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LUDWIG'S ANGINA

Louis Rosenbladt
LUDWIG'S ANGINA

When, one of the most dangerous and fatal phlegmons known to the medical profession, can be treated in such a manner as to reduce the mortality rate a marked degree and the morbidity to practically nil by certain methods of approach while some of the men well rated in the profession still advise indefinite treatment and report mortality rates as high as forty to sixty percent, it will naturally arouse the curiosity of a student who has seen clear cut cases in advanced stages, operated and walk out of the hospital in excellent condition on the fourth day. Such seems to be the status of the infective process involving definite anatomical areas in the floor of the mouth and the neck, known as Ludwig's Angina.

Ludwig's Angina is an acute cellulitis involving the areolar tissues, both of the floor of the mouth and of the submaxillary area. Various organisms may cause it but the streptococcus is the most common. By extension to the larynx or to the
mediastinum it causes as a rule, death, unless promptly treated by surgical incision through the mylo-hyoid muscle.

Hippocrates of Cos, the master physician of the fourth century before the Christian era, makes many references to conditions of the floor of the mouth and the neck which resemble the disease now known as Ludwig's Angina.

In "The Prognostics" (Section 23) he tells of patients with swelling both within and without the throat, of swelling and redness determined in some cases outwardly and in others to the lungs with serious prognosis.

"Regimen in Acute Diseases" (appendix 2, section 6) gives a vivid description of the tongue which changes from broad and becomes round, its natural color becomes livid, from a soft consistency it grows hard, instead of being flexible it becomes inflexible, so that the patient would soon be suffocated unless speedily relieved.

Acute septic diseases of the throat were called by the Greek writers cyanche and by the Latin writers angina. Muckleston says the literal Greek translation is "dog choking."
Muckleston gleaned from the Proceedings of the First Annual Meeting of the American Laryng. Association of New York, 1879, the report of William Cullen, an early scotch physician, classifying cyanche which seems not to include the condition known today as Ludwig's Angina. However, Dr. James Statton, President of the New Jersey Medical Society, in 1789 spoke on Cullen's classification saying that cyanche tonsillaris included simple tonsillitis and quinsy, also more severe infection with extension to the trachea and a resultant suffocation. Cyanche maligna was faucial and pharyngeal diphtheria. Cyanche trachealis included croup and laryngeal diptheria. Cyanche pharyngea was an extension of tonsillitis to the pharynx and cyanche parotidea was epidemic parotitis or mumps.

Frankenthal says the condition was observed in America as early as 1735.

In 1836 Wilhelm von Ludwig of Stuttgart gave the first comprehensive discussion of the disease and his consideration of the onset, symptoms and progress of the disease have really never been improved upon up to the present time. Ashhurst gives an interesting account of Ludwig's life.
Wilhelm Friedrich von Ludwig (1790-1865) was the son of the parson of Ulbach, near Stuttgart in Wurttenberg. He studied medicine at the University of Tubingen and was graduated in 1811.

He served a term in the army of King Friedrich of Wurttenberg, first as a common soldier and later as unterarzt (assistant physician).

He accompanied the army during the Napoleonic invasion of Russia reaching Smolensk.

During the retreat he was taken seriously ill (Typhus) and made a prisoner by the Russians and was carried back to Pensa in Russia. Attached, as a surgeon to the suite of a princess and her train, he went south of Russia where he performed many operations.

He was sent home from Russia when the king of Wurttenberg joined allies against Napoleon in 1814. In 1815 by royal decree he was made Professor of Surgery and Midwifery in the University of Tubinger.

In 1816 he was made Leibmedicus to King Friedrich (personal physician) and when the King's son became the new king in 1817, Ludwig returned to Stuttgart at his Leibmedicus physician. He there acquired a large practice and under the third king
became counsellor of the Medical College and with gradual promotion became in 1844 the Director of the Medical College.

Ludwig retired from the Medical College duties in 1855 and was the President of Wurttengergische arzliche Verline from 1835 to 1846. Wrote little and in 1836 described inflammation of the neck which has since been known by his name, and stated by his biographer: "The short, clear, and comprehensive account which Ludwig gave of it, has been surpassed by no later description of the disease." In 1847 he published a short notice on the cure of artificial anus.

In 1861 his jubilee was fittingly celebrated. Thereafter his health broke; he developed a cataract and had a stone in bladder for which two operations (presumably lithotry) were performed. On December 7th, an inflation of the neck developed, which boded the worst, and in the early morning of December 14th, 1865 he lost his life suddenly and without any immediate warning. It would seem from this description that he died of the disease to which his name is given.

The Stuttgart Journal in which Ludwig's
original reports were published did not have a wide circulation, but Ludwig's paper was noticed at considerable length in the Gazette Medicale de Paris and in Schmidt's Jahrbucher for 1837.

It was from the later sources and not from the original that knowledge of the condition gradually spread.

"Cellus Tissue" was the term used in the early part of the Nineteenth century for all tissues with interstitial meshes, subcutaneous, subarachnoid, submucous etc. The term is still used for subcutaneous and retroperitoneal tissue but "areolar" is a better term. The word cellular refers to the fact that honeycomb-like spaces are present. Cellulitis is the term in English speaking countries for inflammation of this tissue. French prefer phlegmon. Germans say Zellgewebssentzundung, inflammation of cellular tissues.

The work angina, from the Latin, angere (to strangle, to suffocate) is sufficiently descriptive of the malady to be retained in connection with the name of Ludwig.

Thomas states Ludwig's own description warns repetition "a sore throat of a rheumatic or erysipelasous nature proceeds as a rule the first symptoms which are:
a slight rising temperature with a number of chills, headache, weakness, some loss of appetite, slightly coated tongue, some difficulty in swallowing, which is in the early stages and in some cases scarcely noticeable, then a hard swelling develops either on both sides of the throat or more commonly only on one side, usually in the cellular tissue that surrounds the submaxillary glands, less frequently in that around the sublingual or parotid. This hard growth extends with some tissue changes gradually around the throat then under the jaw to the chin and down over the larynx and often backwards in the cellular tissues of the parotid, also causing a marked swelling on the outside. This growth also extends through the cellular tissues that cover the musculature between the larynx and oral cavity and even these muscles themselves are sometimes apparently involved in the hardening process; The tongue deep red in color rest 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 upwards and somewhat backwards; 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, and gurgling. Swallowing becomes very difficult with straining of all the throat muscles do undoubtedly, chiefly, if not entirely, to the mechanic pressure of the growth, because an inflammatory swelling of the mucous membrane is present either at the beginning of the disease or later if mercurial drugs are used in treatment. Yet with the progress of the disease a considerable amount of mucous 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 feeling are a little altered, appetite and ability 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 noticed; inside the mouth frequent exudations of inflammatory lymph appears (if this has not occurred
before), the growth under the tongue seems softer, as if the serum under the mucous membrane was diffused and partly coagulated. 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 sometime 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 at the root of the tongue, or more to the front on the under side of the inferior maxilla, break out and discharge a thin, greyish or reddish brown fluid, with a bad odor, which becomes more and more like the ischor of a necrotic process. With the beginning of this process, which is of the nature of real mortification the general symptoms becomes more marked. The fever is higher with exacerbation usually in forenoon, the sleep is broken with heavy sweats, frightful dreams, somnambulism; there is a marked increase of sediments of the urine, sometimes slight delirium. Notwithstanding the fact that the growth may diminish in size swallowing is
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 of five days, the tenth or twelfth from the beginning of the disease, coma and death result with evidence of an edema of the lungs. There are variations in the symptoms as noticed, especially in development of the fever, the time of the development of the disease and the severity of the local process, which I will not discuss in detail as my purpose is to give a general picture of the disease that will aid in a recognition at the bedside." (Muckleston, translation from address of C. B. Coakley.)

Ludwig's Angina is a condition which has escaped the notice of a great many physicians, but there are about three hundred cases in the literature.

Yerger in a report of twenty-three cases of true Ludwig's Angina in Cook County Hospital gives the proportion of males to females, ten to one, and the ages from seven to fifty-six with the majority
occurring in the third and fourth decade. Thomas reports seventy six males and twenty females and says the discrepancy is due to the fact that males are subject to more extreme atmospheric changes and take poorer care of the teeth. The majority of his cases were in the latter part of the second and in the third decade. This period he terms as the age of carious teeth. He reports one case present at birth and the oldest patient was a woman age seventy years.

Ludwig was the first to discuss its causes and nature. Thomas in a review of eighteen cases said there were streptococci alone in six; Streptococci with other organisms in eight; staphlococci alone in two; pneumococci alone in one; undetermined in one. He reports that Lockwood found streptococci in Gibson's case though Gibson could find no microorganisms in the tissue.

After reviewing the literature and accounts of culture of Ludwig's Angina Thomas comes to the conclusion that there is no specific infection responsible although the evidence toward the streptococcus is very strong.

Blassingame reports practically the same
bacterial excitants but stresses bacillus septicus which is anaerobic and gas forming, and is difficult to grow in culture. He deals at length on the presence of this organism in one case and even in this one it was found in conjunction with other organisms so its roll in etiology is questionable.

Carco says the etiological factor is often represented by a streptococcus but there may be forms provoked by the pneumococcus, staphlococcus, and by bacterial associations. He reports a case which he says was caused by a pure strain of streptococci because of its response to anti-streptococcal serum.

Stein reports a case which on culture yielded staph., minococcus catarrhalis and a fusiform bacillus (probably an association of Vincent's with a staph infection). Heart says the strep. is the usual offender. Voorhees reports a case complicated by gas gangrene though no B. Wilchii could be demonstrated. Ashhurst reports eighteen cultured cases in which Strep. predominate and six produced pure cultures of Staph. However, all of his cases were opened by through and through drainage between the neck and the oral cavity so the results
of the culture are questionable. Hervey's case had Strep. in the pus evacuated and the blood culture was negative. Hirsch reports Strep. as the most common cause but sayt Staph, colon and many other organism may cause the disease.

Van Wagemen and Costells say, "the true way to look upon the disease is to regard it first as an intensely infectious phlegmon and second, as occurring under peculiar anatomical conditions. Strep. they believe is the infecting organism in the primary type while Staph. predominates in the secondary type. Careful investigation would, they believe, show the presence of spirochetes in pus heretofore reported as sterile.

Dental sepsis plays a major role in the predisposing cause and original source of infection. Compound fractures of lower jaw, impetigo of face, wounds of floor of mouth, tonsillitis, scarlatine and diphtheritic infection, peritonsillar abscess, stone in Wharton's duct, herpetic ulcer, tonsillitis have all been mentioned as sites of origin for true Ludwig's Angina. Alloway even reports mastoiditis as a forerunner of Ludwig's Angina.
Ashhurst quotes Moty who says:

1- That dental caries gives rise to dental pulp gangrene which spreads to the apex then to the alveolus and absorption of cement and wall of alveolus takes place. Pus discharges through the canal unless it is plugged or very small in which case a phlegmon results.

2 - Pus pierces the alveolus in the thinnest part, giving rise to dento-alveolar abscess in the surrounding cellular tissue. It seldom works its way between the alveolus and the neck of the tube as the later is united to the gingival musosa by resistant fasnia; so it points to the skin or places in buccal musosa more or less distant from the diseased tube.

3- But, when acute (as after untimely filling of tooth) there is not time for absorption of the alveolus, so the infection enters the dental canal of the mandible, spreads toward spine of spix and makes its way toward the cellular tissue which separates the upper surface of the mylohyoid from the gum, or toward that which surrounds the carotid vessels in the neck.
4. If a back or wisdom tooth is infected in this way the outer wall of the mandible is so thick that perforation occurs only through the inner wall so the sublingual tissues are infected at once.

Van Wagenan and Costello divide cases of Ludwig's Angina into primary and secondary. They do this because of the different immediate sites of cervical infection which give rise to the dominating symptom—sublingual phlegmon—on the different paths over which the sublingual tissues become infected, and on the different relationships which the phlegmon bears to the whole mass of infection.

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Thomas says that the so called idiopathic cases originate in lymphnode as evidenced by a "lump under the jaw" for a period of a week prior to the development of the angina in one of his cases.

Blassingame says that Ludwig's Angina is a phlegmonous process arising from infections within the floor of the mouth and localized in a definite anatomic space. This space is secured by boundaries, having as its floor, the mylohyoid muscle; as its posteriord wall, the muscles which unite to from the base of the tongue and the deep part of the submaxillary gland, as its roof, the tongue and mura covering the floor of the mouth. The abscess he sayd may be located above or below lateral or medial to the geniohyoid muscle. In his first statement he contradicts all other writers on the subject in that he does not believe there is any involvement in the sub-maxillary region. Other writers, notably, Thomas, Davis, Price and Ashhurst believe the infection is carried to the sub-maxillary region and when pus develops it forces its way around the posterios edge of the mylohyoid muscle with the submaxillary salivary gland to reach the sublingual spaces. Blassingame's conception is slightly inconsistent since as Thomas said,
Ludwig's Angina begins either in the sublingual or submaxillary regions but is not fully developed until both regions are involved. He supported the statement of Ludwig that it begins in the region of the angle of the jaw.

Thomas' anatomical studies are more complete than those of Blassingame and the views of the former are accepted rather than those of the latter.

The buccal-pharyngeal wall is intact from the posterior border of one mylohyoid to the posterior border of the other. There is a gap between the posterior borders of the mylohyoid and the anterior border of the middle constrictor, extending from the hyoid upward and backward to the inner side of the lower jaw near its angle, partially filled with hyoglossus muscle which passes through it. Structures passing from the mouth to the neck or vice versa do so through this opening: glossopharyngeal and hypoglossal nerves, lingual artery and vein and stylo glossus muscle. The deeper portion of the submaxillary salivary gland projects into the floor of the mouth near the root of the tongue where it lies just under mucous membrane. Connective tissue of the submaxillary fossa is directly continuous with that in the floor of the mouth, so extension of a submaxillary cellulitis to the sub
lingual region, which occurs so early and so constantly in Ludwig's Angina is readily understood.

Asnhurst's experimental injection are worth repeating. Using dilute chinese ink as staining fluid he demonstrated the immediate spread of this stain from its point of infection under the mucous membrane covering the floor of the mouth in one gingivolingual sulcus. The first effect of such an injection of forty or forty-five cc of the solution is to raise the same side of the tongue against the palate. Almost simultaneously the dye appears in the submaxillary region beneath the facia colli. If a greater quantity of dye is used the stain soon traverses the facis colli and also stains platysma, subcutaneous fat and even in one case a small area of over lying skin. The stain was arrested posteriorily by the attachment of the facia to the sternoceleidomastoid muscle; but the stain extended between the upper end of the larynx and the pharynx to the mid line of the body and spread up the anterior pillar of the fauces on the side on which the injection was made. Its stained the thyro hyoid membrane forward to the mid line. The under surface of the mylo hyoid remained unstained, except at the posterior border; here the stain was dark and was continuous with that infiltrated all interstices of the
the sub-maxillary salivary gland and sublingual tissues. The submaxillary gland itself was hot stained, only the fascia around its lobules. The stain folled the external maxillary artery from within the submaxillary capsule up on the surface of the masseter muscle. The stain did not extend appreciably beyond the midline, either within the mouth or the neck. In one specimen however, the stain, after extending across the sublingual tissues to the other side of the mouth, filled the submaxillary region on this side as well as on the side of 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. These few experiments merely serve to confirm what was already known of spread of infection from clinical observation.

In general the symptoms and course are typical and essentially constant. Primary Ludwig's angina begins and continues to its termination as a fulminating, rapidly spreading infection of the nature of a cellulitis. Ludwig's points for diagnosis are still valuable:

1. Comparitively slight inflammation of the throat itself.
2. The so-called wood-like hardness of the swelling of the cellular tissue which is peculiar to the disease.
3. The hard swelling under the tongue.
4. The even progress of the whole swelling.
5. The unaffectedness of the regional lymphatic glands which appeared quite free in many cases.

The onset may be that of pain from carious teeth or at the site of origin. This is quickly obscured by pain in the submaxillary gland region due to distension of tissues. Tension on the cervical tissues, say Van Wagenen and Costello, by the third day or fourth is sufficient to force an extension to the sublingual spaces. Here the process of sublingual phlegmon may continue for three or four days before definitely invading the larynx. The laryngeal obstruction may arise with extreme suddenness. The secondary type of Ludwig's angina is least fulminating and laryngeal involvement is much later though the means of invasion is the same. Laryngeal obstruction symptoms appear fairly late in the disease and vary with the rapidity of spread of the infection. Before
the above mentioned symptom develops there is a surprising lack of constitutional symptoms, most being local manifestations.

Thomas' suggestion of a postero-lateral spread of the infection from the space deep to the mylohyoid, to the larynx is upheld by Van Wagenen and Costello. There is a hiatus between the posterior edge of the mylohyoid muscle and the middle constrictor of the pharynx. The tendon of the stylohyoid muscle or the glossopharyngeal nerve through this space may either serve as a ready guide to a spreading cellulitis. Van Wagenen and Costello in an attempt to determine if this pathway for infection could be demonstrated experimentally, made injections into the sublingual tissues. They traced their injection medium into two locations in each location - first, about the sublingual salivary gland and out into the tissues of the submaxillary region, and secondly, and quite distinctly, into the tissues at the side of the epiglottis and larynx. The space through which it reached the larynx was the one above mentioned.

The external swelling may be present first or the sublingual may be the first to appear. In either case it will more than likely spread to
the uninvolved area and until such time the case does not constitute a true Ludwig's. The symptoms and signs were given in the original description by the man whose name it now bears, but briefly, again, they are: brawny swelling of the neck, elevation of the floor of the mouth, thickening and lividity of the tongue which becomes protruberent, inability to open mouth or to close it completely, dysphagia, drooling of saliva, difficulty in speaking, and later, dyspnea.

Some cases assume a grave aspect in twelve to twenty-four hours while others are mild for days then suddenly assume an alarming character. The latter type could be explained on the basis of a secondary Ludwig's angina. Hirsch says that the diagnosis is easy if a person keep in mind the fact that there is such a condition.

All authorities agree that the proper treatment is surgical drainage immediately the diagnosis is made. Compromising, palliative, watchful waiting measures are worse than useless. They rob the patient of his best chances for recovery.

Spontaneous openings into the floor of the mouth have been reported but as a rule are inadequate and further incision is necessitated. Thomas says that cases reported as clearing up by so-called spontaneous resolution, have a small
unrecognized perforation in the mouth.

The simplest incision is recommended by Ashhurst and applicable in situations where complete hospital accommodations are not available, and where formal dissections of the neck to and through the mylohyoid muscle are not advisable. It consists of through and through drainage from the neck to the floor of the mouth in either two or three areas. The first of these is a puncture in the midline between the chin and the hyoid bone, passing through the median raphe into the gingivo-lingual sulcus just back of the symphysis. By means of a curved forceps, a rubber drain is drawn from the mouth to the neck through this tract, and each end transfixed with a safety pin. His second incision four or five centimeters long and through the skin and platysma only, is made parallel to the jaw and well below the angle on the side affected. A long curved forceps is then thrust into this 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 by this forceps and pulled through the neck, both ends being again transfixed by safety pins. These two incisions in the hands
of the originator usually serve to arrest the course of the disease, but if it spreads to the opposite side he advises, on this side, an incision in all respects like the second one described. This method of approach does have points of superiority in that it can be done by anyone without fear of hemorrhage or nerve injury. Its disadvantage lies in the fact that an already infected or infective one is opened into the deep structures of the neck. This disadvantage can be somewhat discounted by viewing Ashhurst's uniformly good results. Credit is rightly given to G. G. Davis for systematizing these incisions.

Park advocates the same type of incisions as those given above with the exception that he believes they should not extend through the mucous membrane, just to it.

Hervey tried abortive measures for thirty-six hours and nearly lost the patient before incising deep into the submaxillary gland and likely dividing the posterior portion of the mylohyoid, liberating a considerable amount of pus, recovery.

Blair, advises incision in the midline down to the hyoid; and from the lower end of this backward to the angle of the jaw.
Colp advocates excision of the entire submaxillary gland, which functionally is not missed but leaves a cosmetic defect.

White says the point of election is over the region of the most swelling, and parallel with mandible. Sharp dissection should carry this incision through the deep fascia to the mylohyoid muscle and submaxillary gland. The space deep to this is best opened bluntly after the mylohyoid is split forward from its posterior edge.

Yerger believes the original focus should, if accessible, be removed. Then the submaxillary incision and if necessary a midline incision is of benefit.

Hirsch makes an added effort after an incision not unlike White's, by exploring the surrounding tissues with an aspirating needle for the possible presence of pus.

Thomas used the incision of Belorm, over the submaxillary gland parallel with the lower jaw, freely dividing the mylohyoid muscle. The finger is passed upward until only mucous membrane covers it.

Bailey, Van Wagenen, and Costello all concur in advising wide incision below and parallel to the lower border of the jaw. The facial artery is divided between ligatures and the mylohyoid
muscle is completely divided on one side or on both sides if necessary.

There has been considerable discussion over the type of anesthetic to use in these cases. Robertson even goes so far as to say a general even if a tracheotomy must be performed to administer it, so a more complete dissection can be carried out. The majority advocate local anesthetic or local in conjunction with some nitrous oxide in small amounts.

All agree that the armamentarium for a tracheotomy should be ever present from the time of diagnosis until a marked subsidence of the symptoms occurs and the patient is well on the way to recovery.

One case here reported was opened through the supposed original focus of infection in the mouth, and the others were opened below the body of the mandible and about midway between the midline and the angle of the jaw.

There is some doubt as to what causes death in the fatal cases. Ashhurst believes the most common cause is sepsis, although most of his cases exhibited marked dyspnea at and before death.
Park says the prognosis is grave, not for toxemia but for edema of the glottis. Yerger reports marked edema of neck struction especially of the glottis in two of his cases coming to necropsy.

Hisch says, "edema of glottis with interference of respiration causes termination." There may be edema of the glottis and death possibly without any previous dyspnea.

Paulsen says fourteen out of sixteen larynges examined at autopsy after Ludwig's Angina showed edema of glottis.

Thomas say patient dies in state of septic intoxication but reports dyspnea absent in only three out of one hundred six cases.

Van Wagemen and Costello say toxicily from marked sepsis undoubtedly has a great deal to do with death. Partial asphyxia may also contribute. Only occasionally they believe does a sudden total occlusion of the larynx by edema of the glottis cause death. In case III reported here death would surely have resulted from edema glottidis without immediate surgical intervention.

Up to 1908 the mortality in reported cases as stated by Thomas was forty per cent. From that time to the present the mortality has been twenty-
eight per cent. This decrease is due, likely to the wider recognition of the necessity for prompt radical treatment.

The disease is so clear cut that it is not likely to be confused with other conditions. However, Holmes reports three cases of ruptured aneurysm into the mediastinum and deep cervical fascia with the symptoms of Ludwig's Angina. "Although these cases showed signs of infectious cervical cellulitis they had had symptoms suggestive of thoracic aneurysm lasting for many months. Onset was more severe than even the severe infection. All began with severe, sharp, cutting pains and feeling of much anxiety. The prognosis is always bad and treatment is only to alleviate distress. Incision will result seriously. The skin is red, floor of the mouth and tongue are not swollen and hard."

White, gives a list of somewhat similar ailments for use in differential diagnosis. Ordinary adenitis in children in damp climates; leads to softening, suppuration, no alarming pain or toxemia. Osteitis and Periostitis of the mandible with severe submaxillary cellulitis from tooth socket infection, usually in children (front), not usually dangerous—simple incision. Submaxillary gland infection from a stone in Wharton's Duct can be differentiated by the presence of a salivary colic. Lumps, septic parotitis, tuberculosis adenitis
and erysipelas are usually obvious.

The disease usually terminates the life of the individual by sepsis or glottic edema before time has lapsed for complications to arise unless properly treated. However, Sproule reports a case at autopsy with acute purulent pericarditis. White reports a case dying from profuse hemorrhage due to slough of the carotid wall. Ashhurst reports a case with infection extended into the mediastinum. With autopsy, however, he says the cause of death was uremia. In this sort of cases the extension downward follows the plane between pretracheal and prevertebral fascias.

Although proper incision early, does much to arrest the course of the disease, additional treatment can be carried out. Tracheotomy will do much to relieve the dyspnea but Ashhurst says that it should be reserved until the patient is almost moribund because proper incision will also relieve this symptom. He irrigates through the drainage tubes with saline solution and uses moist dressings. Careful nursing, cautious feeding, fluids by Murphy drip or subcutaneously until the patient can swallow should be employed. Hervey used in addition adrenalin, digifoline and hot packs. Antistreptococci serum is a value in selected cases.
If the original focus is on the surface it could be cauterized with carbolic acid and neutralized with alcohol.

Frankenthal warns that the dentists can do much in prophylaxis of the disease by using caution when dealing with lower molars.
Case Reports

I

R.J.M. a male farmer age 29, entered Hosp. Aug. 35, 1930 complaining of swelling of the neck, swelling under tongue, difficulty in breathing and difficulty in swallowing. Onset: patient had sore throat for about four weeks, in about three weeks a swelling appeared which continued and became painful. On the side of the neck the swelling was of a hard wood like character. Later breathing became difficult and he had a constant choking sensation and swallowing became difficult. This persisted until incision was made and drainage was established.

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W.B.C. 11,700 Polys 79

Incision was made transversely in the left submental region and considerable pus was evacuated from the space deep to the mylohyoid. A small rubber dam drain inserted, removed on the third day and patient went home on the fifth day after an uneventful recovery.
II  W.J.A. male, mechanic age 28 entered Hosp. Oct. 28, 1928. Had had pain in the region of the right lower second molar for two days before onset of swelling in the neck, which began five days before being seen by the operating surgeon. He had been transported a distance of about thirty miles and when first seen had a hard brawny induration on the right side of the neck extending to the sternal angle, and a marked swelling in the floor of the mouth. He was dyspneic and had a marked dysphagia with saliva dribbling from the angles of the mouth. Speech was very difficult. He was hospitalized immediately and before the operating room was made ready the patient was getting cyanotic. The same incision as reported in case I was made under a small amount of novocain anesthetic. Pus was encountered under the floor of the mouth and a small drain left in place. T. 99 P. 37 R. 28 and labored.

Blood count was not done before operation. First two days considerable drainage wick removed on the third day. Patient dismissed on the eighth day in good condition.
H.T., male, laborer, age 25, complained of first pain and swelling at the angle of the jaw (right). Two days later this had increased to such an extent as to cause fixation of the neck. At about this time, the floor of the mouth became raised and the tongue swollen. Dysphagia, difficulty in talking were present but the constitutional symptoms were not marked until the time he was first seen.

Immediate hospitalization. P.90
T. 100    R. 20    W.B.C. 9,800

Incision in all respects similar to the first two was made. Recovery was rapid and he was dismissed on the sixth post operative day.

No original focus was found but three months later he had an impacted lower right wisdom removed. In this case and the first one novocain was used in conjunction with a little gas-oxygen mixture.
IV

A. K. male, student age 19 had a right lower third molar removed by a good, careful dentist who found the entire ramus of the mandible, clear to the coronoid process, involved in a necrotic process. He developed in the course of three days to a week the symptoms of a fulminating Ludwig's Angina.

The same incision was made as in the previous cases. Recovery was delayed by the process in the ramus of the mandible. Wound was irrigated with saline t.i.d. for a week. In the course of three weeks the boy recovered.

This case under the combined local and general anesthetic mentioned in the former case.

These first four cases were taken from the practice of the writer's father, and were seen while a student.
M.C. a male, student, age 20 thinks he got a straw stuck in the gingivolingual sulcus on the right side opposite the second molar. Had a feeling of discomfort for one day then the area started to swell. In one week there was a hard swelling of the neck of that side and a sublingual swelling which pushed the tongue to the opposite side. Marked dysphagia and some dyspnoea were present. On entrance to the hospital a puncture at the base of the tongue and behind the anterior pillar on that side was made with no results.

He came in at night and after the puncture had made he was placed in bed with hot packs to the neck. He awoke in the morning with much pus in the mouth. The swelling subsided for a time but in three days again became marked. Incision under local was made in the right gingivolingual sulcus and a foul exudate was evacuated from which pneumococcus, a fusiform bacillus, and a spirillum were taken in the lab. Recovery was then uneventful. An infected molar (lower) on that side was extracted two months later and possibly the source of the infection.
Conclusions:

Ludwig's Angina is a severe infection in the space deep to the mylohyoid muscle.

The predominate cause is infection of a lower molar.

There is no specific organism responsible for the infection.

Treatment should be instituted early, in fact as soon as the diagnosis is made.

Such treatment should be radical and consist of incision through the mylohyoid muscle.

The wound should be drained for a period depending on developments, by some loose fitting material to keep the tract open.
BIBLIOGRAPHY


7- Blair, Vilray, P. Surgery & Diseases of Mouth & Jaws C. V. Mosby Co., St. Louis, 1917

8- Blair-Ivy Essentials of Oral Surgery Page 171


19- Holms, Ruptured Aneurysm Mediastinum and deep cervical Facia with Symptoms of Ludwig's Angina. Laryngoscope 26:1246- October 1916

20- Jackson Coates Diseases of Nose & Throat Page 126 1922


BIBLIOGRAPHY CONTINUED

24- Molt, Wm. F. Ludwig's Angina J. Ind. M. A. 15:196-200 June 1922


27- Parker, R. Wm. Remarks on Cellulitis of the Neck—Ludwig's Angina Lancet 2:570-1819


31- Sautier, C. M. Ludwig's Angina and Mediastinal Abscess following tonsilectomy; operation; recovery. J. A. M. A. 87:1831- Nov. 1926


33- Stein, E. J. Ludwig's Angina, Laryngoscope 39:672-673 Oct 1929

BIBLIOGRAPHY CONTINUED


36- Voorhes, I. W. Diffuse Infection of Neck Following Tooth Extraction J. Oral N. Y. 1:84-86 Sept 1922
