of the characters of the Mosterlarial--'I'd like to be turned into an angina, that I might squeeze that she-devil by the throat'.

"Hippocrates of Cos, a physician of the fourth century before the Christian era, makes many references to cynache. 'The Prognostics' (Section 23) tells of patients with swelling both within and without the throat, of acute affections of the throat in association

with erysipelas, of swelling and redness, determined in some cases outwardly, and others of the lungs with serious prognosis. In the 'Regiem in Acute Diseases' (Appendix II, Section VI) there is a vivid description of the tongue, which states that it 'changes from broad and becomes round, its natural color becomes livid, from a soft consistence it grows hard, instead of being flexible it becomes unflexible, so that the patient would soon become suffocated unless speedily relieved.'

"In 'The Epidemics'(Book III) there is a case report which relates the following instance: 'The woman affected with cynache (translated, "quinsy"), who lodged in the house of Aristion: her complaint being in the tongue; speech inarticulate; tongue reddened and parched. On the first day, felt chilly, and afterwards became heated. On the third day a rigor; acute fever; a reddish and hard swelling on both sides of the neck and chest; extremities cold and livid; respiration elevated; the drink returned by nose; she could not swallow; alvine and urinary discharges surpressed. On the fourth, all the symptoms were exacerbated. On the sixth day she died of cynache'.

"Also in 'The Aphorisms'(Section VI, 37) we read

that 'it is a good symptom when swelling on the outside of the neck seizes a person ill of cynache(translated, "quinsy"), for the disease is turned outwardly'.

"Aretaeus, a distinguished contemporary of Galen, living in the second and third centuries in our era, made a distinction between cynache and synache; in the former the parts being swollen and beset by other symptoms, and in the latter the parts being contracted with suffocation impending. There is little room for question that the latter group were victims of epidemic diphtheria.

"Galen described, in his 'Methodus Medendi' (Book I), a rare species of cynache, in which the tongue is so swelled that the mouth cannot contain it.

"This age was strongly theraputic. Galen and Aretaeus, Aetieus in the sixth century, and Paulus of Aegina one hundred years later, give a multitude of remedies to be used in treating disease of the throat, of which the following are the more common: '--and dog's dung dried and powdered, and rubbed in with honey, is an excellent application, more especially the white kind; also the dung of wild swallows, in a like manner . . . Cupping instruments, or leeches are to be

applied to the chin and neck and the patients must use the stronger gargles from iris, hyssop, gith, southern wood, licorice, dried figs, boiled in honied water, or in the juice of rue with milk, or mustard, with orymel'. (Paulus Aegineta).

"Sinapisms, hot fomentations, purgation, and other means of relief form a long list; venesection, both by sublingyal and jugular veins is recommended, but laryngotomy, though counseled by an earlier writer, Archigines, is by these authorities spoken of only to be condemned.

(The translation of the works of Hippocrates and of Paulus Aegineta, of which Muckleston availed himself, are those of a Scotch physician, Francis Adams, and were published in 1844 and 1845, the expense of the publication being borne, at least in part, by the Council of the Sydenham Society.)

"A layman's brief account of the death in April, 1585, of Pope Gregory XIII, the promulgator of the Gregorian calendar, is here quoted because of the typical sequence of illness. 'The head of the great Augsburg bankers, the House of Fugger, received through "Antwerp Courier" from their Venice agent the following

item of news: "We would inform you that His Holiness the Pope died in the Lord, on the 10'th day of this month at 12 o'clock. May the Almighty be merciful to his soul. Within the space of ten hours he was first in good health, then ill, then dead, suffocated by a catarrh. Farnese showed no elation and all those who were sick abed were made to rise through this shock.."'

"The classical division of diseases by genus and species held its own in the eighteenth century. William Cullen, prominent among the Scotch clinicians, taught that cynache was a genus with five species: the tonsillaris, the maligna, the trachealis, the pharyngea, and the parotidea. Its characteristics were 'Pyrexi ar aliquando Typhoides; rubor et dolor faucium; deglutitio et spiratio difficiles, cum angustiae in faucibus sensu', which may be translated thus: fevers, at times with a likeness to Typhus, reddening and pain in the throat, difficulty in swallowing and breathing, along with a feeling of tightness in the throat. Dr. James Stratton, President of the New Jersey Medical Society in 1789, delivered a dissertation on this subject, embodying in it the teachings of Cullen. Cynache tonsillaris included simple tonsillitis and quinsy, and

also more severe infections with extension to the trachea and suffocation. Cynache maligna, covered faucal and laryngeal dyphtheria, and the mixed infections of scarlet fever and diphtheria. Cynache trachealis meant croup and laryngeal diphtheria. Cynache pharyngeoparotidea was decidedly mumps, even the metastatic involvment of the ovary and testicle being mentioned. However, the distinctions seem to have not always been clear cut, there having been some overlapping in the tonsillar and tracheal species.

"Ten years after the delivery of Dr. Stratton's address, the perils of cynache were brought forcibly to the attention of this country by the illness and death of General Washington.

"Craik and Dick give a short account of the death of General Washington, which resulted from an attack of Cynache Trachealis. The disease commenced with a violent ague, accompanied with some pain in the upper and foremost part of the throat, a sense of stricture in the same part, a cough and a difficult, rather than a painful,deglutition, which were soon succeeded by fever and a quick laborious respiration. He was bled, some twelve or fourteen ounces of blood being taken.

Consulting physicians were called in, and frequent bleedings followed, until he had lost some ninety ounces of blood. Vapors of vinegar and water were inhaled, ten grains of calomel were given, succeeded by repeated doses of tarter emetic. Blisters were applied to the extremities, and a cataplasm of bran and vinegar to the throat. Speaking became painful and finally impossible; and without a struggle he died in twenty-four hours after being attacked.

"J. Reid, a London physician, in reviewing the above account concluded that the General was bled and doctored to death, as none but the strongest constitution could survive such vigorous treatment.

"A compilation of records of epidemics, in this country, of sore throat and of diseases of the throat in general, from the early part of the seventeenth century and up to the introduction of laryngoscopy, is found in the proceedings of the First Annual Meeting of the American Laryng ological Association, New York, 1879. The first president was Dr. Louis Elsberg of New York, and this exhaustive narrative is a monument to his labors." (From this source Muckleston took the address of Dr. James Stratton and the account of the

last illness of General Washington.)

Dr. Kirkland in his "Enquiry into the Present State of Medical Surgery"(Volume II, Page 159, 1783) describes a condition which he calls Angina Externa, which is quite comparable to the cases reported by Ludwig.

George Gregory in "Case of Cynache Cellularis with Remarks"(The London Medical and Physical Journal, Volume 48, Page 287, 1822) makes a number of observations relative to a condition which he believed to be closely comparable to Kirkland's description of Angina Externa. He says,"It has never occurred to myself to witness anything at all similar to it, either before or since, nor have I been able to ascertain that in the practice of my professional friends, one analagous case has ever presented itself. The disease consisted in an extensive inflammation of the cellular membrane of the neck and anterior mediastinum, of a highly malignant character. Its course was rapid, and the symptoms which attended it were of unusual severity. It bore in the first instance, the appearance of a rheumatic affection of the joints of the cervical vertebrae. At a somewhat later period of the disease, it was imagined that the thyroid

gland was the immediate seat of the inflammation, but it was not until after death that the exact nature of the case was understood. Books have afforded us but very scanty information concerning this affection."

Gregory's patient, a twenty-five year old white housewife,died seven days after the onset of cynache. There appeared to be a terminal tracheal obstruction; and at the end of the disease the patient complained of pain at the top of the sternum.

With reference to this case Gregory states: "The cellular membrane beneath the skin of the throat and around the trachea, as well as that which connects the pharynx and palate to the surrounding bones was everywhere in a state of disease--doubtless the result of inflammatory action. In some places, actual sphacelus had occurred; in others, it was in a state of what might be called imperfect suppuration. In one or two points purulent material could be directly traced. The same disorganized condition of the cellular membrane pervaded the whole extent of the anterior mediastinum, even as low as the point of the ensiform cartilage.

"While such was the state of the cellular membrane of the throat, the mucous expansions of the palate,

pharynx, oesophagus, and trachea were healthy except in so far as they were covered with a preternaturally abundant secretion of mucus. The lungs and the different abdominal viscera were free from any trace of disease.

"To this singular variety of quinsy I have ventured to apply the term Cynache Cellularis, from the belief that it has not yet received a more appropriate appellation."

In 1836 and 1837 there appeared a series of articles in the Wuerttemberger Korrespondenzblatt für Aertze, the subject of which was brawny infection of the cellular tissues of the neck. Various names were given the disease by the authors, such as,"Brandige Zellgewebsverhartening am Halse", "Cynache Cellularis Maligna", "Morbus Strangularotius".

The leader of the group and the author of the first article at this time was W ilhelm Friedrich von Ludwig of Stuttgart, Leibartz(personal physician) to the King of Wuerttemberg. Ludwig was born in 1790, and died in 1865. His leadership was recognized by his contemporary, Cramer of Langenau, for this writer first called the disease after Ludwig, as has been pointed out by Thomas.

Ludwig's own description is such an inclusive, concise, and characteristic account that it will serve well for a definition of the disease. It is as follows:

"A sore throat of a rheumatic or erysipelatous nature, precedes as a rule the first symptoms, which are: slight rise in temperature, with a number of chills, headache, weakness, some loss of appetite, slightly coated tongue, some difficulty in swallowing, which is slight at the early stage, and in some cases scarcely noticeable; then a hard swelling develops, either on both sides of the throat or more commonly on one side usually in the cellular tissues of the submaxillary glands, less frequently in the tissues that surround the parotid and sublingual glands. This hard growth extends, with some tissue changes, gradually around the throat, and then under the jaw to the chin, and down over the larynx, and backwards into the cellular tissues of the parotid, also causing a marked swelling on the outside. This growth extends through the cellular tissues that cover the musculature between the larynx and the oral cavity, and even these muscles themselves are sometimes apparently involved in this hardening process; the tongue, deep red in color, rests

on the hardened mass thus formed, which is felt as a hard indurated ring on the inner circumference of the larynx. The ability to open the mouth is much limited, and every attempt is painful; the tongue is pressed upward and somewhat backward; the speech is made difficult, and partly because of the pressure on the larynx, and partly because of the morbid changes in the smaller throat muscles, it is rough and not clear, gurling. Swallowing becomes more difficult with the straining of all the throat muscles, due undoubtedly,chiefly, if not entirely,to the mechanical pressure of the growth, because an inflammatory swelling of the mucous membrane of the pharynx is present either in the beginning of the disease, or later if murcurial drugs are used in the treatment. Yet with the progress of the disease a considerable amount of mucus gathers in the throat, which is expectorated with difficulty. The skin over the growth appears normal, not reddened, at least in the earlier stages, although stretched according to the size of the growth--evidence that its cellular layers are not involved in the hardening process. During the first four to six days of development of this local growth, the constitutional symptoms are not marked,

the fever is usually very moderate, the strength and general feelings are little altered, appetite and abil-- ity to sleep are not lost entirely, there is some thirst, secretions and excretions are fairly normal. As the disease progresses some areas of redness of the skin are noted, inside the mouth frequent areas of inflam- matory lymph appear (if this has not occurred before), the growth under the tongue seems softer as if the serum under the mucous membrane was effused and partly coagu- lated. On the outside certain areas become softer, sinking in somewhat, and if pressed with the finger feel as if there were air under the skin, or sometimes they become more prominent and fluctuating, as if pus was going to break through, but this does not occur, and the swelling diminishes--or, sometimes at the beginning, and sometimes later in the course of the local process some spots in the oral cavity, either at the back or sides, or at the root of the tongue, or more to the front of the under side of the inferior maxilla, break out and discharge a thin greyish or reddish brown fluid, with bad odor, which becomes more and more like the ichor of a necrotic process. With the beginning of this process, which is of the

nature of real mortification, the general symptoms become marked. The fever is higher, with exacerbations usually in the morning, the sleep is broken with heavy sweats, frightful dreams, somnambulism, there is a marked increase in the sediment in the urine, sometimes slight delerium. Notwithstanding the fact that the growth may diminish in size, swallowing is still very difficult, there are periods of anxiety with fairly free intervals, a non-mechanical, but apparently nervous interference with respiration. The symptoms now develop rapidly, simulating the course of a putrid-typhous process, and in four or five days, the tenth or twelfth from the beginning of the disease, coma and death result with evidence of edema of the lungs. There are variations in the symptoms noted, especially in the development of fever, the time of the development of the disease and the severity of the process, which I will not discuss in detail, as my purpose is to give a general picture of the disease that will aid in its recognition at the bedside." (Translation taken from an address of C. G. Coakley--Muckleston.)

A precise definition of Ludwig's angina is quite

difficult even after this description, but five points are outstanding: (1) A slight inflammation of the throat itself which disappears after a day or two, and when present must be considered as of secondary importance. (2) The peculiar woody character of the connective tissue which does not pit on pressure. (3) A hard swelling under the tongue, with a hard swelling around the inner border of the lower jaw, with a deep red, or cyanotic-like flush. (4) A uniform spread of induration in such a way that it is sharply bounded by a zone of entirely unaffected cellular tissue. (5) Escape of glands, although the disease attacks the cellular tissue surrounding them and may even commence near the gland--a point not fully agreed on by all observers.

There has been much argument as to whether or not Ludwig's angina is to be considered a distinct clinical entity. At the close of the nineteenth century there was an almost even division of opinion on this point. Parker points out that Nelation was one of the leaders of the school which held that Ludwig's angina was not recognizable as a separate disease and had the French Surgical Society in 1892 pass a resolution to

this effect. Delorme, on the other hand, held that Ludwig's angina was a clear cut entity and had the resolution revoked at the meeting on the following year. In 1895, Felix Semon of St. Thomas' Hospital in London, in a paper before the Medico-Chirurgical Society, claimed that the various affections hitherto described as acute oedema of the larynx, oedematous laryngitis, exysipelas of the pharynx and larynx, and phlegmon of the pharynx and larynx and Angina Ludovici, were simply forms of acute septic inflammation of the throat and neck, and were pathologically identical; also that they were merely a representation of varied degrees of virulence of one and the same process; the question of their primary location and subsequent development depends in all probability upon the accidental breaches in the protecting surface through which the pathogenic micro-organisms find entrance; and that it is absolutely impossible to draw at any point a definite line of demarcation between the purely local and the more complicated, or between the edematous, and the suppurative forms. His views were more or less accepted until 1905-1909, at which time G. G. Davis and T. T. Thomas reported their studies on Ludwig's Angina.

Even at the present time the matter is not closed, as shown by the comment concerning Ludwig's angina made by Foote and Livingston in 1930: "It is not, however, a distinct disease, and the symptoms of the patients who are considered to have it, vary according to the severity of the infection, and still more in accordance with the part of the neck which is the seat of the inflammation. Like all other suppurations, they tend to develop more or less rapidly along the lines of least resistance; while early and free drainage aids the body in limiting and overcoming the infection located here, just as it does in infection in every other part of the body."

The relative infrequency with which typical symptoms of Ludwig's angina are encountered, particularly by the general practioner, has been conducive to indifference and obscurity concerning the various pathological phases of the process, and any variation which might be encountered.

Van Wagnen and Costello summarize the present status of Ludwig's angina quite well by saying: "There is a tendency for Ludwig's angina to be a catch all for any of the infections of the teeth, mouth, neck, pharynx, and larynx; The reason for this is because

even though not true Ludwig's angina, they have one cardinal symptom like Ludwig's angina, that is: Sublingual phlegmon."

LUDWIG'S ANGINA

Chapter II

Etiology

Etiology

The most commonly accepted agent in Ludwig's angina is dental infection. Moty gives a very complete dissertation on such infection as an etiological agent which may well be quoted. "The lesions involved in Ludwig's angina are as follows: (1) Caries may cause gangrene of the dental pulp, the infection spreading from there to the apex of the tooth and then to the alveolus, there causing fungosities with absorption of the cement of the tooth and the corresponding wall of the alveolus. The pus discharges first by the canal inside the tooth, but if the canal is obstructed, or is insufficient in size a phlegmon develops. (2) Usually the pus pierces the alveolus at its thinnest part, on the outer surface of the mandible and forms a dento-alveolar abcess in the surrounding cellular tissue. The pus seldom works its way between the alveolus and the neck of the tooth because the latter is united to the gingival mucosa by resistant fascia; so it points toward the skin or toward places in the buccal mucosa more or less distant from the diseased tooth. (3) When the process is acute (as is seen in ill timed filling of a tooth), there is no time for

the absorption of the alveolus to occur; so the infection enters the dental canal of the mandible, spreads toward the spine of Spix and makes its way toward the cellular tissue which separates the upper surface of the myelohyoid from the gum, or toward that which surrounds the carotid vessels in the neck. The strangulation resulting from the inflammation of the dental canal may cause osteomyelitis of the mandible. (4) If a back tooth or wisdom tooth is infected in this way, the outer wall of the mandible is so thick that the perforation occurs only on its inner wall so that the sublingual tissues are infected at once."

Ivy considers Ludwig's angina to be a fulminating type of dento-alveolar abcess. He says: "Occasionally, the inner or lingual plate of the mandible is perforated by a dento-alveolar abcess, in which case a tender edematous swelling appears in the floor of the mouth. When spreading rapidly, with little tendency to localization, extending down through the fascial planes toward the pharynx and epiglottis, the condition is termed Ludwig's angina. Ludwig's angina is not exclusively a sequel of dental infections, although that is the most common cause. It may follow tonsillitis and inflammatory lesions of the soft tissues of the region."

Haman, Ashurst and Bassingame recognize other infections as being etiologic factors in Ludwig's angina at times, but hold that dental and peridental infections are the most frequent cause of extensive submaxillary suppurations.

Gingrass believes ill advised removal of teeth is significant for he points out that the majority of cases of Ludwig's angina follows the extraction of teeth. He also suggests that low resistance on the part of the individual coupled with a particularly virulent infective organism determines, in many cases, whether or not the individual becomes acutely infected.

Second in the list of etiological factors is tonsillar infection, or acute tonsillitis. Casselberry reports a case of Ludwig's angina which originated from a primary tonsillar infection. Hochbaum says that Ludwig's angina may follow acute tonsillitis, of peritonsillar infection. Balinger states: "The lingual tonsil and some of the adjacent lymphatic structures appear to me to be the important etiological factors in Ludwig's angina".

Aldrich cites a case in which there was a combination of introduced infection, and trauma of the frenum of the tongue, due to severence of this membranous fold

with a dirty brass safety pin. This was done by an ignorant midwife because she believed that the baby for whom she was caring was tongue tied.

Lederman reports a case of Ludwig's angina beginning two days after the onset of acute otitis. It was his opinion that the otitis was an obvious primary infection from which the Ludwig's angina developed.

H. Bailey believed the infection in Ludwig's angina to be primary in the region of the submaxillary gland. It was his opinion that the process might be either cellulitis, or lymphangitis. In listing the origin of infection he says:"1.Abcesses connected with the last lower molar infect the periglandular space, because alveolar abcesses commonly point on the labial side of the mandible for the outer alveolus is thinner. In the region of the wisdom tooth the inner alveolus is weaker, consequently if an abcess connected with the last molar breaks it is more liable to burst through the inner surface of the jaw. 2. Infections may arise from an inflammed submaxillary gland. Infection by this route is infrequent, and some do not believe it occurs. In two out of five patients seen,there was stone in Wharton's duct. 3. Infection from lymphangitis: the

main lymphatic glands of the submaxillary triangle are superficial to the fascial space in question, doubtless infection can be conveyed thither by the abundant lymphatic supply around the submaxillary gland, and a boil on the chin thus act as a common primary focus".

T. Turner Thomas is not so definite concerning the etiology of Ludwig's angina. He points out that: "--the etiology and pathology of Ludwig's angina are still obscure because there is much obscurity associated with the cause of death in which the chief question is as to whether it results from septic intoxication, or from invasion of the air passages. Probably both conditions exist to a degree in typical cases; but the relative importance of each has never been established.

"The primary focus of infection in the majority of cases is some very minor lesion in the mouth, as a carious tooth, an herpetic, or other ulcer, or tonsillitis. However, in some cases no lesion can be demonstrated."

Semon was of the opinion that some small abrasion on the side of the neck exposed to the action of pathoginic organisms, which subsequently invaded the body from the outside, caused Angina Ludovici. He says:

"The original focus is purely accidental."

White is of the opinion that metastatic infection is of significance in the origin of Ludwig's angina, but this has never been clearly demonstrated.

Ferguson, in 1888 stained organisms from a case of Ludwig's angina and noted: "--large colonies of micrococci. They were the same as those ordinarily found in erysipelas."

There have been numerous studies made relative to the bacteriology in Ludwig's angina, but these have been somewhat inconclusive. Coakly suggests that the etiological agent may be some form of filterable virus. Bailey reports that he has cultured Streptococci in 70 percent of his cases of Ludwig's angina. Gingrass states that the most of his cases were infected with Streptococci. Blassingame,too, finds that Streptococci were the preponderant organisms found.In the remainder of his cases Staphylocci were cultured. Stetten reported a case of Ludwig's angina originating from a boil on the chin, and,-in this particular case, the organism cultured was Staphyloccus albus.

Mixed infections usually involving Streptococci and some other group of bacteria are most frequently

reported. Bacillus coli, Diplococci, diptheroids, and Bacillus septicus have been found.

G. G. Davis in reporting the bacteriology in twelve of his own cases noted five cases of pure Streptococci, seven cases of mixed Streptococcic organisms with pneumococci, with staphylococci, or with some unidentified organisms.

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Chapter III

Smyptomatology

Smyptomatology

Symtomatology in Ludwig's angina is not remarkably varied. Typical symptoms include: pain in the floor of the mouth, stiffness of movement of the tongue, pain in efforts to clear the throat, and pain in salivation. Fever is not ordinarily a constant finding. A point of tenderness may be found in the tissues below the jaw, usually about midway between symphysis menti and the angle of the jaw. In the submaxillary tissues, swelling appears early and becomes board-like in hardness, and does not pit in pressure.

An increase of the swelling in the floor of the mouth displaces the tongue upward and backwards, sometimes later in a forward direction, protruding the organ between the teeth. Coughing or swallowing causes severe pain. Breathing is interfered with by the displacement of the tongue and narrowing of the pharyngeal space. The degree of dyspnéa varies in different patients and from day to day in the same patient.

The constitutional symptoms are those of toxemia. Fever may be intermittent.

The localization of the infection in the formation of an abcess is to be welcomed. Evacuation of the pus

is often immediately followed by the relief of urgent dyspnea. Salivary drooling consequent to the inflammation in and about the salivary glands, and upon the extreme pain attendant upon effects at its expulsion, is ineventable and most distressing.

There may be periods of delerium in the more toxic patients. There is an utter inability to rest, pain in moving the head and neck, and ineffectual attempts to clear the throat making rest impossible without opiates.

A gradual decline in resistance occurs and death follows from respiratory obstruction, toxemia, streptococcic pneumonia, and in some cases mediastinitis.

Newcombe points out that the local symptoms of Ludwig's angina include: "1. A wood-like induration of the affected region, sharply defined from the surrounding normal tissue. 2. The thrusting forward and upward of the tongue by the accumulated inflammatory products, so that the tongue approaches the palital vault. 3. Severe dyspnea, with attendant danger of laryngeal edema. 4. The presence of a hard pad, or button-like swelling at the internal aspect of the dental arcade."

Furstenberg points out that the local inflammation makes the tongue broad, red, and swollen, so that it is relatively immobile as well as being pressed to the hard palate. Congestion and edema of the mucous membrane covering the floor of the mouth is characteristic and this may allow the imprint of the teeth in the edematous tissues, as they are pressed against them.

The external symptoms are those of a more or less marked inflammatory swelling below the symphysis of the jaw, although fluctuation is very slight. The external swelling may be extremely tender on pressure and show typical signs of inflammation, although pus is not often felt in this region. The speech is gutteral; saliva drools from the mouth, swallowing becomes difficult and later the upward displacement of the tongue gives respiratory embarrassment.

When the infection has broken down its barriers and migrated to the soft tissues of the neck an extensive phlegmon may arise and circumscribe the entire cervical region.

With the above signs, Furstenberg also points out that fever, chills, and general symptoms of infection are to be noted particularly if a septicemia is asso-

ciated with the primary Ludwig's infection.

Lederman, describes the symptoms noted in one of his cases. The symptoms are so typical that it may be well to repeat them here. "My patients expression was one of great suffering, intensified by the appearance of the tongue, which was pushed out over the edge of the lower teeth, on account of the marked swelling of the sublingual tissues. The flow of saliva was constant and nothing could be swallowed as the movement of the tongue was restricted. After repeated attempts, a pharyngeal and laryngeal picture was obtained, but no edema of these parts could be seen. Breathing was not materially effected. Though the tongue was of normal size, it appeared larger, owing to its protrusion. No distinct fluctuation could be felt in the swelling along the floor of the mouth. At a point near the fraenum on the left side, the mucous membrane appeared to be changing color."

Such symptoms do not vary remarkably from the classical description of Ludwig given earlier. They do point out the apparent urgency of the treatment, and do give an indication of the speed with which the process develops.

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Chapter IV

Differential Diagnosis

Differential Diagnosis

The differential diagnosis of Ludwig's angina is comparatively difficult, in its inception, from other acute inflammatory processes in the mouth and neck region.

Statistics are of little value in dealing with the condition, for septic infections in the neck are dangerous, according to their extent, and the infection may by of any grade or degree. The mortality varies in direct proportion with the seriousness of the infection.

In diagnosis of inflammatory and oedematous affections of the throat and neck, Davis differentiates two tpyes of processes: in one type of case the affection is local in character and usually remains local, for this type of infection does not show the tendency to spread as seen in the other type; the other type may arise from mechanical and chemical irritants, from interference with the blood supply producing oedema, from surface inflammations as glossitis, stomatitis, pharyngitis, laryngitis, from inflammation of the neighboring organs as the tonsil, salivary and lymphatic glands, syphiletic and tuberculous ulcerations, and may all be confounded with acute septic

infection.

In some cases it is impossible to draw the distinguishing line, particularly in the early stages; yet, it is essential that the true character be recognized as soon as possible because one class follows a relatively benign course while the other persues a decidedly dangerous one. Yerger feels that Ludwig's angina should not be diagnosed unless there is present a sublingual phlegmon in addition to the submaxillary phlegmon, and that the cases in which the phlegmonous processes begins in the throat in the immediate vicinity of the larynx, should be excluded.

Yerger is also of the opinion that submaxillary cellulitis is the only condition that should be confused with Ludwig's angina, because in both instances a submaxillary cellulitis is present. However, in Ludwig's angina there is also a sublingual cellulitis present, and for this reason diagnosis of Ludwig's angina does not always prove to be easy.

Acute submaxillary lymphadenitis is nearly always due to ulcerations of the oral soft tissues--the gums, vestibule, and the floor of the mouth and tongue. In

tonsillitis and inflammations around the fauces, the lymph nodes beneath the angle of the jaw may be involved. Acute swellings and suppurations of these nodes may also be caused by infection of the scalp arising from head lice, but lymphatic infections of this type are almost never accompanied by trismus, and ordinarily are not as acute and fulminating as Ludwig's angina.

Calculus of Wharton's duct may give rise to acute, painful enlargement of the submaxillary salivery gland. In most of these cases there is a painful edematous swelling under the tongue, difficulty in deglutition, but the history usually is that of recurrent attacks, with increase of pain and swelling, especially during meals.

In osteomyelitis of the jaw there is no limited focus of inflammation; the bone is affected in its entirety; the inflammatory process is more generalized; and the subhyoid region is rarely involved.

Simple adeno-phlegmon of the submaxillary gland is more superficial; the submaxillary gland and its envelope are more accessible; there is no wooden hardness; and incision of the superficial fascia will give exit to pus, and the inflammation is localized at the

outset behind the internal face of the maxilla. None of these symptoms occur in Ludwig's angina.

Newcomb mentions a rare condition, the so-called Fleischman's "hygroma", which must be differentiated from Ludwig's angina. Hygroma is sudden in onset, without local evidence of inflammation in the median line and is devoid of constitutional symptoms.

LUDWIG'S ANGINA

Chapter V

Anatomy

Anatomy

The anatomy of the neck has been studied, for the most part, by two techniques, with, of course, some individual variations. These methods are: dissection studies, as made by the earlier anatomists, and injection of the fascial spaces with media to delimit the region injected with an easily recognizable material.

Thomas, in his studies, used the first of these techniques. He made the first dissections on the mouth and neck region with the view of showing the anatomic pathology in Ludwig's angina. His observations and findings have been largely borne out by later anatomists, including Barnhill, Colp, and Berryhill.

The early work of Thomas may well be accepted as giving the most satisfactory general description of the anatomic area involved. His observations in this region give special emphasis to the relationship of the submaxillary salivary gland to the floor of the mouth, and it is this original observation which makes his study outstanding. His description reads: "The muscular floor of the mouth is formed by the two mylohyoid muscles which fuse with each other at the anterior median raphe. This muscular diaphragm separates the mouth from the

neck, is complete from the posterior edge of one mylohyoid muscle to that of the other, and is comparatively strong. There are no openings in it for the passage of planes of connective tissue between the mouth and neck. From the posterior border of the mylohyoid on each side extends backward the constrictor muscles of the pharynx, separating the pharynx from the neck, the muscles on the two sides fusing at the posterior median raphe. The three constrictiors of the pharynx, superior, middle and inferior, overlap each other, so that here also the submucous tissue of the pharynx is not continuous with the connective tissue of the neck through these muscles. Between the posterior edge of the mylohyoid, and the anterior border of the middle constrictor, however, there is a considerable deficiency in the buccopharyngeal muscular wall. This opening extends from the hyoid bone upward and backward to the inner side of the lower jaw near its angle. The hyoglossus muscle, which viewed externally, forms a part of the submaxillary triangle, does not enter into the formation of the floor of the mouth or pharyngeal wall. It passes upward through this muscular opening, or gap, to become a part of the root of the tongue, and fills the gap considerably. Those

structures which pass from the neck to the mouth, or in the opposite direction, do so through this opening. The structures include the glossopharyngeal and hypoglossal nerves, the lingual artery, vein, and nerve, and the styloglossus muscle. The greater part of the opening, however, is occupied by the deeper portions of the submaxillary salivary gland where it projects into the floor of the mouth, near the root of the tongue lying just under the mucous menbrane. The gland may, therefore, be said to form a small part of the floor of the mouth. The submaxillary gland within the mouth is adjacent to the posterior part of the sublingual gland and is attached to it by the surrounding connective tissue. We thus see that the connective tissue in the submaxillary fossa is directly continuous with that of the floor of the mouth, so that the extension or a submaxillary cellulitis to the sublingual region which occurs so early and constantly in Ludwig's angina is readily understood."

If all cases of Ludwig's angina could be seen and treated early, Thomas' description would give all the anatomic knowledge required. However, late cases of Ludwig's angina show fascial space involvement; hence,

knowledge of these spaces is essential for recognition of the extent and site of the infection, the spread of which occurs after the process has broken through the barriers of the mouth, and is descending toward the root of the neck.

Barnhill has made additional studies which supplement those made by Thomas. He has described the lateral sublingual space in which the submucosal portion of the sublingual salivary gland lies.He calls attention to the thick fascial wall which separates it from the pharyngomaxillary space, and points out that the latter space is only involved if the lateral sublingual space is not drained early, or sufficiently well.

Barnhill is careful to point out that though the lateral sublingual space is the typical and characteristic site of its development, it is not the only possible site of development of Ludwig's angina. Abcesses do occur in the lateral sublingual space, but, at best, are migratory when confined in such loosely bounded connective tissue spaces. This is particularly true when pressure is developed in the space, as will occur in neglected cases with long standing abcess formation. There seems to be an almost invariable downward descent

of infection so that it tends to occupy the lower planes of the mouth.

Neglected infections may break through the confines of the lateral sublingual space into the pharyngomaxillary cavity. They may rupture through the fascial wall to sag beneath the chin where they are seen as hard phlegmonous mass between the symphysis menti and the hyoid bone. Finally the phlegmon may easily pass to the opposite side of the floor of the mouth since all the spaces in this region are truly potential rather than actual.

In considering the relationships of the region, Colp divides the submental triangle of the neck into three subdivisions, or spaces: the submaxillary, the sublingual, and the retromandibular spaces. The entire triangle is roofed off by the superficial fascia and the deep cervical fascia which contains the submaxillary salivary gland between its two leaves. He believes that of the submaxillary space communicates anteriorly with that of the sublingual space. In all probability, these two subdivisions constitute the lateral sublingual space (Barnhill). Posteriorly the submaxillary space is potentially separated from the retromandibular space, but, according to Colp, it is the route by which

Ludwig's infection reaches the deep spaces of the neck.

Berryhill has gone even farther than these aforementioned students of Ludwig's angina, in that he was among the first observers to consider the possibility of the spread of infection by continuity from the tissues of the submaxillary gland, along the fascial layers, or along the lingual and fascial arteries to the sheath of the carotid vessels.

Because of the similarity of an injected media to a burrowing suppurative process, Blassingame conceived the idea of injecting material into the fascial spaces which would mark out the probable course of such an infective process, if seen late or when improperly treated. In 1928 he first made injections of India ink in dogs to show the anatomic and lymphatic relations. His work was followed, in 1929 by that of Ashurst who injected India ink into the mouth of cadavers to outline the course of infection in the floor of the mouth. In 1936, Furstenberg made similar studies. His work was closely followed by Grodinsky and Holyoke, whose injection studies were made to demonstrate the normal anatomy of the fascial spaces of the head and neck and to show changes wrought by infective processes.

Of the injection studies, Furstenberg's is the most clear to the individual who wishes to know the anatomy of Ludwig's angina. He made clinical and anatomic studies of the fascial spaces of the neck and suggests that the clinical manifestations of the various neck infections depend upon the route of spread as well as the virulence of the primary process.

He describes the fascial relationships in two groups, each of which gives off innumerable connective tissue processes which unite to form a structural support for the tissues within the neck.

The first process appears to start at the summit of the neck and descends with the cervical muscles to become attached to the upper end of the thoracic cage. This group includes the fascia of the muscles, as well as the superficial layer of the deep cervical fascia. It also includes the fascia enveloping the omohyoid and the sternohyoid muscles at the deeper levels is the fascia which follows the plane of the sternothyroid and thyrohyoid muscles.

Furstenberg says that this group of fascial layers have been variously described as the anterior, or perivisceral compartments. They do not directly communicate

with any part of the mediastinum.

The other group of fascias is of greater interest in Ludwig's angina and other mouth infections, because they form definite pathways along which infections pass from the neck of the mediastinum. Furstenberg divides these fascias into two main systems along which infections pass:

First, the vasculovisceral fascial system, as the name implies, envelops the viscera of the neck and mediastinum, and surrounds the carotid sheath, the aorta (its arch and descending portion), and its branches. It forms a cylindrical covering for the pharynx, larynx and trachea, thyroid gland, and aorta with its branches which ascend into the neck. Within the thoracic cavity, the fascial coat blends with pericardial sac anteriorly, and behind, surrounds the aorta and extends laterally with the parietal pleura. At the level of the dome of the pleura this fascial layer is greatly strengthened to enclose the subclavian artery and from the suspensory ligament of the pleural dome.

Second, the prevertebral system, lies in front of the bodies of the cervical and dorsal vertebra from the basalar process of the occipital bone down to the lumbar

region where it blends with the external perymysium to the psoas muscle and the crura of the diaphragm. In the cervical region this fascia encloses the prevertebral muscles and on each side of the median line it is reinforced by the perymysium of the muscles. In the region of the posterior mediastium, below the third and fourth dorsal vertebra, the prevertebral fascia loses its reinforcement and becomes exceedingly thin. It is this fascial system which forms the posterior compartment of the neck. This space acts as a pathway for infections descending from the upper cervical regions to the mediastinum. Deep in the chest this fascia forms the interpleural ligament and the sheath of the descending aorta.

Furstenberg uses iodized oil to outline the retrovisceral space, which he says acts as a pathway for infections descending from the retrophayngeal region to the mediastinum. In the injections of oil there is no spread of oil to the adjacent parts of the neck, for it is confined to the retrovisceral space as it descends into the thoracic cavity.

Studies on the fascial spaces of the neck and mouth region have been correlated by Grodinsky and Holyoke.

In the main, they confirm studies made by earlier observers. They subdivide the vasculovisceral space (Furstenberg) into four spaces: "1", "2a", "2b," "3". The prevertebral system is classified by them as space "4". They agree, however, that the fascias surrounding the muscles of the mouth are only potential and do communicate. They also call attention to the fact that when the neck is involved by descending infection the spaces are involved with great rapidity, and pressure may cause rupture of spaces ordinarily separate from each other, and that infection may follow down the course of the great vessels by direct continuity.

LUDWIG'S ANGINA

Chapter VI

Pathology

Pathology

In any discussion of the pathological findings in Ludwig's angina it must be admitted at the outset that all points pertaining to the pathogenisis and pathology have not been solved; necropsy should be obtained in all fatal cases. Yerger observes that there are at least four points which are open to dispute. These are: (1). Is the pathological process solely a cellulitis?. (2). Is it primarily a lymphadenitis and secondarily a cellulitis?. (3). Is the cellulitis situated primarily in the submaxillary or in the sublingual region? (4). What is the cause of death?

The greatest divergence of opinion regarding the pathology seems to lie in the question of whether or not the primary pathological process is a cellulitis, and to what extent lymphatic involvement is a factor in the condition.

Van Wagnen and Costello, discuss the pathological process in Ludwig's angina as an aid in the differential diagnosis from sublingual phlegmon, which they consider a clinical entity. They point out that the primary origin of infection in Ludwig's angina is in

the mouth or in the sublingual area. Ordinarily, tonsil, teeth, and oral mucous membranes are the most frequent portals of entry. The structures next involved are not positively determined. Probably Thomas is correct in his view that cellulitis of the submaxillary region practically always precedes the sublingual phlegmon. Thomas has said: "This spreads as a perilymphadenitis which extends to the floor of the mouth and pharynx."

The path of spread to the sublingual tissues is one of extension around the posterior border of the mylohyoid muscle which makes up the muscular diaphragm of the floor of the mouth. Van Wagnen and Costello note a small amount of salivary gland tissue about the posterior edge of the muscle which serves as a guide for the spreading cellulitis which is decompressing itself medially and upward by extension along the loose cellular tissue planes. Once inside the mouth it may spread to the opposite side with ease by following the horseshoe-shaped cellular tissue arrangement about the anterior part of the tongue.

Davis denies the possibility of primary lymphatic involvement in Ludwig's angina. In discussing the patho-

logical findings in this condition, he says that the site of attack depends almost entirely upon the site of injury. Inflammation may then involve the tonsil, epiglottis and larynx. In some instances the purulent discharge breaks into the larynx. Oedema of the glottis may require tracheotomy and may lead to death. If the teeth are the primary site, the inflammation may involve the periostium of the lower jaw and thence invade all the surrounding tissue. The infection spreads from its point of origin and involves all the tissues within its scope. As Davis points out, regardless of its etiology, its spread is along the connective tissues by direct continuity. It is not transmitted by lymphatics.

He believes that any lymphatic involvement is secondary: the lymphatic glands are not enlarged by the infection carried to them by the lymphstream from the infective focus, but are involved in the infected connective tissue surrounding them, and show change as a result of this local cellulitis.

The pathological process is one of gangrene of the deep laying connective tissues and the muscles within them. The process seems to experience difficulty in piercing the deep fascia, hence the skin and underlying

connective tissue may be affected but little.

Davis further observes that commonly, particularly early in the first stages of the disease, there may be little tendency to the formation or localization of pus, and when the epiglottis and larynx become involved, edema supervenes and gives rise to symptoms of suffocation. Early incisions frequently give exit only to serum, and at that time, no pus is found.

Pus usually makes its appearance later. It has a characteristic dark color and a particularly offensive odor. The cellulitis may then progress along the fascial planes of the neck. In fatal cases the infection follows the cervical tissues down the neck and into the mediastinum, giving rise to a septic pneumonia. The progress of the disease is comparatively acute, running its course in six to twelve days. The process may stop at any time or may progressively increase until death occurs from sepsis, or in laryngeal cases, from suffocation.

Ashurst agrees with Davis in considering the process to be a cellulitis. He defines the pathological process in Ludwig's angina as an acute inflammatory process involving the cellular tissues of the floor of

the mouth and the submaxillary region of one or both sides of the neck. The process may involve the connective tissue spaces, but is in no sense to be considered a lymphangitis or lymphadenitis. He believes that the submaxillary and sublingual salivary glands are not primarily diseased, but are secondarily invaded. Infection involves the sublingual and cervical tissues, but need not be confined to either. It may begin in either situation, but until it spreads from the submaxillary tissues to the sublingual tissues, it does not constitute Ludwig's angina in a strict sense of the definition. As long as it is a submaxillary or cervical cellulitis, and as long as it is confined to the sublingual tissues, the process is merely a sublingual cellulitis, even though the pathological process is almost as acute as that seen in Ludwig's angina.

Ashurst differentiates lymphangitis from cellulitis very carefully and points out that lymphangitis clinically arises from the skin itself, not from lesions of the deeper structures; it is not as a rule attended by much swelling, there is little, if any, evidence of cellulitis and the lymph nodes are habitually involved.

(Often there is no visible involvement of the lymphatic vessels of the limb itself.)

In contrast to lymphangitis, cellulitis seldom, or never, develops as a complication of superficial skin lesions, but is almost always due to septic wounds which penetrate beneath the skin or, at least, as far as its deeper layers; it is attended by much swelling, but not by a lymphangitis; the skin itself is not reddened until quite late in the disease, or until suppuration impends; and the infection seldom gives rise to lymphadenitis.

The possibility of combined lymphadenitis and cellulitis is considered, by Ashurst, a process comparable to phlegmonous erysipelas. Lymphadenitis in the cervical or submaxillary region arises, as elsewhere in the body, from surface lesions: i.e., lesions of the tonsils, the mucous surfaces of the cheeks, mouth, tongue, and nose, of all the air sinuses, the faucies, the nasopharynx, orthopharynx, larynx, and from surface lesions of the face and scalp.

Infections arising in the teeth and their sockets are among the commonest causes of cellulitis, and it is a gross mistake to consider cervical abcess of such

origin as instances of suppurative cervical lymphadenitis. As a matter of fact they are suppurative cellulitis. Sebileau showed this by removing a diseased tooth. His observations are: "(1). After extraction of the disease d tooth, pressure on the submaxillary swelling will cause pus to be discharged from the socket of the tooth, and in many cases the cervical abcess may be emptied in this way, or, after careful probing, through the socket of the tooth or alongside the alveolus. (Such evacuation, as Ivy said, could not possibly occur through inflammed lymph channels.) (2). After the submaxillary swelling is opened(as is required in all patients not seen before the overlying skin is affected) probing detects bare bone at the bottom of the cavity. (3). The existence of trismus, always present when the molar teeth are the focus, indicates that the mandible is fused with the inflammatory mass in the neck: such fusion does not occur in lymphadenitis, but is more characteristic of inflammatory cellulitis."

In the injection studies of Ashurst it has been found that from the point of injection there is immediate spread under the mucous membrane covering the

the floor of the mouth in one gingivolingual sulcus. The effect of such an injection, 40 to 45 cc of solution, was to raise the same side of the tongue against the palate. Almost simultaneously the dye appeared in the submaxillary region beneath the fascia colli. If a greater quantity of dye was used, the stain transversed the fascia colli and also stained the platysma, the subcutaneous fat, and even(in one case) a small area of the overlying skin. The stain was arrested posteriorly by the attachment of the fascia to the sternomastoid muscle, but the stain extended, very faintly, between the upper end of the laynx and pharynx almost to the midline of the body and spread up the anterior pillar of the fauces on the side on which the injection was made. The dye had stained the thyrohyoid membrane forward to the midline. The under surface of the mylohyoid remained unstained, except at its posterior border, where the stain was darker and continuous with that which infiltrated all the interstices of the submaxillary salivary gland, and the sublingual tissues. The submaxillary salivary gland itself was not stained--only the fascia around its lobules. The stain followed the external maxillary artery from within the submax-

illary capsule up onto the surface of the masseter muscle. The stain did not extend appreciably beyond the midline, either within the mouth, or in the neck. In one case, however, the stain, after extending across the sublingual tissues to the other side of the mouth, had filled the submaxillary region on this side as well as on the side of the injection, and finally escaped from the submaxillary capsule along the course of the lingual artery into the neck on the other side of the cadaver from that in which the injection was made.

Blassingame injected dogs in a somewhat similar manner and came to the following conclusions: (1). Ink injected into the peritonsillar region did not enter into the floor of the mouth. (2). Material injected under the mucosa into the posterior part of the mouth passed through the floor down to the deep cervical lymph glands. (3). Ink injected in the anterior floor of the mouth and the mylohyoid muscle did not pass backward, but ran into the lymphatics superficial to the mylohyoid muscle, and finally to the superficial nodes around the submaxillary gland. He concluded that other lymphatic infections other than those of the mucosa of the floor of the mouth might cause Ludwig's

angina, but in general his experiments indicated that infection in the superficial structures outside the floor of the mouth, in the region under the chin and in the superficial submaxillary region, might have its source inside the mouth near the lower front teeth, in the gums, and around the teeth on the external side or outside the midgingival border. He considered,also, that they indicated that infections around the tonsil do not cause Ludwig's angina, and that it is doubtful if the tonsil or any structure below the Styloglossus ligament can ever be the primary source of Ludwig's angina.

Grodinsky and Holyoke injected a gelatine and India ink preparation into the mucous membrane of the floor of the mouth in cadavers and found a rather typical picture in every case. They observed passage of the gelatenous mass around the lateral broder of the genioglossus muscle, which passed on to lie between the hyoglossus and the mylohyoid muscles, as has been mentioned by Thomas. They noted very little tendency for the mass to spread from the submucous region through the median raphe, but extension across the midline occured,as was observed by Ashurst. They found that the

sublingual salivary gland had a tendency to act as a guide for infection, which then passed on into the superficial tissues of the upper neck and face.

They noted, further, that deeper breaks occurred laterally in the sheath of the sternocleidomastoid muscle, and inferiorly through the attachment of the stylohyoid and the posterior belly of the digastric sheaths to the carotid sheath. From the carotid sheath infection might erode the large vessels or break into the prevertebral space,(space "4").

They considered infections starting at the tip of the tongue more apt to localize in the submental region, and hence, take longer to reach the lateral pharyngeal space than those starting farther posteriorly. Accordingly, the element of danger was increased as the portal of entry from the mouth proceeded posteriorly.

Muckleston has summarized the argument and findings of the earlier investigators who considered that the pathology in Ludwig's angina was that of a lymphangitis. His view is that the condition is a lymph-borne disease, regardless of source. The disease implants itself in the cluster of lymph nodes lying about the submaxillary salivary gland. He notes that

Frankenthal believed these ten or twelve glands receive the lymph flow from the lower part of the face, the gums of the lower jaw and the entire floor of the mouth.

Streptococcic lymphadenitis and perilymphadenitis will lead quickly to invasion of the cellular tissue around the submaxillary salivary gland, and the clinical course of Ludwig's angina begins at this stage.

The submaxillary gland facilatates the spreading cellulitis. The floor of the mouth is involved easily, because the gland is about one-third intra-buccal in position and the tissues of the neck take part in the extension. The neck tissues are then attacked from the superficial two-thirds of the gland which lies close under the mandible.

The cellulitis spreads to the valleculae from within the mouth and pharynx: the pillars of the fauces, and the lateral walls of the pharynx are involved. Muckleston believes there is an actual increase in the cubic contents of the mouth as a result of the board-like infiltration characteristic of Ludwig's angina, but others believe this is only a displacement phenomenon.

There is no relaxation of the floor of the mouth,

because the mylohyoid diaphragm is physiologically limited and this diaphragm makes the mass more constrained because its fibers are irritated by a myositis. The tongue follows the path of least resistance with upward displacement to the hard palate. It projects itself forward between the teeth, and backward into the airway of the pharynx.

At this point the infection may progress toward healing by spontaneous resolution, or, as is the case in the overwhelming majority, may progress to a more serious inflammatory process with board-like induration, with or without pus formation.

Abcess formation occurs in the majority of cases. Thomas found pus in sixty-six of his one hundred six collected cases. Other writers observe about this same ratio.

Glogau has reported a case of Ludwig's angina, in which, at operation, an abcess was opened on the left side of the neck which reached behind the mylohyoideus and under the hyoid bone to the site corresponding to the pyriform sinus, and a second portion of it reached anteriorly to the larynx, into the region of the vallecula.

Thomas has shown that the extension and crossing of a submaxillary cellulitis from one side to the other is due to the structural formation of the floor of the mouth. He suggests that the infection passes along the floor of the mouth, along the sublingual sulci and out through the opening in the floor of the mouth on the opposite side: or, even more frequently the pathway may be along the external connective tissue under the symphysis menti. He notes three possible routes of a spread: (1). From below the chin, involving the fascial planes and the connective tissue spaces adjacent to the lingual and fascial arteries, and conveying the inflammatory process to the sheath of the great vessels. (2). Lesions of the hypopharynx, and the upper esophagus will lead to a paraesophagitis and to a cellulitis about the same structure. (3). When the direction of the extension is downward, induration or abcess alongside the thyroid gland and the trachea may arise. If the continuation should be downward, as will sooner or later be the case, the way to the mediastinum is opened, either under the pretracheal fascia to the arch of the aorta and the pericardium, or in front of the prevertebral fascia to the pleura and the posterior part of the mediastinum.

Coplin reports two autopsies, with complete evisceration of the cervical region, in which the lymph nodes were examined microscopically and showed practically no infiltration. He says, "The lymph vessels, in conveying the primary infection, may be compared to the fuse leading to the high explosive shell, the general cellulitis to which the lymph-borne bacteria gives rise."

E. R. Hirsch summarizes his views of the pathology of Ludwig's angina in his explanation of the pathological process seen in the disease. He reasons that from some focus in the upper oral, or mouth regions, the lymphatics, usually within the capsule of the submaxillary salivary gland, become infected with comparatively virulent infection from such a minor focus. The infection then remains within the capsule, or it soon spreads without, giving a periadenitis and a rapidly spreading cellulitis. In the former case, the products of the inflammation are under great tension owing to the denseness of the capsule, and the tension gives rise to great pain, which, if not soon relieved, results in necrosis, abcess formation, or a rapidly spreading and rapidly fatal gangrene. In the latter, more fre-

quent type of case, the infection soon travels outside the capsule, giving a wider area of infection. The course of infection is in the cellular tissues between the mucous membrane of the floor of the mouth and the mylohyoid muscle. The submaxillary salivary gland is involved as often as is the sublingual. The swelling seen in the tongue has now occurred in all the contiguous parts, so that the whole mass is surrounded in front and on both sides by the inelastic structures: i.e. mandible. Therefore, any extension must be backward and downward through the larynx, a distance of only about 5 centimeters from the point at which the infection reaches the mouth. Then, edema of the glottis and interference of respiration produces a fatal termination.

Paulson is of the opinion that the pharynx and larynx are affected directly through the lymphatics, without spreading of the original infection itself.

Coakly believes the pathological process to be a cellulitis, but differentiates one pathological type on the basis of abcess formation, from a non-localizing(erysipelatous type) without abcess formation. In the erysipelatous type, he observed by laryngoscopy

that the mucous membrane is deeply congested and injected with some ecchymotic spots visible,as well as edema of the areytenoids. Coakly says: "These cases run their courses without the formation of any abcess and I believe them to be a streptococcic invasion of the pharyngeal tissues, simulating if not identical with erysipelas."

LUDWIG'S ANGINA

Chapter VII

Treatment

1. Conservative Treatment
2. Surgical Treatment

Conservative Treatment

The following are methods of treatment as set out by observers of Ludwig's angina:

Prophylactic treatment of etiological sites is suggested by Muckleston. He feels that this is more advisable than allowing development of Ludwig's angina, for once the disease process begins, treatment is supportive and surgical.

Newcombe(1895) considered any procedure other than surgery only of historic interest. Surgery has been considered to be the most certain and effective method of treatment in Ludwig's angina. In favoring early and ample incision with an object of relieving tension, rather than evacuating the pus, he quotes Gerster: "There is no pointing, so the entire area of the gland (submaxillary) should be exposed."

Gill and Ridell believe that there is an intimate association between Vincent's angina and Ludwig's angina; because of this they suggest that intravenous neoarsphenamine is theraputically effective in addition to supportive measures used in early conservative treatment of Ludwig's angina.

Treatment of a case of Ludwig's angina with 20cc

of intravenous acriflavine repeated at the end of two days is reported by Levy. His was a single case report, so that its evaluation is difficult, and the effectiveness of the treatment poorly proven.

Fulghum (1938) reports a case which he considered to be Ludwig's angina in a three year old girl. In this case he gave sulfanilamide with recovery in approximately six days after onset. The factual basis behind this treatment lies in the fact that the infective organism in most cases of Ludwig's angina is the Streptococcus, for which sulfanilamide seems to be specific.

Eradication of infected teeth, and hot packs applied locally is the method of conservative treatment suggested by Novitsky. He discourages the use of ice packs to the neck, because Brown (1919--Monographs Rockefeller Institute) has found that cold increases hemolysis by anarobic streptococci. This method is also sanctioned by Hervey and Webber, particularly when using Boric acid. Hervey is of the opinion that hot packs were the factor that turned the course of one of his cases to a favorable outcome.

Dabney in 1924 pointed out the need for stimulants

(whiskey, brandy, or strychnia) and nourishing food to aid in maintaining the patient. He holds antistreptococcic serum in high regard, and about this he says: "Twenty cubic centimeters should be given as early as possible and repeated in twenty-four hours if necessary. I have used it in only one case, but recovery in that case was so rapid that I would certainly use it again and do so promptly."

Dan McKensie first mentions the use of antistreptococcic serum and, in his treatment, gives 25cc as soon as the diagnosis is made. Repeated in twenty-four hours or oftener as indicated.

Babcock gives the best general summary of conservative treatment in Ludwig's angina. His treatment consists of keeping the patient in a semi-sitting position in bed to facilitate drainage; frequent use of antiseptic mouth washes; fomentations of aluminum acetate applied to the neck; tincture of iron in large doses, along with streptococcic serum and general supportive measure. If the condition progresses in spite of this treatment more radical measures must be resorted to.

To the above must be added the opinion of

Frankenthal in which he doubts the value of any chemotheraputic agent in the treatment of Ludwig's angina.

Surgical Treatment

Colp denies the value of any form of conservative or expectant treatment in Ludwig's angina. He believes that adequate drainage must be instituted immediately and that operation must be preformed under local anesthesia in order to avoid anesthetic irritation of the respiratory passages. The old emperical incision extending from the median line from the under surface of the chin to the hyoid bone must be discarded as routine surgical treatment for it simply divides the muscle planes of the floor of the mouth and tongue in the median line, a site in which the infection is practically never located. As noted in the section on pathology, infection is usually in the areolar tissue of the submaxillary triangle: hence, the incision of choice is the lateral one. Several factors must be borne in mind in making any incision. If the infection is in the alveolar lingual sulcus with the most of the swelling in the submental region, a lateral incision parallel to the mandible must do more than divide the deep cervical fascia of the neck. The incision must divide the mylohyoid muscle at right angles to its fibers from its lateral border to the median raphe, separating, if necessary, the anterior belly of the digastric near its

mandibular attachment. This affords free drainage to the space without danger of injury to the ranine vessels, Wharton's duct, or the sublingual glands. If the infection has already spread to the submaxillary triangle, the usual incision is made, for excision of the submaxillary salivary gland must be anticipated. This gives both free drainage and relieves the pressure against the pharynx and indirectly the larynx.

Simple division of the fascia leaves the submaxillary gland blocking not only the drainage of the sublingual space, but the submaxillary space and the retromandibular space. gland removal relieves this.

R. A. Leonardo points out that at the time of operation on the gland there is no abcess formed. If the area of infection is incised at this time, only a few drops of pus are found, associated with a very tense edema. He accounts for the symptoms on the basis of the extensive edema of the floor of the mouth and on the oral side of the mylohyoid diaphragm, giving rise to pressure which must(as Colp suggests) be release. Leonardo holds that the release of pressure is of more importance than incision and drainage, although how such release could be effected without incision is not explained.

He also favors a transverse incision through the mylohyoid diaphragm, its lateral extension being on each side almost throughout its entire breadth. With this he also uses a horizontal incision parallel to the jaw, and about one finger breadth below the jaw.

With regard to glandular excision, as suggested by Colp, he says: "---excision should be reserved for very late and very unresponsive cases."

Another early investigator, G. G. Davis, considered the condition to be only a local affection, which could be stopped by fearless surgical treatment. His method of treatment has not been changed, or greatly improved to the present time.

His treatment consists of administration of primary anesthesia with ethyl chloride, ether, or chloroform, and incision in the median line between the symphysis and the hyoid bone, carrying it through all the tissues, all the way into the mouth, or at least, until the point of the knife can be recognized by the finger inside the mouth, beneath the tongue. Such an incision is easily made, devoid of danger, and effectively drains the infected area. If made early no pus will be found, only blood or a little serum. The relief,

however, is immediate. If swelling is more toward the angle of the jaw, or in the parotid region, incise the skin, and , with a pair of haemostatic forceps, bore slowly into the swollen tissues, spreading the blades, and, if necessary, inserting drainage tubes. In severe cases, the larger the incisions the better. In this affection pus does not show a tendency to accumulate, and the large incision relieves tension, allowing the gangrenous tissues to be cast off.

In cases which develop edema of the epiglottis and larynx, ice and spray inhalations of cocaine and adrenaline may be of value, but tracheotomy should not be deferred too long. A high tracheotomy is the one of preference in order to avoid excessive hemorrhage.

Blassingame also favors early external incision, unless there is a tendency for the abcess to point inside the mouth. The site of incision should be over the area of greatest swelling and tenderness, and it should be large enough to admit two rubber drain tubes.

With regard to the type of external incision Blassingame uses a lateral incision if the abcess is lateral to the geniohyoid muscle. If the abcess is in the midline, its location may be in either one of two

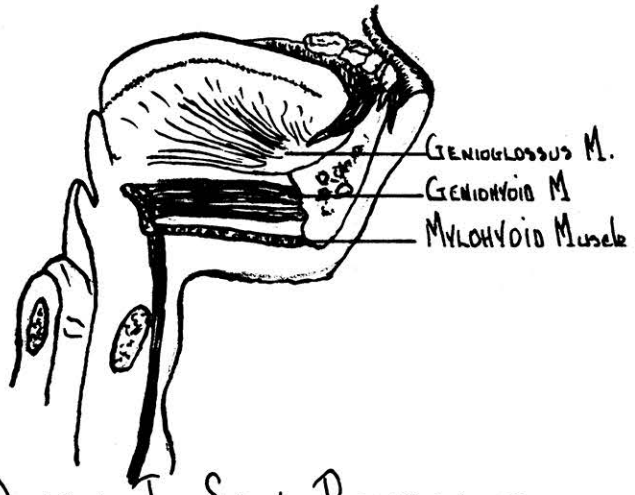

Diagram To Show Relation of the Muscles of the Floor of the Mouth. (After Blassingame)

Relation of Abcess Above the Genio-Hyoid Muscle (After Blassingame)

Relation of Abcess Below the Genio-Hyoid Muscle (After Blassingame)

places: above the geniohyoid muscle between the muscle and the genioglossus muscle, or below the geniohyoid muscle. In the first case Blassingame believes that the incision should be deep enough to divide the mylohyoid and the geniohyoid muscles in the midline. In the second case it will be necessary to divide only the mylohyoid in the midline.

Sautter differs in that he incises for drainage parallel with and anterior to the border of the sternomastoid muscles, and packs the lower end of the incision to minimize the possibility of descending infection. He reminds all surgeons that incision should be made at the lowest level of the cellulitis.

In cases which are not doing well, Sautter makes frequent roentgen ray examinations in order to anticipate the development of any mediastinal infiltration.

Furstenberg believes, on the basis of clinical evidence and dissection studies, that there are four points in the sublingual region in which abcess formation or localization should occur. Such abcesses may be found in the potential space between the muscles of the base of the tongue and the geniohyoid muscles. They may be observed to be at a lower level between the

geniohyoid and the mylohyoid muscles, or they may occupy the corresponding positions on the opposite side of the median line.

He goes even further and outlines two modes of approach for surgical drainage: (1). intra-oral, and (2). external incision. If the abcess is situated beneath the geniohyoid muscle, as indicated by a large external swelling under the chin with only minor changes in the mouth and tongue, it may be drained by an external incision and blunt probing for the purulent collection. When found, a drainage tube is introduced and the region irrigated with Dakin's solution. If, however, the inflammatory reaction in the mouth and tongue preponderate, the abcess may be drained through an incision through the floor of the mouth, on one side or the other of the median line. The importance of early diagnosis and early drainage and unfailing recognition of the four possible locations of pus in the anatomic spaces under the tongue can not be emphasized too much.

Houser suggests the advisability of intra-oral incision for drainage in Ludwig's angina. He notes that in Thomas' report of cases, there were seventeen cases of spontaneous evacuation and all were internal.

His explanation of the relatively few attempts

to evacuate such an abcess by the oral route may be that many of the patients go to surgeons who have had little practice in the use of the head mirror for illumination, and hence, incise in a manner with which they are more familiar.

The argument that one should hesitate to drain pus into the mouth is scarcely tenable, for he points out that no one hesitates to open an abcess in quinsy, or a retropharyngeal abcess, and in support of this view, it must be said that dire results from pus so evacuated are quite unusual. If the mouth can not be opened then external incision alone is feasible. However, if the patient is seen early there is a possibility of bringing the infection to a speedy termination with relatively simple intra-oral surgical maneuvers. External incision may still be performed if this fails.

Yerger points out three objectives in surgery in Ludwig's angina: (1). To relieve tension in the tissues.(Suggested also by Davis, Leonardo, Blassingame, and others.) (2). To provide an avenue of escape from the tissues of the infecting bacteria and their toxins. (3). To drain any collection of pus present.

As regards incision and drainage, Yerger makes an

external incision like that of Leonardo's, parallel to the lower jaw over the submaxillary gland, which will freely expose the region of the submaxillary lymphatic gland, and will locate pus if present. However, if there is a collection of pus in the submental region a median suprahyoid incision will evacuate it. When no pus is found, Yerger suggests that the sublingual region should be investigated by dividing the mylohyoid and searching the cellular spaces of the floor of the mouth. He also believes that the original focus of infection should be removed, if possible, as an alveolar abcess is drained or a carious tooth extracted. When dyspnia is present, due to edema of the larynx, a tracheotomy, but never an intubation, should be done.

Another who advises early ample surgical drainage is Hirsch. In incising, he finds that the most satisfactory method is as follows: incision is made one-fourth to one-half inch mesial to the ramus of the mandible and carried up to the salivary gland, the capsule of which is opened, and incision made into the gland substance; dissection, preferably blunt, is carried through the myolhyoid muscle to the floor of the mouth. An aspirating needle may be used after

incision has been carried through the fascia, in hope of locating a pocket of pus. If found, it is opened, if possible, by blunt dissection. When drained the wound should be left open. If, however, no pus is found, Hirsch makes multiple incisions, separating the tissues in may directions in each incision and inserting drains in all of the separated spaces.

Boyne insists upon wide exposure in the region to be investigated. He incises along the mandibular border so the submaxillary gland can be elevated. He inserts a finger beneath this gland and carries it backward until the stylomandibular ligament is felt. From this position exploration may be carried upward until the styloid process is felt. Then, by carrying the finger downward and forward to the tonsil region, the floor of the mouth is palpated. Finally, if necessary, the vessel sheath may be drained from this point as it lies deep to the styloid process.

Ashurst stresses the point that drainage should be anatomic and not haphazard in any sense. The following are three incisions for drainage as set down by him: "The first incision should be made in the midline between the chin and the hyoid bone, and the knife

should pass directly through the median raphe into the gingivolingual sulcus just back of the symphysis. Into the tract (for it needs little more than a puncture) thus made, a long curved hemostat is passed; its jaws are made to emerge between the teeth, and a perforated rubber tube drain is drawn down from the mouth to the neck. Each end of the tube(one end beneath the tip of the tongue and the other at the skin level beneath the chin) is transfixed with a safety pin.

"A second incision is to be made anteroposteriorly well below the angle of the jaw to the side affected, about 4 or 5 centimeters long through the skin and platysma only. A long curved forceps is then thrust into the incision transversing the submaxillary space, and is brought out through the mucous membrane at the side of the tongue opposite the molar teeth. A second perforated rubber tube is grasped in the forceps and pulled through the neck and a safety pin placed on each end of this tube.

"A third incision is not usually required, and is in all respects similar to the one described above, but is on the opposite side of the neck. When the patient is first seen by the surgeon, the disease is seldom

spread to the second side of the neck; if the two incisions first described are made promptly, the disease is usually arrested.

"Local infiltration of the superficial tissues is all that is required."

Bailey suggests an external incision similar to that used by Hirsch, but in addition, he inserts drainage tubes into the incisions and uses hot magnesium sulphate fomentations at frequent intervals.

Van Wagnen and Costello find that the primary division of the cervical diaphragm gives excellent results. They advocate the division of the mylohyoid as a standard procedure in Ludwig's angina. The incision that they suggest is made at the base of the submaxillary triangle and follows the lower border of the jaw. The facial artery is divided between ligatures. The submaxillary gland is retracted and the mylohyoid muscle is completely divided. Pus is usually found in this area, but in some cases they find it necessary to carry the incisions across to the opposite side. In severe cases with marked edema they suggest the use of a midline incision to convert this U-shaped transverse incision into a T-shaped incision.

In the realm of emergency procedures required in Ludwig's angina, tracheotomy is of much importance. Casselberry says: "---tracheotomy to be of avail in cases of impending suffocation must be done at once, else the continued suction upon the pulmonary blood vessels produced by the muscular efforts in respiration when the glottis is closed will result in fatal edema notwithstanding tardy relief."

Fetterolf and Gordon B. New concur in the value of early tracheotomy. New agrees that drainage of the phlegmon usually causes the edema to subside. However, in the cases in which the edema is progressive and there is no fluctuation developed in the neck to suggest that the diffuse cellulitis is localizing, an early tracheotomy is advisable. He says: "Tracheotomy in the cases reported did not produce an exacerbation of the infection as it is sometimes reported to do."

In late cases radical surgery is sometimes required and the extent of such surgery is only determined by the degree and extent of the involvement of the structures about the mouth and in the neck.

Colp discusses the precedure in removing the sub-

maxillary gland in the following manner: "After the cervical fascia has been divided the gland capsule is easily recognizable and by working from the hyoid part of the gland upward, carefully watching for the facial vein, which is superficial, and facial artery which is deep or in the substance of the gland, exterpation may be accomplished without difficulty. Care should be taken to avoid injury to the lingual vessels which lie quite superficially. In some cases the dyspnea may become so threatening that tracheotomy may become necessary."

Otto Glogau gives the most radical, almost heroic, surgical procedures of any writer on the subject of Ludwig's angina. His cases appear to have been of a grossly mishandled type, seen late. His observations and opinions are summarized in the next few sentences. "Abcesses in the upper respiratory and alimentary tract, including phlegmon, may easily descend by way of the regional lymph vessels and glands into the parapharyngeal, retropharyngeal and retroesophageal loose connective tissue, causing phlegmon of the neck, and descending along the vascular sheath into the mediastinum causing a mediastinitis, sepsis and death. Conservative

treatment including local incision may be tried. However, with the swelling in the neck increasing in size and becoming more tender and discolored, with the occurrence of pains in the jugulum and the appearance of severe general symptoms, the typical external operation should be resorted to. It consists of wide exposure of the vascular sheath and direct opening and draining from there of the involved spaces. Such deep and descending abcesses are frequently complicated by suppurating glands intimately connected with the jugular vein, which may itself be flask-like, extended. This site can hardly be reached by simple incision from without or within, without endangering the life of the patient by puncturing the large vessels.

In severe cases where mediastinitis is threatening or is present, the anterior or posterior collar mediastinum is exposed; it is sealed off with iodoform gauze if found still healthy and drained if diseased. When early resorted to, the typical external operation is a life saving operation. This operation should be given a trial in the most desperate case.

LUDWIG'S ANGINA

Chapter VIII

Complications

Complications

When Ludwig's angina has destroyed its fascial barriers, its course may be extensive and varied.

Furstenberg considers extension into the lateral pharyngeal and pharyngo-maxillary fossa, a space limited above by the base of the skull, continues down the neck as the carotid sheath and opens below into the posterior mediastinum. At the level of the angle of the jaw the fossa is bounded anteriorly by the superior constrictor of the pharynx, laterally by the ramus of the mandible covered by the internal pterygoid muscle; and the inner prolongation of the parotid gland posteriorly by the muscles which cover the bodies of the cervical spine. (Prevertebral space of Grodinsky and Holyoke.) The lateral pharyngeal fossa contains areolar tissue, a dense plexus of lymphatics and the contents of the carotid sheath (space "3" of Grodinsky and Holyoke). It is exposed to infection on all sides. The anatomical relationships follow the course of the primary suppurative process in the throat through the lateral pharyngeal fossa and thence into the parotid gland. By the same token the abcess may extend in the opposite direction from its original focus in the

parotid gland to some point within the region of the pharynx. Such a sequence of events occurs frequently enough to be regarded as a clinical entity.

Suppurative processes in the neck may attack and do great injury to the contents of the carotid sheath. This is particularly true of the internal jugular vein which may become the seat of phlebitis or thrombophlebitis.

If the large vessels of the neck are eroded by a suppurative disease, fatal hemorrhage arises.

Blassingame also has noted a case of thrombosis of the jugular vein on the involved side. At autopsy - in another case gangrenous and septic alteration were seen running from the floor of the mouth to the inside of the thorax.

Thomas considers bronchopneumonia as the most frequent complication in Ludwig's angina as in diphtheria, or about 75 percent.

Gill and Ridall include as complications of Ludwig's angina bronchopneumonia, general sepsis, slough through the carotid sheath with hemorrhage, asphxia, and mediastinal abcess.

Gingrass says that in untreated cases of two to

three weeks duration the patient dies of aspiration pneumonia, edema of the glottis, or sepsis from diffuse suppuration or gangrene.

Osteomyelitis of the mandible is still another complication of Ludwig's angina listed by Stetten and Boyne.

Ferguson in 1888 reported the autopsy of a case of Ludwig's angina of dental origin. At autopsy he found that there was about a quart of purulent fluid in the pericardium and both layers of the pericardium were covered with "recent lymph". There were numerous gangrenous cavities under the root of the tongue, which communicated with the wound along the lower border of the jaw and from which pus extended downward through the mediastinum to the pericardium. The pus infiltrated the planes of fibrous tissue along the trachea, oesophagus, and great vessels of the neck.

A similar autopsy with essentially the same findings is reported by Hochbaum. In this case he believed the abcess was primary at the region of the tonsil.

LUDWIG'S ANGINA

Chapter IX

Prognosis and Duration

Prognosis and Duration

Prognosis in most cases of Ludwig's angina is very grave. Untreated patients usually die in about two to three weeks. Bailey says that unless the tension is relieved the patient's life may be threatened or taken in twelve to twenty-four hours.

Davis believes that death occurs in ten to twelve days in most fatal cases. He describes the course of the disease in the following manner: "--during the first four to six days, the skin is not red and the constitution is not much affected. Later openings occur posteriorly on the inside of the mouth, and a thin grey or red-brown exudate develops. A gangrenous odor develops, the lungs become affected, and death ensues in ten to twelve days."

A single case cured with Sulfanilamide in a period of twenty-four to forty-eight hours is reported by Fulghum.

Thomas' statistical summary of one hundred six cases reported in the literature and in his own practice gives a fatal termination in forty-three patients or about 40 percent.

Twenty-three cases seen in the Cook County

Hospital, in which all recovered after free external incision into the abcessed region, are summarized by Yerger.

Barryhill suggests that the greatest single factor in the prognosis of Ludwig's angina is the extent of the spread of the condition.

Therefore, prognosis may be said to be increasingly grave in direct proportion to the delay in treatment, other factors remaining constant.

LUDWIG'S ANGINA

Chapter X

Conclusions

Conclusion

Only a few summarizing statements need be made relative to Ludwig's angina. The simple knowledge that such a condition does exist is sufficient; however, after having made the foregoing study of Ludwig's angina, it is my opinion that the following facts might be of value when neck and mouth infections are encountered.

Ludwig's angina is a relatively rare condition, but the process must always be considered in making a differential diagnosis from other neck affections, including that of acute cervical cellulitis, abcess of the salivary gland, stone in the duct of the salivary glands.

Treatment of Ludwig's angina requires a thorough knowledge of the structure and anatomical relations of the floor of the mouth, and the fascial relationships of the sheaths of the mouth and neck, in order to recognize the site of primary infection, and the course of spread taken by a progressing process.

The commonest etiological agent in Ludwig's angina is dental infection, or trauma of dental origin. Gross trauma, penetration wounds, and other infective processes

in the naso-pharynx and mouth may also play a part in the development of the condition. General supportive treatment with fluids and sedatives are essential aids.

The bacteriological findings vary in individual cases. Streptococci are the rule, in either pure culture, or mixed with other organisms, including Staphylococci, Colon bacilli, and Diptheroids.

Symptoms of the condition locally are those of pain in the region of the mouth, marked dysphagia, change in voice and subjective symptoms of suffociation. The systemic symptoms are those of toxemia; they become very marked in a short time unless treatment is instituted.

Abcess formation may or may not occur, depending upon the virulence of the organisms, the resistance of the individual infected, and the speed with which treatment is instutited.

Treatment of Ludwig's angina is necessarily rapid and early, in contradistinction to the other infectious processes in the neck which may be cared for by supportive treatment alone and waiting for a site of localization to develop.

Early treatment of the condition properly given affords almost complete relief from symptoms immediately. Subjective relief from symtoms can be used as a means of determining the effectiveness of the therapy, and whether or not sufficient treatment has been carried out.

Conservative treatment is not held to be of constant value. It may be applicable in early cases in which the process is carefully watched and controlled by surgery if responding poorly.

Complications in Ludwig's angina include: bronchopneumonia, edema of the glottis resulting in death from suffocation, and death from sepsis. Others less frequent, include massive hemorrhage from erosion of the large vessels of the neck, orteomyelitis of the mandible.

The mortality is relatively high. Thomas reported a mortality of 40 percent in his review of 106 cases. Currently, this value has been lowered somewhat, but the morbidity and mortality are much too high, probably because of the delay occurring before the condition is recognized and treatment is begun.

LUDWIG'S ANGINA

Chapter XI

Case Report

Case Report

There has been one case of Ludwig's anging treated at the University of Nebraska Hospital, which I shall report because its course and manner of treatment appear to be characteristic.

The patient, a white single American girl of twenty-one years, entered the University Hospital for the first time February 27, 1927. She complained of:

(1). Swelling of the right lower jaw and right cheek.
(2). Pain in the entire lower jaw.
(3). Fever following chill.
(4). Painful difficult swallowing.

The patient was well until February 25, 1937 at which time she noticed a swelling of the lower jaw in the region of the third molar, but there was no pain at that time.

She consulted her dentist on the morning of February 26th, and he advised extraction of the third molar, but deferred this until the acute swelling from the process had passed somewhat.

The patient then noticed that she could not chew food easily, for after the onset of pain in the lower jaw, the pain was made worse by chewing.

On the evening of February 26, 1937, she found that

she was unable to open her mouth fully and pain at the mandibular articulation was greatly increased.

At 2:00 o'clock a. m., February 27, she awoke with a hard chill and a rise in temperature. By morning she was unable to swallow solid food because of the pain and swelling in the back of the mouth and throat. (At the time of admission to the hospital, swallowing liquids was very painful.)

The right cheek continued to swell, so that the patient presented a picture similar to mumps. The swelling was most tender at the angle of the mandible, and extended upward toward the ear.

The patient had run a temperature daily since the onset of pain. Mucus collected in the back of the throat, and the patient was unable to swallow or expectorate this material without extreme difficulty.

She also complained of frontal headache from the onset of her illness.

There was no history of previous difficulty with the teeth.

The examining interne made the following notes regarding the patient: "The patient is lying toward the left side holding her neck rather rigidly, because

of severe pain which accompanies movement of the head or neck. The breath is foul--with almost a gangrenous odor. The jaw seems to be set, and the patient cannot spread the incisors more than 2 cm. apart. Any movement of the jaw is painful.

"The entire right side of the face is swollen and very tender. There is also a swollen enlargement of the tissues beneath the chin, painful to touch. Another localized tender painful swelling may be seen overlying the upper portion of the sternum.

"Satisfactory exposure of the oral structures is difficult. The teeth appear normal. The tongue can not be protruded but look normal as far as it can be exposed.

"There appears to be a swelling in the region of the lower right second, and third molar teeth, with a collection of purulent material in that region. No definite abcess sinuses can be seen. Any attempt at swallowing causes excruciating pain."

The day after admission the patient's mouth was x-rayed. The x-ray report reads: "P.A. and oblique radiographic studies of the right mandible show a

sharply circumscribed area of rarifaction in the second and third molar region measuring 1½ by 2½ cm. having the general appearance of a cyst. The third molar tooth is impacted at nearly right angles against the second molar."

The patient was seen immediately after x-ray examination by Dr. Schearer, who suggested immediate operation. At operation the impacted molar was exposed and extracted with forceps. The lower right second molar was also extracted. A cyst cavity inferior to there teeth was exposed about the size of a "hickory nut" (3-4 cm.). This cyst cavity was filled with bloody purulent material which was removed. The cyst cavity was curetted and free drainage of the purulent material was established. The patient had some difficulty with respiration due to the fact that the mouth could not be opened widely--special care was used to be certain that no aspiration into the lungs occurred. The purulent material was removed by suction and the pharynx aspirated well.

The patient was returned to the ward semiconscious, but in a fair condition.

After returning to the ward, the patient continued to complain of pain in the right ear and right jaw region, which was made worse by eating.

One thousand cubic centimeters of Fischer's solution was given by proctoclysis post operatively. Hot magnesium sulphate dressings were applied to the entire cervical region every hour. Saline mouth irrigations were given.

The patient continued on this regiem from February 27th until March 3rd, at which time there appeared to be some signs of localization below the chin. An anesthesia of less than 6/10gm. of Avertin was given: an incision(midline) was made below the chin; the area was opened by probing into it with a hemostat. A moderate amount of thick foul smelling pus was obtained. A rubber tube drain was inserted into the incision.

The hot packs to the area were continued, as were the saline irrigations. Pain persisted almost unchanged even after the pus was evacuated. Mucus in the mouth and throat continued,as before, tenaceous, heavy, and very troublesome. The patient now showed some signs of exhaustion, and nervousness.

On March 18, 1937, a third operative procedure

was undertaken. An incision one inch long and one inch to the right and one-half inch below the former incision was made. No pus was found. A rubber tube drain was left in place.

Following this incision the patient showed gradual improvement. On March 20th, two days after her last operation, the hot packs showed purulent drainage, and this incision then continued to drain until March 25th, when the drain was removed.

From this time on, the patient showed rapid improvement. She was kept on high carbohydrate diet until her dismissal on April 14th, 1937, having been confined to the hospital for forty-seven days.

On dismissal Dr. Schearer suggested that the patient continue a rich diet, and rather light work until she regained her normal weight and strength. Her progress has continued satisfactorily, and she has had no recurrence of symptoms.

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