STATED MEETING, HELD NOVEMBER 4, 1907.

The President, Dr. John B. Roberts, in the Chair.

ESOPHAGOTOMY FOR IMPACTED COIN IN A NINETEEN MONTHS OLD INFANT.

Dr. Chas. F. Nassau presented a child who had been referred to him by Dr. Bridgett of West Philadelphia, on account of a suspicion that the child had swallowed a five-cent piece. She was not particularly ill for a while, but she could take only liquid food, even soft potato being vomited. Shortly the child had a quite serious gastro-intestinal upset, as it was supposed. When she was finally brought to Dr. Nassau he had an X-ray plate made, which showed the nickel piece lodged in the esophagus just above the suprasternal notch. On the following day, the twelfth after the swallowing of the nickel the child was admitted to St. Joseph's Hospital. Dr. Nassau passed esophageal forceps readily down the esophagus and could feel them strike a metallic object, but he was not able, with some little pains, to grasp this object. Considering the length of time this foreign body had been imbedded in the child's esophagus he thought it wisest to do an esophagotomy rather than try to force the nickel out. The operation consumed but fourteen minutes, and there was little trouble about the operation. There were no vessels requiring ligation; the wound was closed after the introduction of a gauze drain, without suturing the esophagus. The nickel lay in the anterior portion of the esophagus with the edge turned up a little toward the left and spanning it tightly, as though in a pocket. There was no leakage of either fluids or food and the child made a perfectly uninterrupted recovery.

Dr. Nassau also referred to a second recent case. The patient, a physician, swallowed a set of caps and pivot teeth at 3 A.M., and the operation was performed at about 9.30 following, there being no question about attempting to remove them by any other method. The patient had a rather stoutish neck which it was impossible to stretch out quite as desired. The operation was performed in a country house and took about twenty minutes. This esophagus was sutured and the entire lower half of the

wound was drained. The pack was inserted for about 4½ inches, which Dr. Nassau afterwards considered extremely wise as the wound was badly infected within two days. This wound is now very well cleaned up, there being healthy granulation and no leakage. Any kind of liquid food can be swallowed without pain.

Dr. William J. Taylor stated that in 1900 he operated upon a child of 16 months who had swallowed a good sized metal clip. This was in the child's throat for seven months. It was a nursing baby and therefore had swallowed its milk fairly well, but it was absolutely impossible for it to swallow solid food. An X-ray picture was taken soon after the swallowing occurred but unfortunately the child was not etherized and the plate was a failure. When the child was referred to Dr. Taylor he had Dr. Leonard take a skiagraph, which gave a most excellent view of this clip which was open. The child was etherized, and but slight efforts were made to reach the object with instruments. Dr. Taylor agrees with Dr. Nassau that the only safe plan when foreign bodies have been in the esophagus a long time is to do an esophagotomy. This was done in his case and the child made a very satisfactory recovery and is now a strong healthy boy with no stricture of his esophagus and has had no difficulty whatever in swallowing. Dr. Taylor showed the corresponding clip to that which had been swallowed and called attention to its nickel-plated condition, stating that the nickel-plating of the clip which had been swallowed had been absorbed while the clip was in the child's throat.

Dr. Taylor desired to repeat his statement that he thought it always safest, after a foreign body had been for some time imbedded in the esophagus, to do an immediate esophagotomy rather than try to remove the object with a coin catcher or forceps.

Dr. John H. Gibbon considered Dr. Taylor's attitude rather radical. He referred to a case in which he had removed an ordinary campaign button which had been in the esophagus for eleven days. This patient made a good recovery. He thought one had to be guided entirely by the character of the body in the esophagus and by the symptoms. Esophagotomy carries with it a certain amount of danger especially from pneumonia, and a case in which an esophagotomy is done in the presence of ulcers is always in danger of a pneumonia. He considered it wise to make an endeavor to remove the foreign body unless the evidence

goes to show that such an attempt would be dangerous. He did not believe any rule could be laid down as to the performance of an esophagotomy after the foreign body had remained any certain time in the esophagus, especially when the foreign body was smooth or round.

Dr. John B. Roberts mentioned the case of an infant who had swallowed a jackstone. The patient was referred to him last spring, a day or two after the accident. It had been seen by other physicians in the meantime. Dr. Roberts tried unsuccessfully to get the jackstone out by the mouth. Finally an esophagotomy was done, and unfortunately, on account of not being able to get a guide into the esophagus he made a slight puncture in the trachea. He removed a six-ended jackstone from the child's esophagus. The patient did fairly well for a few days but the wound finally became very septic and she died of a capillary bronchitis. Dr. Roberts thought that if he had seen the patient earlier and had resisted the temptation to attempt removal through the mouth, and done esophagotomy earlier he might have had a better result.

Last winter, with an ordinary coin catcher he succeeded in removing a coin from the esophagus, after it had been swallowed but a few hours.

Dr. A. C. Wood agreed with Dr. Gibbon that some judgment should be exercised in adapting the method of removal to the kind of body, as well as to the time that had elapsed since it was swallowed. An irregular object, such as a jackstone, would cause ulceration more rapidly than one that was smooth and round such as a coin. There is good reason to believe that it would have been dangerous to attempt to fish out the clip shown by Dr. Taylor.

He referred to his experience in five cases in which jackstones had been swallowed. In two of these the jackstone was removed by means of a gastrotomy, after efforts at removing it through the mouth failed. The stones were brought into the stomach and removed without serious consequences, the children making normal recoveries. He had tried various esophageal forceps without success in three cases in which he was able barely to touch the jackstone with the tip of the index finger. By using this finger as a guide and employing a hook like a tenaculum, bent to the proper curve, he was able in these three cases to get

the body up without difficulty and without danger to the child. He considers esophagotomy such a serious operation in itself that it should be resorted to only when all other appropriate means have failed.

Dr. John H. Jopson recalled several cases in this connection. In one case he was able to extract a jackstone by passing an English catheter alongside of it and withdrawing catheter and jackstone together. He has never had much success with the esophageal forceps in children. He referred to an unfortunate case at the Children's Hospital this Spring where a nickel had been imbedded in the esophagus for several days. The X-rays located it in the neighborhood of the cricoid cartilage and an attempt at extraction was made with some new instruments. The coin catcher was too large and almost became impacted. Jopson feared it would be necessary to do an esophagotomy, but on the following day his assistant brought a coin catcher from the University Hospital and with this the coin was brought out with the first effort. Dr. Jopson therefore considers the shapes and sizes of coin catchers important. This child was taken home that night against his advice, and had an attack from which it died in a few hours. The cause of death was not determined, but there may have occurred a pressure perforation of the esophagus or an edema of the glottis.

Dr. John H. Gibbon also referred to a case which was under his care at the Pennsylvania Hospital last winter. The patient was a child four or five years of age who had swallowed a jackstone. Numerous attempts had been made at removal of the stone before her admission to the hospital. Dr. Gibbon thought he could feel the stone with the forceps but was unable to remove it. The child was anesthetized and the stone seen distinctly through the fluoroscope. This case illustrates well the advantage of the fluoroscope. This stone and forceps could be distinctly watched throughout the removal: the forceps grasped first the smooth end of the jackstone and slipped off, the stone was then turned round and the knobbed end of the jackstone caught. This was one of the most satisfactory uses of the X-ray in the removal of foreign bodies that Dr. Gibbon has ever experienced. This child developed a pneumonia from which she died two or three days after the removal of the stone.

Dr. Charles L. Leonard (by invitation) referred to a case

sent to him from North Carolina for examination by the X-ray. The patient had been X-rayed but no foreign body found. He discovered a coin in the esophagus, which was finally removed with the coin catcher some 18 months after it had been swallowed. This was in a boy of twelve years. Dr. Leonard also stated that it was not now necessary to make an X-ray examination under ether, because these examinations could now be made with exposures of ten seconds, or less.

Dr. Charles F. Nassau, in closing, said there is no question whatever that when given a foreign body, either smooth or a jackstone, attempts may be made to extract the body. With this baby he made reasonable efforts after touching the object with the esophageal forceps, which he had no difficulty in introducing. He thinks a difference should be made between bodies which have been for a comparatively short time and those which have been in for months, for where an object has been in only a short time infection there is severe; if it had been there for a long time Nature will have done, as she does everywhere, build a wall round that body which will protect the tissues outside from the extension of infection due to reasonable manipulation. He does not believe from his small experience that esophagotomy is such a serious operation as one would suppose. In the case of this child he cut no vessels, while in that of the heavily built man with the plate of teeth in his esophagus, Dr. Nassau tied the inferior thyroid and one small branch running anteriorly from the vessel and put only two ligatures in the wound. When he opened this esophagus there was a gush of purulent material, and of course with this condition present it would have been death to his patient to have attempted to remove the object by any other means. In neither of his cases could the object be felt by a finger in the throat, they were both lodged in the esophagus. They could, however, be touched with the forceps. As to the use of a guide Dr. Nassau said that after feeling the foreign body he took out the forceps, made the incision as far as the esophagus, and then reintroduced the forceps in the case of the man, but not in the baby. The prongs and edges of the plate of teeth had imbedded themselves and sepsis was beginning at a serious rate, and he found the forceps a great aid in this condition. The patient's temperature went up that night to 104°, but on the fourth day was normal.

SIGMOID DIVERTICULITIS (MESOSIGMOIDITIS) IN A CHILD.

Dr. Astley Paston Cooper Ashhurst presented a boy aged seven years and nine months, whom he had seen on the evening of July 18, 1906. In the absence of Dr. Hutchinson, to whom he was indebted for the privilege of operating and of reporting the operation, he was called to the Children's Hospital to see the patient, who had just been admitted with the diagnosis of appendicitis. The patient's family history was negative; he had had measles and mumps, but not recently. For the past two weeks he had had pains in the abdomen, chiefly around the umbilicus, and not very severe until three days before admission. Then he lay on the bed, doubled up as if with cramps, but did not vomit until the day he was first seen by Dr. Ashhurst. His mother said that his bowels had been opened several times daily. The pain was said to be paroxysmal, becoming very severe at times. On admission, at 9 P.M., the temperature was 101.4° F., pulse 128, respirations 32 per minute. The abdomen was held very rigid throughout, but it seemed to be a voluntary rigidity, and there did not appear to be diffuse peritonitis. There was retention of urine, the dulness due to the distended bladder being evident on percussion in the hypogastric region. The urine was drawn twice by catheter, but subsequently was voided spontaneously.

The presence of appendicitis was excluded after the first examination, but no satisfactory diagnosis was made. Rectal examination was negative. It was decided to await the development of more certain symptoms before undertaking an exploratory operation. The bowels were opened only by enema. No purges were given at this time.

Not until the third day after admission was palpation of the abdomen entirely satisfactory. It was now possible to feel a mass in the left iliac fossa. This mass was firm and tender on palpation, and seemed attached to the iliac bone in the neighborhood of the left sacro-iliac synchondrosis. The mass extended nearly half way from Poupart's ligament to the umbilicus. It was dull on deep percussion, and did not seem to be in close contact with the anterior abdominal wall. The rest of the abdomen was flaccid, and there was no tenderness except on firm pressure over the tumor. The tumor could not be reached by

the finger in the rectum, and rectal examination was in no way painful. No polyp was detected. The question of diagnosis was still undetermined, but lay between sarcoma of the sigmoid and an inflammatory mass, which latter, it was thought, might have been caused by a previous attack of appendicitis. Psoas abscess was excluded on account of the absence of all bone lesions, and because of the presence of early symptoms of intraperitoneal irritation. Iliac abscess, of traumatic or tuberculous origin, was also excluded for the latter reason.

The child was seen by various members of the staff, both surgical and medical, but no positive diagnosis was suggested. Purges and enemata were administered until the possibility of fæcal impaction was absolutely excluded. The leucocyte count was 6,400 the day after admission. One week later 7,200.

Exploratory laparotomy was done on July 27, nine days after admission. An incision, nearly three inches in length, was made in the left rectus muscle above Poupart's ligament. There was much bleeding from the abdominal wall, and the transversalis fascia and peritoneum were much thickened. On opening the peritoneum there escaped several drachms of clear serous fluid, with no odor. Its appearance suggested the possibility of a rupture of the bladder, with the extravasated urine encapsulated by adhesions. There were light inflammatory adhesions between the outer layer of the mesosigmoid, and the parietal peritoneum. A gauze pack was introduced to exclude the small intestines from the field of operation, and in doing this there was detected in the mesosigmoid a dense mass, nodular, stony, hard in places. The sigmoid with its attached mesentery was then partially delivered through the wound, the mesosigmoid turning on its attachment to the posterior abdominal wall like a door on its hinges. The tumor in the mesosigmoid was the size of a goose egg, and several enlarged lymph nodes were seen on its surface, just beneath the serous covering. The sigmoid itself was in no way obstructed, but passed over the surface of the growth, and was normal to all appearances. No tubercles could be seen on the tumor, the sigmoid, the parietal peritoneum, or elsewhere in the field of operation. The tumor was of such cartilaginous hardness in places that it seemed impossible for it to be merely inflammatory in nature. It was thought to be a retroperitoneal sarcoma, and as its removal would have required resection of the

sigmoid from the level of the iliac crest down into the true pelvis, all thought of radical operation was abandoned. One enlarged gland, close to the mesenteric border of the sigmoid, was removed from the surface of the tumor beneath the external layer of the mesosigmoid; the incision in the mesosigmoid was sutured; and the abdominal wound was closed in layers. The time of the operation was forty minutes. The convalescence was uneventful. The wound was dressed at the end of a week, the last sutures were removed three days later, and on the twelfth day the patient was allowed out of bed. He was discharged August 11, 1906. An examination of the blood, made August 1st, five days after the operation, showed that the leucocytes numbered 13,200, and that the hæmoglobin was 55 per cent. On the same day Dr. C. Y. White reported that microscopical examination of the gland removed at operation showed marked inflammatory exudate throughout its structure. No evidence of tuberculosis could be detected.

The patient was seen again in the Dispensary three weeks after operation. The wound was firmly healed, but the tumor seemed to be nearer the median line of the abdomen, and was not apparently attached to the left iliac bone as before the operation. His bowels had been opened normally, without enema or purge, twice daily since leaving the hospital. The patient's mother was informed that an inoperable tumor had been found, and a gloomy prognosis was given.

On November 17, 1906, about three months and a half after the operation, Dr. Ashhurst examined the patient at his home. He was playing around the streets, and had been in excellent health. His bowels opened normally, his appetite was good, and he never had any pain. Careful abdominal examination failed to reveal any evidences of the tumor. He had seen the child at intervals since then, and presented him to the Academy in perfect health, and without the slightest evidence of tumor.

Dr. Ashhurst said that until within the past year, very little surgical attention had been devoted to inflammatory lesions of the sigmoid and its mesocolon. During that time a large number of contributions have appeared, and the pathology and nomenclature of these affections are becoming better understood. The literature of acquired intestinal diverticula, up to 1904, has been admirably summarized by Dr. Edwin Beer of New York, and

within the past year the diagnosis and treatment of inflammatory affections of the sigmoid have been discussed by Brewer, Lejars, Mayo, Monsarrat, Patel, Ries, Rosenheim, Sieur, and others. The lesions reported by these authors may be classified as follows:

1. Sigmoiditis—inflammatory hyperplasia of the walls of the sigmoid converting it into a rigid tube, and usually causing a certain amount of obstruction.

2. Perisigmoiditis—suppuration, usually localized, due in most cases to perforation of a sigmoid diverticulum. Appendicitis is still recognized as a possible cause of perisigmoiditis.[1]

3. Mesosigmoiditis—which, in the patients reported by Ries, was characterized by the presence of cicatricial bands in the mesosigmoid, leading in one case to volvulus, these bands being the result of previous more or less acute inflammatory changes.

Most of the cases reported have belonged to one of the former classes, a majority probably being characterized by perisigmoid suppuration. It seems probable that in this case, described by the term mesosigmoiditis, the original lesion was a diverticulitis within the layers of the mesosigmoid. It is well known that diverticula occur in this situation, as well as on the free border of the sigmoid; and though their presence in any but adults is denied by many writers, other authorities acknowledge the existence of congenital diverticula. In none of the reported cases, however, so far as he had been able to ascertain, was the patient below the age of puberty;[2] and in none has there been such a marked tumor of the mesosigmoid, with so little perisigmoiditis. Dr. Deaver, however, had informed him that he had operated on a patient (an adult) in whom the pathological lesions considerably resembled those in the patient now reported; except that in Dr. Deaver's patient the mass in the mesosigmoid was much softer, the sigmoid itself was quite strictured, and when the bowel was opened an ulcerated spot (not a diverticulum) was found at its mesenteric attachment.

The treatment to be adopted depends very much on whether the condition is recognized as a purely inflammatory one, or

[1] Perhaps the term pseudo-sigmoiditis might be employed to describe inflammatory lesion in the neighborhood of the sigmoid, caused by primary disease of the appendix, ovary, or Fallopian tube.

[2] Patel, in a paper published since the above was written, refers to a case in a girl of 10 years, reported by Walcha.

whether, as in most of the earlier cases, it is considered malignant. In the latter case resection will be adopted for the operable cases; and the inoperable cases will be treated by either colostomy, enteroanastomosis, or exclusion if there is obstruction, or the abdomen will be closed, as in the present case, when no obstruction exists. If the presence of pus, or the history of early inflammatory symptoms, on which as a diagnostic point Lejars lays so much stress, make it seem probable that the condition is inflammatory, it will probably be best merely to drain the purulent focus and release such adhesions as obstruct the lumen of the sigmoid.

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Dr. A. C. Wood considers the pathology of these inflammatory lesions about the sigmoid more complex than might be supposed at first thought. They are not all secondary to diverticula; probably but a small minority are due to this cause. He has read of cases that were due to perforation of the sigmoid by foreign bodies; in one instance a pin had passed through the wall of the bowel, causing an abscess, and in another fragments of straw had in like manner perforated the bowel. Cases are reported in which the epiploic appendages were involved in these inflammatory swellings. Although the case reported by Dr. Ashhurst is the youngest he has heard of, he believes it is generally admitted that these diverticula may be either congenital or acquired, and if congenital, there is no reason why they may not cause trouble in early life. He does not consider the explanation that the diverticula result from constipation and distention of the bowel with protrusion of pouches of mucous membrane through the muscle fibres a satisfactory one.

Dr. Astley P. C. Ashhurst, in closing, said that in his case the diagnosis was of course largely conjectural; he thought

however, that the mass certainly was one of enlarged glands, but he believes that if these glands had been simply tuberculous in character, which he considers a rarer condition in the mesosigmoid than the presence of diverticulum, there would have been symptoms of tuberculous disease and the course of the case would not have been so favorable. Although the condition is a rare one he sees no reason why this should not be considered a case of diverticulitis.

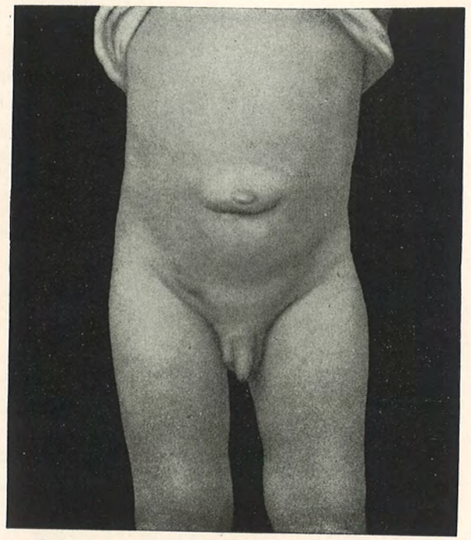
RADICAL CURE OF UMBILICAL HERNIA IN A CHILD WITH PRESERVATION OF THE NAVEL.

DR. ASHHURST reported the case of Thomas S., aged two and a half years, who had suffered since infancy with an umbilical hernia, which on admission was the size of an English walnut, and was easily reducible. The ring admitted the little finger. There was also a right inguinal hernia.

Having seen the suggestion that the navel be preserved in operating on children, especially boys, for the radical cure of umbilical hernia, he determined, at the risk of being thought to do a complicated operation where a simple would suffice, to attempt such an operation in this case. For the privilege of operating and of reporting the operation, he was indebted to Dr. Hodge, in whose service at the Children's Hospital the patient was treated.

The operation was done July 25, 1907. A crescentic incision was made below and surrounding the navel, down to the sheaths of the recti muscles. The flap of skin and subcutaneous fat thus outlined was dissected upwards, for an inch or more above the navel, the hernial sac being opened just beneath the umbilicus. The flap containing the navel was then turned upwards, and the sheath of the rectus muscle on each side was opened transversely at the level of the ring. The sheaths with the intervening linea alba were then dissected free from the underlying transversalis fascia and peritoneum. Then with three mattress sutures of chromic catgut the aponeurosis below the ring was drawn upwards into the slit between the transversalis fascia beneath and the sheath of the recti muscles superficially. The flap of aponeurosis on the thoracic side of the hernial ring was then sutured (with continuous stitches of chromic gut) to the sheaths of the recti muscles below, thus interposing, as in the usual overlapping

FIG. 1.



Result of operation for umbilical hernia with preservation of the navel.

operation, two layers of aponeurosis between the peritoneal cavity and the subcutaneous tissues. The skin flap was then sutured back in place, and a small catgut drain was introduced beneath it at one angle of the incision, because the absence of the hernia and the overlapping of the aponeurosis had made the skin flap somewhat redundant, and it was feared that some serum might collect beneath it were no drain employed. This drain was absorbed, having fulfilled its purpose, before the first dressing of the wound, when union was found firm throughout. The operation took only twenty minutes to do, and as the scar fades away in the natural creases of the abdomen it will be barely possible to tell that any operation has been done (Fig. 1). The boy at least will not be an object of ridicule among his companions in bathing, etc.

The inguinal hernia was operated on at the same sitting. It was a hernia into a patulous processus vaginalis testis, and the Bassini operation was done. Both scars are now perfectly firm, and the boy is in excellent health.

DR. JOHN H. JOPSON said that in 1906 he had seen Dr. James Stone of Boston operate for umbilical hernia in a child at the Boston Children's Hospital, and Dr. Stone advanced the same reasons for preserving the umbilicus that Dr. Ashhrust had mentioned. He did not do as Dr. Ashhurst described, but made a linear vertical incision. Dr Jopson repeated this operation on a child at the Presbyterian Hospital last winter. Referring to Dr. Ashhurst's first case it seemed to Dr. Jopson that the diagnosis of diverticulum was only a matter of conjecture, and that in the absence of an opportunity for resection and examination of the tumor and as there were undoubtedly enlarged glands in the mesentery it might just as well have been considered a case of enlarged glands in the mesosigmoid as the rare condition of diverticulitis.

LUDWIG'S ANGINA.

AN ANATOMICAL, CLINICAL AND STATISTICAL STUDY

BY T. TURNER THOMAS, M.D.,

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Ludwig in 1836, described a condition which he considered a morbid entity, and which since that time has been designated, more or less universally, as Angina Ludovici. Various attempts have been made to establish it upon a definite pathological basis, but the results of none of these can be said to have been generally accepted. That it is an infection there can be no doubt, but the character of the infection if it has a special character, has never been decided. That it is more rapidly fatal than similar infections occurring in other parts of the neck than the submaxillary region, is amply proved by the recorded cases, but why or how it acquires so dangerous a character, has never been clearly demonstrated. It is well known that certain cases assume a grave aspect and threaten or take the life of the patient in twelve to twenty-four hours, while others begin and continue as a comparatively mild affection for days and then suddenly assume an alarming character. That its general course and symptoms are typical and essentially constant is convincingly shown by the numerous cases that have been reported. Yet many have been and some are still being reported, which should not be designated by this term. The following case led the writer to make a study of the subject.

W. W., male, age 32 years, machinist, admitted to drunk ward of the Philadelphia Hospital, August 10, 1903. On admission the temperature was 98, pulse 110, respiration 30. Has been drinking for about a month. Is nervous and has marked tremors of the hands and tongue. He cannot eat or sleep. The heart is rapid but the sounds are good, and there are no murmurs.

He has no hallucinations, and is well nourished. He complains of a small, painful swelling under the right side of the lower jaw which has been there for about a week. On August 13 he was transferred to the surgical ward, in the service of Dr. A. C. Wood, to whom the writer is indebted for the privilege of reporting the case. Temperature 98.2, pulse 78, respiration 22. The swelling is increasing in size. The pain keeps him awake at night and prevents him from taking his nourishment. Incision made in the submaxillary region, the index finger being introduced its entire length without evacuating any pus. August 14th swelling is increasing rapidly, is hard, non-fluctuating, and involves the whole under side of the jaw. Speech is difficult, and he is having such difficulty in swallowing that he is being fed with a spoon. Temperature 101, pulse 100, respiration 26. General condition otherwise good. August 15, at 8 P.M., he was cyanotic and respiration was very difficult. Tongue swollen. Oxygen inhalations given for a time with some relief. Then he became rapidly worse. Pulse intermittent, rapid and weak. At 10 P.M. tracheotomy was performed by the resident in charge, Dr. Speese. Incision below the cricoid cartilage. Profuse bleeding from the veins in front of the trachea. Surrounding tissues very œdematous. From the time the trachea was opened the patient's condition rapidly became worse, and although he breathed through the tube, he could not be kept alive by artificial respiration, which was continued for about fifteen or twenty minutes.

Autopsy—Pathological Diagnosis.—Œdema of the glottis; unilateral interstitial nephritis; hemorrhagic infiltration of intestinal mucosa. The tissues about the glottis and epiglottis are intensely swollen. This swelling is so extensive about the glottis that only a chink, about 2 mm. in breadth and 6 mm. in length of the glottis remains.

The writer regards this case as a typical Ludwig's Angina. The following case was reported, September 4, 1905, before the North West Medical Society of Philadelphia, as a "Gun Shot Wound of the Lower Jaw, followed by Submaxillary Cellulitis, simulating Ludwig's Angina." Since then a study of the literature has shown other cases, reported as Ludwig's Anginas, which were essentially of the same

type. The writer considers that this grade of infection in this region has every dangerous characteristic, indeed somewhat exaggerated, of a typical Ludwig's Angina. The reader is referred to the later discussion on etiology and pathology, for the writer's reasons for including it here as an example of this disease.

J. W., colored, age 31 years, admitted to the Philadelphia Hospital, August 9, 1905, in the service of Dr. A. C. Wood, with whose permission the writer reports the case. His general health and strength were excellent.

On August 8th, in a quarrel, the patient was shot twice by a revolver in the hands of a companion, who stood in front of him, and about five or six feet away.

There are three wounds of the face, one of which is a well-rounded and perforating wound of the cheek, about three-eighths inch in diameter, just to the right of the symphysis. A second wound with irregular edges is situated on the right cheek, about one inch in front of the lobule of the ear. A fragment of a bullet was removed from this wound. The third perforation, which was so insignificant and covered by stubby beard that it was not discovered for a few days, is shaped like the first, and is situated on the left cheek about two and a half inches posterior to the angle of the mouth. The probe enters this wound for about two inches, when it strikes what at first was thought to be the ramus of the jaw. A skiagraph later showed a bullet lodged in the tissues in about the situation of this opening. At first the patient did not complain of this wound, and it was then thought that the two on the right side were produced by the two bullets. It seems evident now that they were due to a single bullet which entered near the symphysis, struck the jaw, splintered it, and was divided, one fragment glancing off and producing the wound in the right cheek near the lobule of the ear. Both wounds of the right cheek met within the mouth at the injured portion of the jaw.

The tissues of the interior of the mouth, internal and external to the jaw, are intensely swollen, particularly internal to the jaw in the floor of the mouth. The tongue almost fills the mouth and interferes with normal respiration. Speech and deglutition

are disturbed. There are four teeth missing in the lower jaw in the right molar region. The patient says the teeth were not missing before the shooting. In the space corresponding to the missing teeth the alveolar border of the lower jaw is splintered, the loose fragments being removed with forceps. There is a complete fracture of the lower jaw on the right side about an inch anterior to the ascending ramus. The wounds were all washed out and packed with gauze, dressings applied, and a cardboard cup was fitted to the jaw and held by a Barton's bandage.

On the following day the patient's condition became alarming on account of the difficulty in breathing. The face was more swollen, particularly in the submaxillary region. The tongue and the floor of the mouth were more swollen than on the preceding day, and the tissues in the floor of the mouth were more brawny to the feel. The patient was etherized and the two wounds on the right side of the face were enlarged into the mouth. All loose fragments of bone and soft tissue were removed and the wounded tissues irrigated with boric solution. An incision about two and a half inches long was made parallel with the lower jaw and about midway between the hyoid bone and jaw. This wound was deepened until the finger was close to the mucous membrane of the floor of the mouth in the region of the damaged portion of the lower jaw. Irrigation and dressing as before. Temperature 101.3°, pulse 128.

On the following day, August 11th, his condition had improved slightly, but the swelling and temperature were about the same. Respiration, deglutition and speech were still disturbed. On examining the region of the injury to the jaw, the wound in the floor of the mouth was found covered with gangrenous sloughing tissue, and the odor was very foul. The wounds in the lip and cheek leading away from this region were discharging foul pus. A mouth gag was introduced on the opposite side of the mouth and the tongue held away from the infected area, thus exposing it. After clearing away all shreds of gangrenous tissue and irrigating with boric solution, the infected surface was cauterized with pure carbolic acid, which was neutralized at once by applications of alcohol. The patient was placed in charge of a special nurse, who cleansed the infected region every half hour with peroxide of hydrogen and boric solution.

On the following day a marked improvement was noticeable. The swelling was evidently decreasing, the patient could talk, swallow and breathe better, and said that he felt much better. In the few succeeding days the temperature fell to normal. The discharge was still copious and offensive. The septic condition soon subsided and the case resolved itself into one of healing wounds of the face and neck and fracture of the lower jaw, which later united.

History.—Parker, in 1879, published an interesting historical review of this condition as recorded before the appearance of Ludwig's paper, with particular reference to the cases occurring in England. He gives some details of a case referred to by Auretius which seem to have been those of a Ludwig's Angina. He called the condition, "cynanche." Paulus Aegineta spoke of a somewhat similar condition, which he called "paracynanche." Many of the older authors, both Greek and Arab, including Hippocrates, Galen, Celsus, Aurelianus, Rhases and others, had described the disease. Dr. Fothergill gave an account of "Putrid Sore Throat" (1739–1746), which appeared to have some of the characteristics of Ludwig's Angina. He also gave an historical review of what is believed to have been the same disease. Dr. Kirkland in 1786, Dr. Wells in 1809, and others reported cases of this type.

It remained, however, for Ludwig to present the first accurate description of this dangerous condition, which he called "gangrenous induration of the neck." Cameror, in the following year was the first to apply to it the name, Ludwig's Angina. Following the appearance of Ludwig's paper considerable interest was manifested and an increasing number of cases were reported. Probably, greater interest has never been shown during any one period, than that which was bestowed on it by the French Surgical Society, in 1892. Several successive meetings were devoted to it and many cases cited by those present. There was a marked difference of opinion manifested, which culminated in a division of

the members into two parties, one being led by Nelaton, the other by Delorme. Nelaton took the stand that Ludwig's Angina should not be recognized as a separate disease, and was instrumental in having resolutions to this effect passed by the society. At the following meeting Delorme caused this action to be reconsidered and Ludwig's Angina to be given its proper place in surgical pathology. In the following year Leterrier published a thesis in which he reported 27 cases collected from the literature and communicated four new ones, three of Delorme's and one of his own. The chief object of his paper appeared to be to support the position of his teacher, Delorme, who contended that Ludwig's Angina was primarily a sublingual phlegmon.

In the same year, 1893, Poulsen published the results of a study of 530 abscesses of the neck collected from hospital statistics. In 1886, he had presented a paper in which he reported his observations on a series of lime injections under the deep fascia of the neck, to prove the existence of communicating channels of loose connective tissue between the various adjacent interfascial spaces. In his second paper he attempted to show that infections tended to follow these channels and to invade the various spaces, successively. His explanation of the progress of the infectious process in a Ludwig's Angina will be taken up later in the discussion of the etiology and pathology of this disease.

In 1895, Semon's paper appeared, in which he maintained that acute septic inflammations of the throat and Ludwig's Angina were pathologically identical, and should be included together as one group of diseases, thus eliminating Ludwig's Angina as a separate disease. Since that time nothing new has been offered on this subject so far as the writer can learn.

Etiology and Pathology.—Although fairly authentic cases were recorded before, practically, nothing was presented in the literature to establish the cause and nature of this condition, until Ludwig's paper appeared. Since that time many cases have been reported and much has been written, which is of value in clearing up the obscurities sur-

rounding Ludwig's Angina. Yet its etiology and pathology still remain obscure. In the writer's opinion, one of the basic causes of confusion lies in the obscurity associated with the cause of death, in connection with which, the chief question is as to whether it results from septic intoxication or from invasion of the air passages. Probably, both conditions are always present to some degree, in typical cases; but the relative importance of each has never been established. If septic intoxication is the essential cause of death, then the especially high mortality of this condition is to be explained by the presence of a rare and especially virulent infection. If invasion of the respiratory tract is the dangerous feature, peculiar to this condition, then the mortality is to be explained by extension of the phlegmonous inflammation to the larynx and in some cases to the lungs. Upon the solution of this question depends, in the writer's opinion, the explanation of the etiology and pathology of Ludwig's Angina.

Ludwig suggested that it was epidemic in its nature, that it was allied to erysipelas and that it was a true morbid entity. Every one of these suggestions has been supported and combatted vigorously by many different authorities and it may fairly be said that they remain unsettled up to the present time. Tissier, Roser, and Chabri, for example, agree with Ludwig, as to its being a morbid entity. On the other hand, Boehler, who collected and studied 35 cases, refused to accept this view and tended to suppress the name of Ludwig's Angina. v. Thadden gave to it the name of "submaxillary bubo," while Chantemesse considered it a true erysipelas of the larynx. Roser believed that the disease began in the submaxillary salivary gland. This theory has not been borne out by the post-mortem evidence which has been accumulated. It will not be profitable to discuss here more than a few of the theories which have been offered as to the etiology and pathology of this condition, and it is particularly, to the later authorities that the writer will confine his attention.

As already indicated the investigations have followed two distinct channels; the bacteriological, which attempt to prove that a particular type of infection and therefore a septic intoxication is responsible for the condition; and the anatomical, which try to show that the condition is due to the particular location of the infection and its peculiar opportunities for dangerous extension.

Influence of Septic Intoxication.—Definite and positive convictions on the relative importance of septic intoxication, can not be reached without difficulty. In studying this phase of the subject, first importance should be given to the bacteriological findings. A search of the literature has shown the following cases in which different bacteria were found and reported.

Delorme, staphylococcus in one case and streptococcus in another; Leterrier, undetermined bacillus in one; Macaigne and Vanverts, pneumococcus predominating, with streptococcus and staphylococcus in one; Lockwood, streptococcus, staphylococcus and bacillus of malignant oedema in one of his own cases and in another, streptococcus, cocci and diplococci. In Gibson's case he also found the streptococcus; Biedert and Robertson, streptococcus in one; Gasser, streptococcus and bacillus coli communis in one; Ross, streptococcus and staphylococcus in one; Davis, streptococcus alone in two cases, and streptococcus and staphylococcus in a third; Ombredanne and Keim, streptococcus and staphylococcus in one; Humphrey, pneumococcus alone in one; Duplay, staphylococcus in one; Chantemesse and Widal, streptococcus in one; Magnal, streptococcus in one.

It will thus be seen that of the 18 cases, the streptococcus was found alone in 6 cases; the streptococcus associated with other organisms in 8; the staphylococcus alone in 2; the pneumococcus alone in one; and an undetermined bacillus in one.

The fact that stands out most prominently in this group of cases is that the streptococcus was present in almost all, either alone or associated with other bacteria. That it may be present in some cases in which the investigation has failed

to show it, may be inferred from the fact that Lockwood, by different methods, found the streptococcus in Gibson's case, although Cameron reported that he could find "no specific microörganisms in the tissues." In all the writer's collection of cases, the inflammation of the connective tissues has appeared to be of a severe type, and in a considerable number a gangrenous or fetid process has been present. The inference to be drawn from these facts is that a severe septic infection and a corresponding grade of septic intoxication has been encountered. Yet in many cases the constitutional symptoms have been only moderate or very mild. Even if they were severe in all, this would not show that they were the cause of the high mortality, since the same infections occurring in other parts of the body, giving as severe local and constitutional symptoms, do not produce the same death rate as does Ludwig's Angina. Since the existence of a special infection, capable of explaining the high mortality, has been searched for, carefully, by qualified investigators without success in a fairly large number of cases (probably many more than the writer has found record of), we may assume with some confidence, that none such is present. The clinical as well as the post mortem evidence, so far accumulated, is decidedly against the existence of such a cause; while the evidence in favor of ordinary severe types of infection, particularly, the streptococcus is very strong.

Ludwig, whose description of the clinical course, has remained the standard up to the present, said that in the first four or five days, the constitutional symptoms were not severe, but became so later. From a study of 104 cases collected from the literature and his own two, the writer believes that this change in gravity of the constitutional symptoms, has a definite relation to the invasion of the mouth and pharynx by the phlegmonous process: and that the increase in severity is out of all proportion to the increased area infected, and the corresponding amount of toxins absorbed. This raises the question as to whether the constitutional symptoms are due entirely to septic intoxication, or whether

they may not be due in part to interference with respiration. Davis says "whether these deaths are due to suffocation or heart failure caused partly by sepsis and partly by the impeded respiration is sometimes difficult to say." He also adds that "these sudden deaths occur usually in patients in which the epiglottis and larynx are affected and the dyspnoea marked." One would infer from this statement that Davis believes that these sudden deaths are the result of the affection of the epiglottis and larynx. The writer believes that practically all deaths in Ludwig's Angina are to be accounted for in the same way. Some develop pneumonia and pleurisy, while a few may die of septic intoxication. Engelman says that seventy-five per cent. of children dying of diphtheria have broncho pneumonia. Diphtheria is a severe infection of essentially the same parts of the throat as are involved ultimately in these cases of Ludwig's Angina, and broncho pneumonia should be as likely to result in one as in the other. Septic intoxication, itself, probably, kills no more patients suffering from Ludwig's Angina, than do these same types of infection occurring in other parts of the body, as in the palm of the hand, the forearm or leg, or in other parts of the neck. "In Robertson and Biedert's case," Davis says "sudden death occurred after a tracheotomy had been performed, so that suffocation could not have been the cause." While it would be difficult to show that suffocation, actually, occurred in this case, the fact that the first symptom complained of was dyspnoea, and that six hours after the onset it was so severe that tracheotomy became imperative, points to the fact that disturbance of the respiratory tract probably killed the patient. In this case as in most of the 14 which Semon reported, the phlegmonous process, evidently, began close to the larynx. In Semon's cases extension to the lungs or pleurae occurred in 5 out of the 6 fatal cases. Pneumonia developed in 3, in one on both sides, and in two double pleurisy was present. In two of the eight cases, which recovered, a double patchy pneumonia was noted. On the same point Davis says further: "In one of Ross' cases, like-

wise, sudden death resulted while the opening existing through the larynx was sufficient to preclude respiratory obstruction." In this case the focus from which the phlegmonous inflammation extended was, evidently, the necrotic wisdom tooth, and from this focus pus and gas escaped on prying away the tooth. With the beginning of the process only about two inches away from the larynx and within the mouth close to the pharynx, it is more than likely that oedema of the larynx developed early. On the fourth day after operation, two patches of impaired resonance were made out, one in each lung. It would seem to be evident, therefore, that in both these cases, the invasion of the respiratory tract and not septic intoxication, caused the death of the patients. Why these cases in which the clinical evidence of oedema of the glottis, *i.e.*, the intense dyspnoea, is so pronounced as to demand immediate tracheotomy do not recover when this operation permits an apparently free passage of air to and from the lungs the writer is not prepared to explain. That the deaths in these are, indirectly or directly, the result of the invasion of the respiratory tract, larynx alone or larynx and lungs, the writer believes. One of his own cases breathed through the tube after the tracheotomy had been performed, but could not be kept alive by artificial respiration. In one of Baker's cases, tracheotomy was done soon after his admission to the hospital, but the pulse stopped during the operation and the patient died. The autopsy showed oedema of the glottis (see autopsy cases). In one of Tissier's cases, tracheotomy was performed for intense dyspnoea on the day of his admission to the hospital, the third day of the disease. Notable relief followed the operation, but the patient died the same night. In Weiss' case, a tracheotomy was done on the first day of the disease. It was necessary to continue artificial respiration for a half hour to revive him. He recovered. Fenwick's case required a tracheotomy, 4 hours after the beginning of the disease. Great relief followed the operation, but the patient died three hours later. In Gibson's case, swelling began in the neck

below the lower jaw, at noon of one day. On the following day the swelling was enormous, extending to the chest and zygoma. The floor of the mouth was considerably thickened, and there was slight dyspnoea. He was admitted to the hospital about 1 P.M. At 3 P.M. of the same day, he became intensely dyspnoeic and tracheotomy was performed immediately, followed by artificial respiration. He recovered and the respiration became normal. On the next day at 11 P.M. there was dyspnoea and considerable cyanosis of the face and lips. He gradually became comatose and died at 3.15 P.M. The autopsy showed oedema glottidis (see autopsy cases). There can be little room for doubt that in all these cases the essential cause of death was the invasion of the respiratory tract, larynx alone or larynx and lungs. Septic intoxication, probably, played only a secondary part in bringing about the fatal result.

It is well known that the partial obstruction of the pharynx from faucial and pharyngeal adenoid growths, will impair the general health of a child by interfering with the normal respiration. Much greater interference coming on suddenly in Ludwig's Angina, from pushing the tongue upwards and backwards and crowding the mouth and pharynx should produce a more serious deleterious effect upon the general condition, the signs of which will be added to and confused with those of the septic intoxication which is already present. When we take into consideration the fact that there was oedema of the glottis in, practically, every fatal case in the writer's group of cases, in which the larynx was afterwards exposed at autopsy, it becomes evident that the interference with respiration is greater than is generally supposed. Dyspnoea was noted in nearly all the fatal cases, and in the opinion of the writer it is the invasion of the larynx and lungs, not the septic intoxication, which is the peculiarly dangerous feature of Ludwig's Angina. It is sufficient to explain the high mortality, septic intoxication is not.

While in most of the cases it is difficult or impossible to differentiate between the parts played by these two factors,

in a few it is shown clearly that all the alarming symptoms characteristic of a Ludwig's Angina may develop in the absence of severe constitutional symptoms, as in the following. Where temperature alone is given it should be borne in mind that this was the only symptom mentioned in the report of the case, from which one could infer the degree of the constitutional disturbance; and where it is not given here it was not mentioned in the report, and any statement implying the degree of constitutional disturbance or absence of it was extracted and employed in these brief summaries. In one case reported by Huguet and DeBovis, there was an extensive submaxillary swelling, "enormous" sublingual swelling, dysphagia, dyspnoea and a considerable quantity of fetid pus; yet the temperature never went above 39°C (102°F). In one of Parker's cases, the usual severe symptoms were present except dyspnoea, which may have borne some relation to the presence of a discharging sinus in the floor of the mouth. This may have checked the progress of the inflammation towards the larynx. The general health was not impaired. In another of Parker's cases, the general health was reported to be good. Leube's case, which underwent resolution, had a normal temperature. In Trump's case and in three of Davis' cases, the temperature was only 101°F. In Margerison's, the temperature was 100.8°F, pulse 104, and in Humphrey's it was never above 100°F. Leterrier reported that in his case the general condition was good, the temperature 37.4°C (99.3°F) and that the patient would have taken food if he could swallow. All these cases recovered. Michel's patient was admitted to the hospital on the 5th day of the disease, when he had an enormous submaxillary swelling. On the day preceding admission asphyxia was threatened. He died 4 hours after admission. The temperature was given at 39°C (102°F). One of Schwartz's cases, on the day of admission to the hospital, insisted on going out again to attend to some business, which he was permitted to do. He returned later in the day and died of syncope that night. In Gibson's case, the submaxillary swelling began at noon of one day. On the following day at 1 P.M., when he was admitted to the hospital, the swelling was enormous. A little later the dyspnoea became intense. Tracheotomy was performed and artificial respiration carried out with relief to the patient. At 3 P.M. of the same day he died in coma and dyspnoea. Yet the temperature on admission, 2 hours before death, was only 97.8°. In Fenwick's case, the swelling began in the morning. Two hours later the face was almost unrecognizable. In 4 hours he was cyanosed and could hardly breathe, and in 7 hours he was dead. Yet the temperature was normal, the pulse 140. It would seem, therefore, that in some cases essentially all the symptoms of a Ludwig's Angina may be present, and those of septic intoxication be very moderate or practically absent. Indeed, in only a comparatively small number of the cases collected by the writer, was high temperature referred to, and in the great majority the presence of severe constitutional disturbance could only be inferred from the general gravity of the case. Inspection of the atopsy cases, given later, will confirm this statement.

Influence of the local condition.—While definite results have never been obtained from bacteriological investigations, beyond the fact that the streptococcus is present in nearly all the cases, pure or mixed with other organisms; the study of the local inflammatory conditions have yielded more satisfactory results: The observations of Poulsen, Delorme, Semon and more recently Davis, in the writer's opinion, have been the most valuable of recent years. These writers seemed to consider the infection from a distinctly local standpoint, and to regard the larynx as the essentially vulnerable point of attack.

Poulsen says that the deep cervical fascia in the submaxillary region is dense and resistant, and that the submaxillary salivary and lymphatic glands are enclosed in a fascial space. This submaxillary fossa communicates by means of loose cellular tissue and blood vessels with the deep retromaxillary fossa, so that a cellulitis beginning in one of these spaces readily extended to the

other through this communicating passage. He explains the dangerous symptoms of dyspnœa and dysphagia in Ludwig's Angina, by an extension of the inflammation through the wall of the pharynx to the pharynx and larynx from the retromaxillary fossa. He contended that those cases beginning with a preliminary angina gave secondary involvement of the lymphatic glands in the retromaxillary fossa about the bifurcation of the carotid artery, and that the resulting periglandular cellulitis then passed through the wall of the pharynx. When the phlegmonous process began in the submaxillary lymphatic glands, as from a carious tooth or ulcer in the tongue or floor of the mouth, the overlying strong fascia gave rise to great tension so that the inflammation, seeking the direction of least resistance, passed along the communication to the retromaxillary fossa, and thence through the wall of the pharynx to the pharynx and larynx. Poulsen's conclusions are not based upon strictly anatomical studies, but upon the results of his lime injections. When the lime was injected under the deep fascia in the submaxillary region, it first produced a swelling in this region which was soon followed by extension to the region of the large vessels of the neck, and almost simultaneously to the alveolo-lingual sulcus in the floor of the mouth. In no case did it work its way through the wall of the pharynx, the path by which Poulsen claimed that the inflammation reached the larynx. He obtained hospital statistics of 530 abscesses of the neck, of which 251 occurred in the submaxillary region. Of the 251, there was a swelling in the floor of the mouth or alveolo-lingual sulcus in 22. In 2 of the 22 there was a spontaneous opening in the floor of the mouth, in one at the orifice of Wharton's duct. As a rule the inflammation subsided after incision in the submaxillary region, and only twice was the œdema so abundant that an incision in the mouth was necessary. Of the 251, 11, or 4 per cent., died. Poulsen considered that only three corresponded to the clinical picture of Ludwig's Angina, in which he attached especial significance to the non-fluctuating swelling in the submaxillary region, the lack of large pus foci, the intact skin, and the extension of the swelling to the floor of the mouth. He eliminated one of these because of the absence of an autopsy. The writer has included the other two in his collection of cases, and they will be found among the autopsy cases.

The two points in Poulsen's paper, to which the writer attaches greatest importance are: first, that Ludwig's Angina results from the extension of an infection of the neck to the larynx and pharynx; and secondly, that the cellulitis had its origin in extension from the lymphatic glands. He was far, however, from proving the path of extension. His most important evidence lay in the fact that in several cases, when the abscess was opened the finger of the surgeon could be passed down to the pharyngeal wall, the infection being traced in this way nearer to the pharynx and larynx than in any other direction. He attached considerable significance to the fact that in one case, not regarded by him as a Ludwig's Angina, during the making of an external incision into the abscess, there occurred a spontaneous opening into the throat. In no cases did he demonstrate an opening in the pharyngeal wall. Spontaneous openings have been reported rather frequently, generally in the mouth, some of them occurring near the base of the tongue or in the throat, and are readily explained in another way.

As the result of his clinical observations and experience Delorme concluded that Ludwig's Angina was nothing more than a sublingual phlegmon; although on account of its exact anatomical seat and constant symptoms, he was inclined to view it as a morbid entity and to retain the name of Ludwig's Angina. Leterrier in his thesis, already referred to, offered two arguments to support Delorme's theory. In the first place it was found necessary in all their cases to cut through the mylo-hyoid muscle from the neck, and, therefore, into the sublingual tissues, before pus was reached. In the second place, according to Leterrier, the almost constant swelling in the floor of the mouth and the elevation of the tongue, could be due only to a sublingual phlegmon. He also added that when there was a spontaneous opening made by the pus, it was usually internal. He believed that if this theory was generally accepted and the external incision extended deeply enough, the mortality would be much diminished. All of their cases recovered. A number of writers,

particularly in France, accepted Delorme's view and reported Ludwig's Angina as synonymous with sublingual phlegmon. Huguet and DeBovis, who collected and studied 49 cases, regarded them as sublingual phlegmons, but held that "these sublingual phlegmons can only be the result of diffusion of an inflammation developed more posteriorly in the region of the parotid or angle of the jaw." They believed that its anatomical seat was intramuscular, *i. e.,* that it was a basic glossitis. They could not admit that a purulent collection under the mucous membrane in the floor of the mouth would produce a hard, non-fluctuating swelling; and they added that some surgeons who have intervened by the mouth have not met with success or have had to plunge the bistoury to a considerable depth.

With reference to this point the writer has investigated his 104 collected cases with the following rather indefinite results. Nelaton made a sublingual puncture, only blood escaping. Later he made two external incisions, one a suprahyoid incision exposing a putrid focus, the other a submaxillary incision, only infected serum escaping. Death resulted from syncope. No autopsy. Chauvel made a double sublingual incision and exposed a gangrenous focus above the mylohyoid muscle, extending to the upper border of the thyroid cartilage. (Extension to the thyroid cartilage implies that the focus was below the mylo-hyoid muscle also, and therefore in the neck.) Dubois found phlegmonous pus by a sublingual incision. Haering made buccal scarifications but found no pus. Cuffe made a buccal incision toward the posterior part of the tongue but found only blood. Later the incision was repeated and pus was found. Holthouse made buccal scarifications but found no pus. Ross found no pus by a sublingual incision, but with an external incision located a large abscess. Ripault evacuated 2 or 3 cupfuls of pus by a buccal incision, and by a median external incision also found pus. There were sublingual and retromaxillary fluctuation in this case.

In most of the cases, however, it was the external incision which located the pus, and in only a few was the mylohyoid muscle said to be divided. The writer will show later that the sublingual phlegmon is the result of extension in the great majority of the cases, and that it is not the primary phlegmon as Delorme maintained. Leterrier explains the origin in the sublingual tissues by assuming that the infectious germs gaining entrance by a focus in the mouth as a carious tooth or an ulcer, are carried by the lymphatics to the cellular tissue about the sublingual gland. He says also that Richet has described a chain of lymphatic glands arranged in a horse shoe manner along the internal surface of the inferior maxillary bone, thus implying that if these glands existed, they would explain the frequency of cellulitis by periglandular extension.

Semon's paper, which appeared in 1895, is the most recent to attract wide attention. His conclusions are based upon clinical observations on 14 cases, which he saw in 20 years of special practice as a laryngologist. The main conclusion he reached was that "these acute septic inflammations of the throat and neck, described by a large variety of terms, such as acute oedema of the larynx, oedematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx, and Angina Ludovici, are pathologically identical. They merely represent different degrees of severity of one and the same septic process due to invasion of the throat and neck by various micro-organisms." He adds that this can be finally proved only by a harmonious combination of clinical, pathological and bacteriological evidence. In every one of his cases, except the first he had tried to obtain a bacteriological investigation, but only in the last was this opportunity afforded, and then the evidence was purely negative. He called attention to the fact that Virchow could not exactly define the mutual relationship between erysipelatous and phlegmonous affections. Semon believes that the question of the primary localization and subsequent development depends, in all probability, upon accidental

breaches of the protecting surface, through which the pathogenic microörganisms gain entrance to the tissues.

According to Semon, therefore, we are not concerned with any particular infection, so much as with a special type of inflammation, an acute septic phlegmonous process, which may be due to various microörganisms. Lockwood, who studied this condition from the bacteriological side, reached the conclusion that Ludwig's Angina is a mixed infection of the most complicated kind, and that several pathological conditions are included in this affection. He found that usually the streptococcus was present, though not always; and that this microörganism may be present alone or associated with other organisms, as the staphylococcus. From his study of the subject the writer prefers to accept Semon's view on this point. The complicated nature which Lockwood assigned to this infection, becomes simplified by the fact that whatever microörganism is found, the process is always the same, a rapidly spreading phlegmonous infiltration of the cellular tissues. This is the result usually produced by streptococcus infection, and it may be due to staphylococcus infection. Gasser quotes Queno as saying that any of the pyogenic organisms may be found in these cases. Other organisms may also produce it, as the bacillus of malignant oedema, which Lockwood found in one of his cases. We are not yet familiar with the exact results produced by the various bacteria, and Semon's statement seems sound that "it is absolutely impossible to draw at any point a definite line of demarcation between the purely local and the complicated, or between the oedematous and suppurative forms."

Semon maintains, however, that all acute septic phlegmonous inflammations of the throat and neck should be classified together, and that Ludwig's Angina as a separate disease should be eliminated. That they are all pathologically identical and that the throat, *i.e.,* the larynx, is the most vulnerable point in all, the writer believes. From the standpoint of prognosis and treatment, however, there is a very practical difference between those in which the

phlegmonous process begins in the throat and those in which it begins in the neck, where the condition described by Ludwig had its origin. Many of the latter have shown a preliminary angina, it is true, but this usually disappeared later and did not form a part of the phlegmonous process beyond serving as the portal of entrance for the microörganisms. In most of Semon's cases and in one of the writer's collected cases, the acute septic process began in the throat and spread out from there. These in the writer's opinion, form a distinct group, and are laryngological; those which Ludwig described are distinctly surgical and in the majority of cases respond to surgical treatment. The following advice given by Semon, may be proper for the former but not for the latter. "Should there be anywhere distinct fluctuation or merely justifiable suspicion of such, of course you will incise upon such foci. Our promise for the future must depend on the fact that we have a bacterial infection, and that by the injection of an appropriate antitoxine we may be able to save the patient." Fluctuation or even a suspicion of it is practically never present. Prompt and suitable incision in the absence of any sign of fluctuation, has arrested the progress of many cases, probably, after œdema of the larynx had already set in. Antitoxines may be employed with advantage after incision and drainage have been provided, but not before.

No fact is more evident from a study of the literature, or is so generally conceded, than that the cellular tissue is the essential seat of the inflammatory process, and that the surrounding structures become involved by contiguity. The literature also shows clearly, notwithstanding the claims of Delorme and his followers, that in the great majority of cases the cellulitis originates externally in the submaxillary region and not in the sublingual region, *i.e.,* in the mouth. Of the writer's 106 cases, in 61 the swelling was first noted in the submaxillary region of one side. In 16 it was bilateral and under the jaw when first seen by the physicians reporting them. In 2 there was a submental swelling which may have been a bilateral submaxillary involvement. In 13 others the

swelling was described as involving the cheek and neck, face and neck, parotid region, etc., *i.e.,* it was in the beginning an external swelling. Of the 106, therefore, 92 began in the tissues of the neck external to the mouth and throat. In 8 cases the first swelling was sublingual, and from the description in 3 (Huguet and DeBovis 2, Holthouse 1) the writer considers it doubtful whether a sublingual or a submaxillary swelling first appeared. In two cases (Tordeus and Aldrich) it was described as a submaxillary and sublingual swelling. One case began in the throat as in Semon's cases, and is included here because it was considered by the writers reporting it as a Ludwig's Angina. The writer regards these facts as of much value in establishing the nature of the disease, and considers that they support what Ludwig claimed, that the cellulitis begins in the submaxillary region, at least, in the great majority of cases. Those which begin in the mouth can be easily accounted for, but there has been much dispute concerning the submaxillary origin and the term, idiopathic, has been employed in connection with them. Semon says "A little abrasion on the side of the neck exposed to the action of those pathogenic organisms may, of course, invade the body from the outside and may cause what has hitherto been called an Angina Ludovici. The original focus is purely accidental." One would infer from this that Semon considers that from such an abrasion, invasion occurs by direct continuity of tissue until the throat is involved. Davis says: "When the teeth are the starting point the inflammation involves the periosteum of the lower jaw and thence invades all the surrounding tissues. While the point at which the infection starts localizes the disease at its commencement, it progressively spreads and invades all the tissues within its scope. No matter how it commences it spreads along the connective tissues by direct continuity. It is not transmitted by the lymphatics. The lymphatic glands do not become enlarged by infection carried to them by the lymph stream from the infectious focus, but they are involved

in the connective tissue surrounding them." As already stated Leterrier considered that the infection was transmitted from some focus in the mouth to the cellular tissue about the sublingual gland setting up a cellulitis there. Roser believed that the infection was transmitted to the submaxillary salivary gland, and that the extension occurred to the surrounding cellular tissue.

That the primary focus in the great majority of cases is some insignificant lesion in the mouth, as a carious tooth, an herpetic or other ulcer, a tonsillitis, etc., has been generally admitted and so far as the writer can learn has never been denied. In many, however, no such focus was discovered. If the infection gains entrance to the tissues by such a focus in the mouth and the signs of inflammation first appear in the submaxillary region, external to the mouth and some distance from the original focus, the process can not be said to have extended by direct continuity of tissue. This applies with greater force to those in which no preliminary focus could be found, the typical, so-called, idiopathic cases. The writer's statistics on this point will be found later in connection with the clinical course of this condition.

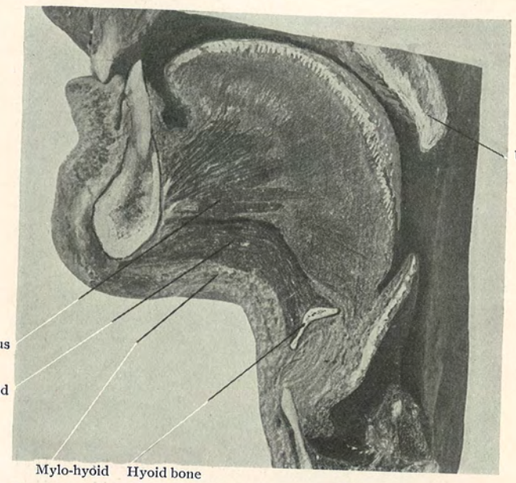
There can be only one explanation for such a transference of infection, and that is by way of the lymphatic vessels to the glands in the submaxillary region. Most infections in this region are of glandular origin. Poulsen said that the great majority of his 251 submaxillary abscesses were cases of simple or localized adenitis, and he takes it for granted that his cases of Ludwig's Angina began also in the lymphatic glands. v. Thadden considered it a lymphatic disease and gave to it the name "Submaxillary bubo." Localization of infection is the rule in any part of the body, and this is particularly true of those which lodge in lymphatic glands. Fulminating cases are rare. Typical Ludwig's Angina is rare and is also fulminating. It is easily conceivable that such an infection might be transferred from some slight focus in the mouth, where there is no retention, the discharge being free, to a submaxillary lymphatic gland where the

infection is confined, and therefore more active, and from there on account of its increased activity invade the peri-glandular tissue so rapidly that its glandular origin is overlooked. In some cases the glandular origin was indicated by an early localized pain in the submaxillary fossa, which was soon followed by rapid swelling.

While the glandular origin was concealed by the rapid swelling in most of the writer's cases, this was not true of all. In one of Tissier's cases there was pain in the left submaxillary region on the first day. Swelling appeared on the following day. In one of Delorme's cases, the condition was first observed in the submaxillary region as "three glands," rapidly increasing in size. Bauer reported one in which the patient had similar attacks before. Ludwig's case in 4 days, had only reached the size of a hen's egg. One of Haering's when first seen was of the same size, Heyfelder's the size of a goose egg, and Timpe's of a five franc piece. Davis says of two of his cases, that one week before, the neck began to swell and later increased rapidly. In Blasburg's case there was an indolent swelling for 8 days and rapid swelling began on the 11th day. In the writer's case there had been a small lump for about a week before rapid swelling began. There can be little doubt of a glandular origin in these cases, and in the writer's opinion, they go far toward proving the glandular origin in the so-called idiopathic cases.

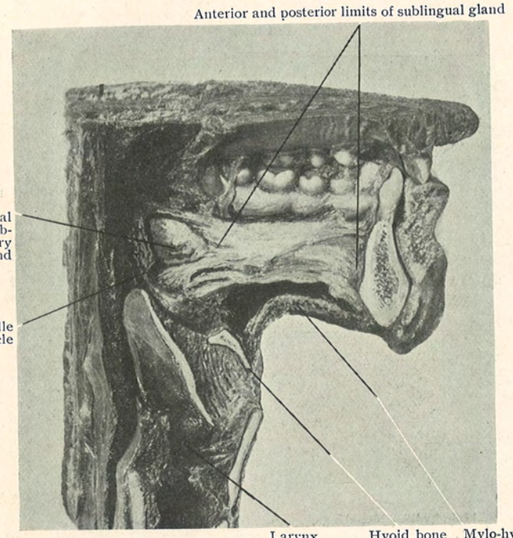
A cellulitis localized to the submaxillary region, regardless of the kind of infection, in the writer's opinion, is not a Ludwig's Angina; but becomes one as soon as the process invades the floor of the mouth and the pharynx. Poulsen as we have seen, assumed that the invasion occurred through the pharyngeal wall. Delorme merely located the phlegmon in the sublingual tissues without attempting to trace its further progress, while Semon simply stated that extension occurred from the throat to the neck, or from the neck to the throat, without reference to the path of progress. Davis seems to agree with Semon, but adds that it spreads along the connective tissue by direct continuity.

FIG. 1.



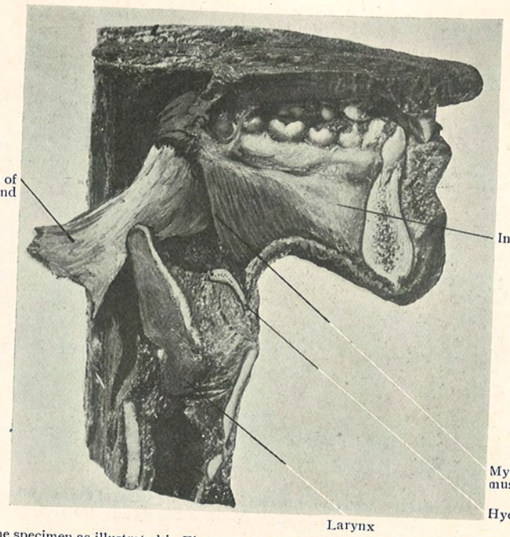
Median, sagittal section of that part of face and neck involved in Ludwig's Angina. Horizontal section about $\frac{1}{16}$ inch below highest part of roof of mouth. Tongue, somewhat shrunken from loss of body fluid, almost fills mouth.

FIG. 2.



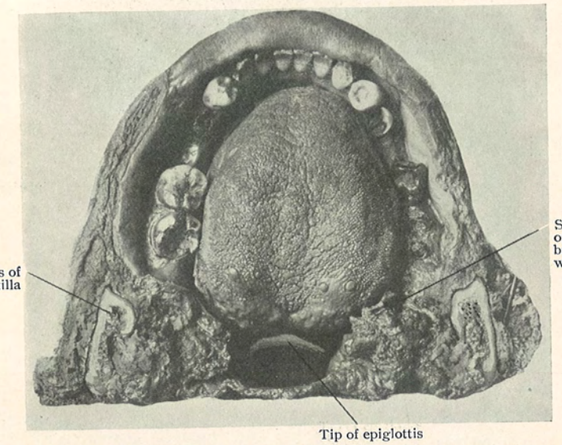
Half of specimen opposite to that illustrated in Fig. 1. Half of tongue removed, to show continuity of cellular tissue about the submaxillary and sublingual salivary glands, and proximity of deep portion of submaxillary gland to larynx.

FIG. 3.



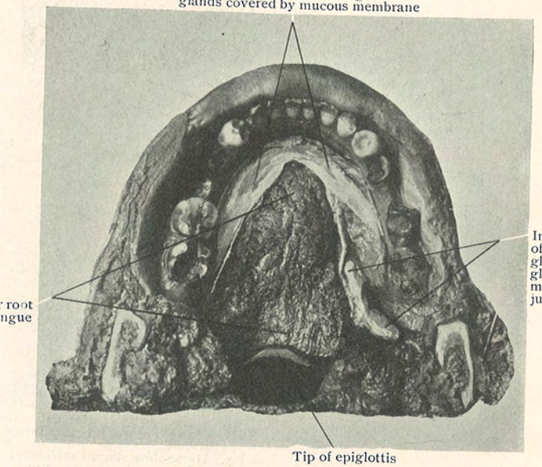
Same specimen as illustrated in Fig. 2. Sublingual gland and deep portion of submaxillary turned backwards, showing from within the mouth the anterior boundary of the opening in the muscular bucco-pharyngeal wall.

FIG. 4



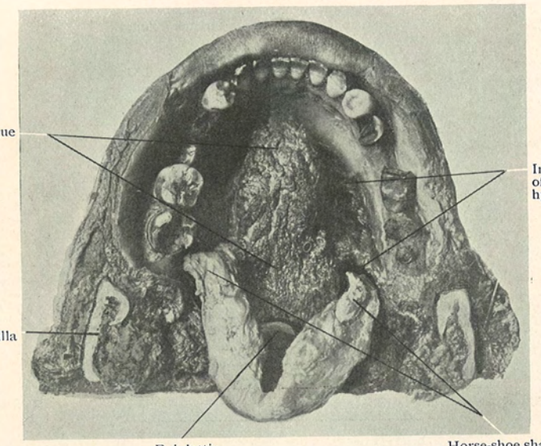
Upper surface of specimen similar to that formed by the union of the two, illustrated in Figs. 1, 2 and 3. Tongue crowds teeth laterally. It is loosely attached and has dropped backwards, slightly.

FIG. 5.



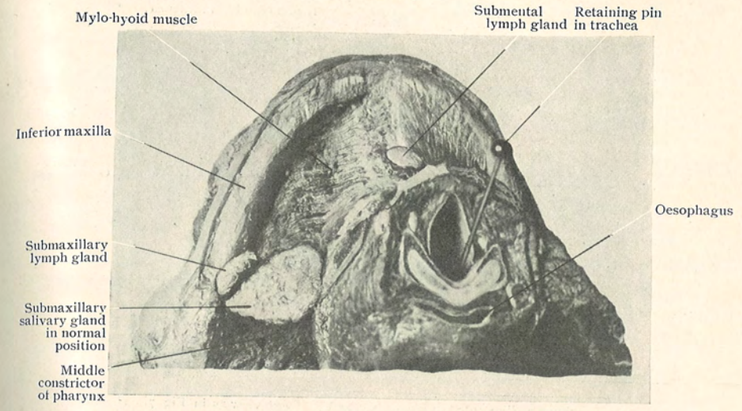
Same specimen as illustrated in Fig. 4. Tongue removed showing horse-shoe arrangement of sublingual cellular tissue and salivary glands.

FIG. 6.



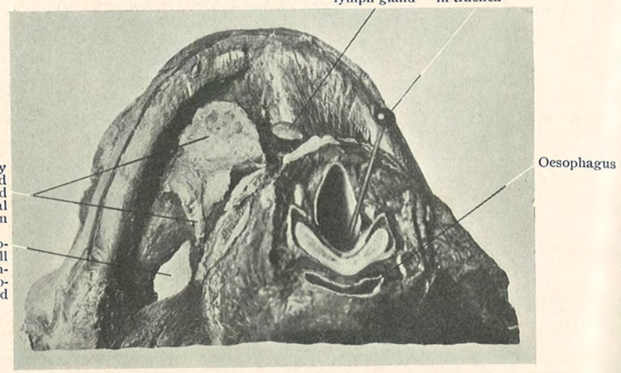
Same specimen as illustrated in Figs. 4 and 5. Horse-shoe shaped sublingual tissues turned backwards showing their continuity with similar tissue in the submaxillary region through the bucco-pharyngeal opening. The alveololingual sulci are also shown.

FIG. 7.



Same specimen as illustrated in Figs. 4, 5 and 6. External view. Submaxillary and submental regions. Bucco-pharyngeal opening plugged by submaxillary salivary gland.

FIG. 8.



Same specimen as illustrated in Figs. 4, 5, 6 and 7. Same view as in Fig. 7. The submaxillary salivary gland being drawn forward the bucco-pharyngeal opening is well shown. The fissures between the mylo-hyoid and middle constrictor muscles, however, extends from the hyoid bone to the angle of the jaw.

The writer hopes to demonstrate how a cellulitis about the submaxillary salivary gland, may progress along planes of connective tissue to the mouth and pharynx, and why such extension so quickly invades the region of the larynx. Leterrier said that not enough attention had been paid to the anatomy of the mouth in connection with Ludwig's Angina, and he is the only author so far as the writer can learn, who has paid any attention to it. He drew his conclusions from a study of the topographical anatomy of Tillaux and the demonstrations of Sebileau. The writer has made a special study of this subject by dissections of this region.

The anatomical work was done in the department of Applied Anatomy of the University of Pennsylvania, and the writer wishes, here, to thank Professor Gwilym G. Davis, the department head, for his kindness in furnishing all the necessary facilities. To Professor Geo. A. Piersol the writer is indebted for the freedom of his anatomical department and his specimens, and to Mr. Erwin F. Faber for valuable assistance in emphasizing in the illustrations those points which are essential to an understanding of the text. This opportunity is taken to acknowledge also the writer's indebtedness to Professor J. William White for kindly criticism and valuable suggestions.

Few portions of the body are so imperfectly dissected by the average student as is this region. As a result few physicians can comprehend with any detail the anatomical relations of the floor of the mouth in its relation to the pharynx and larynx. Special sections were necessary to expose the tongue, the pharynx and the larynx, and the adjacent parts of the neck in the same specimen. By a transverse section of the head above the upper surface of the tongue, and a vertical section through the pharynx, of the lower part removed by the transverse section (see figure I.), a part of the head was obtained which gave a free exposure of the tongue from above and of the posterior part of the tongue, the anterior wall of the pharynx, and the complete larynx. The parts involved in Ludwig's Angina were thus

preserved in this portion of the head and could be dissected from above and below. The facts brought out by these dissections taken in conjunction with the clinical facts repeatedly demonstrated by the recorded cases and with the autopsy reports, seem to clear up many of the obscure points associated with this condition. Only those autopsies which have shown the condition of the larynx have been considered.

Anatomy.—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 separating the mouth from the neck is a complete one from the posterior edge of one mylohyoid muscle to that of the other and is a comparatively strong one. 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, extend backward the constrictor muscles of the pharynx, separating the pharynx from the neck, the muscles of the two sides fusing together at the posterior median raphe. The three constrictors, 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, is a considerable deficiency in the bucco-pharyngeal muscular wall (see figure 8). 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 floor 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 into the mouth or in the opposite direction, do so through this opening. These are the glossopharyngeal and hypoglossal nerves, the lingual artery and vein and the styloglossus muscle. The greater part of the opening, however, is occupied

by the deeper portion of the submaxillary salivary gland which here projects into the floor of the mouth, near the root of the tongue, where it lies just under the mucous membrane. 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 loose connective tissue (see figure 2). We thus see that the connective tissue in the submaxillary fossa is directly continuous with that in the floor of the mouth, so that the extension of a submaxillary cellulitis to the sublingual region, which occurs so early and so constantly in Ludwig's Angina, is readily understood. The observations of Huguet and DeBovis, who, while regarding Ludwig's Angina as a sublingual phlegmon, said that this "can only be the result of diffusion of an inflammation developed more posteriorly in the region of the parotid or angle of the jaw," is seen to have a sound anatomical basis. What is more important, it supports the statements of Ludwig and the great majority of writers reporting these cases, who said that it began in the region of the submaxillary gland. The difficulty in explaining why this extension occurs so rapidly in some cases is not so great as in explaining why such extension does not occur in more cases. Probably it does occur much more frequently than we have suspected and is overlooked because its nature has not been understood. It has probably been arrested many times by prompt incision before alarming symptoms have had time to develop. While walking through one of the wards of a hospital recently, the writer's attention was arrested by a case of extensive submaxillary cellulitis. The mouth could not be opened and when the patient was asked if he experienced any trouble inside the mouth he said that beginning with the day before he had considerable difficulty and pain in swallowing. An incision had been made that day. On the following day he reported that he felt much better and that the dysphagia had disappeared. The inflammation had probably begun to extend

into the mouth in this case, and had been arrested by the incision. Of Poulsen's 251 submaxillary abscesses, as already stated, in 22 the swelling involved the floor of the mouth, and in 2 (not the 2 reported by Poulsen as examples of Ludwig's Angina) this swelling was so abundant that an incision in the mouth was necessary. As a rule the inflammation subsided after incision in the submaxillary region. Poulsen regarded only 3 of the 22 as examples of Ludwig's Angina, and paid little or no attention to the rest; so that we can obtain light on the progress of the other cases, only by inference from the associated facts. Of the 251, 11 or 4 per cent., died. Poulsen says that the great majority were cases of simple or localized adenitis. A death from simple or localized adenitis must be exceedingly rare, so that almost all of the 11 deaths, in all probability, occurred among the 22 in which the floor of the mouth was invaded, since as the writer will show, this must always be a very dangerous condition. If this were true of all the 11 deaths the mortality among the 22 cases would then be 50 per cent., which is approximately that of Ludwig's Angina, as determined by other writers. In the writer's collected cases, the mortality was 40 per cent. In the anatomical specimens it was observed that the connective tissue about the gland in the opening in the muscular floor of the mouth, was small in quantity. The gland being somewhat wedge shaped, with its base external and its apex internal it is possible that a massive exudate external to the gland might force it more snugly into the opening as a plug, thus aiding in localizing the inflammatory process to the external tissues, more effectively in some cases than in others. It was generally the fulminating infections which were present in Ludwig's Angina, in all probability because of the great facility with which they extend along planes of connective tissue.

A phlegmonous cellulitis in the floor of the mouth as from an infected wound, is a menace to the life of the patient, regardless of the kind of microörganism producing it. Relief must be afforded promptly or, the process extending, the larynx will soon be invaded and the patient suffocated. To

appreciate the reason for this a further study of the anatomy of the floor of the mouth is necessary.

The mouth with the jaws closed may be roughly compared to a small box of which one side has been removed. The upper side or roof is represented by the roof of the mouth, the lower side or floor by the two mylohyoid muscles, the front and lateral sides by the teeth and jaws. The posterior side is absent. With the jaws closed the mouth is practically filled by the tongue and the normal sublingual tissue. Therefore, when the cellular tissue under the tongue is invaded by inflammation, as in Ludwig's Angina, the tongue is pushed upward and the mouth must open to make room for the new inflammatory material. Speech and deglutition are necessarily interfered with and the saliva now increased by the inflammation can not be properly swallowed and frequently escapes from the mouth. The tongue crowded for room may show between the teeth and appear to be swollen when it is not. It was actually swollen, in one of Parker's cases, from invasion by the inflammatory process (see page 226). The tip, at least, is probably rarely involved. Posteriorly the tongue becomes wider and dips downward and backward toward the larynx, where the base of the epiglottis is attached to its posterior surface. Laterally the base of the tongue reaches the side of the pharynx, where it receives the attachments of the styloglossus and palatoglossus muscles. These attachments of the sides of the tongue to the walls of the pharynx, make on each side a strong muscular ridge covered by mucous membrane and submucous tissue, the latter being scanty here. This prominent ridge separates the floor of the mouth from the pharynx, so that a submaxillary infection extending through the opening already described, and finding itself in the floor of the mouth in front of this ridge, must extend through it along the intermuscular fascia or over it along the scanty submucous tissue. This explains why the swelling in the floor of the mouth is so well developed before the œdema has produced alarming symptoms in the pharynx and larynx.

The finger placed in the mouth will easily find this ridge. Since the tongue turns downward and backward, the sublingual swelling lies in front of this posterior portion, so that the tongue with the epiglottis attached to its dorsum is pushed backward toward or against the posterior wall of the pharynx, tending to obstruct the air which is passing from the nose and mouth to the lungs. By the same mechanism in anæsthesia, the dropping backward of the tongue and epiglottis may interfere with respiration.

It is probably little appreciated how limited is the space confined within the arch of the lower jaw. It will suffice here to point out that the distance in a straight line from the symphysis along the floor of the mouth to the base of the epiglottis and, therefore, to the upper orifice of the pharynx is, approximately, only 2½ inches (see fig. 1). The submaxillary salivary gland lying in the opening in the floor of the mouth is about on a transverse plane with the base of the tongue, *i.e.*, just anterior to the larynx; so that the portion of the gland projecting into the mouth is only about 2 inches external and anterior to the larynx. The chief protection of the larynx, at first, is the muscular ridge already described.

A further brief description of the floor of the mouth will be of value in explaining some of the points which have attracted the attention of various writers. For instance, it has been frequently reported that the hard sublingual swelling was of a horse shoe shape. The floor is divided into two lateral portions (see figure 6) by the muscles which pass upward from the symphysis of the lower jaw and the hyoid bone to the tongue, the hyoglossus and geniohyoglossus. The geniohyoid aids in forming the lower portion of this median septum (see fig. 1). The two lateral alveololingual sulci thus formed are freely continuous with each other anteriorly under the frænum of the tongue, but are terminated abruptly posteriorly by the lateral attachments of the base of the tongue. They are thus seen to have a horse shoe shape, and as they are filled by the sublingual and portions of the submaxillary glands surrounded by loose cellular tissue, the

swelling due to cellulitis in them will assume a horse shoe shape also. It has been observed in a few cases that a submaxillary cellulitis of one side with extension to the floor of the mouth, has been followed in a short time by a corresponding but smaller swelling on the opposite side, the two not being continuous under the chin. Some of these cases are probably to be explained by extension of a submaxillary cellulitis of one side to the floor of the mouth, along the sublingual sulci and out through the opening in the floor of the mouth on the opposite side. More frequently bilateral swelling results from extension along the external connective tissue under the symphysis.

What has made Ludwig's Angina so important is the frightful rapidity and certainty with which an unchecked case proceeds to a fatal termination. The submaxillary region is already intensely swollen, so that the jaw can not move downward to relieve the crowding of the mouth and pharynx. The sublingual pressure can not find relief in that direction, even if the mylohyoid muscle did not resist its downward progress. Above it meets the resistance of an overhanging probably rigid tongue, which is already pressed against the roof of the mouth. Extension anteriorly or laterally is resisted by bone and teeth. The direction of least resistance is backward, in fact it is the only direction in which the rapidly accumulating new material can force its way. When we take into consideration the fact that it enters the mouth not far from the larynx and that while the inflammation is invading the floor of the mouth it is also more slowly passing backward toward the larynx, we can better appreciate why the dyspnoea follows so soon after the swelling of the floor of the mouth.

Only prompt relief of a pus collection by incision or a spontaneous opening can be expected to give relief, and these have failed in some cases. It is true that spontaneous resolution has occurred in some cases, but this can not be depended on. Leterrier doubted if it ever occurred and believed that in those cases in which it was reported to have taken place,

an unobserved spontaneous opening had developed. In the writer's 106 cases there were 17 in which a spontaneous opening was reported, and in every one there was an internal opening. In only one was an external opening associated. While he would not deny the possibility of spontaneous resolution, the writer would consider it very likely that in some cases in which it was reported to have occurred, a spontaneous opening had been overlooked. It could be so situated under the tongue that it could not be exposed on account of the difficulty in opening the mouth. Only the escape of pus would announce its presence, and this is frequently so small in quantity or so gradual that it could easily escape unrecognized in the abundant and often turbid saliva.

The spontaneous opening is usually internal, probably because pus developed about the submaxillary gland finds itself nearer the mucous surface than the skin of the neck. The inflammatory material inside the jaw and under the tongue is probably under greater pressure than that external to the gland in the neck, which is always abundant so that the inflammatory area should break down more quickly where the pressure is greatest and, therefore, the blood supply most compromised.

Of the writer's cases the following, reported by Parker, illustrates clinically better than any of the others, the mode of origin and path of progress of the cellulitis in a Ludwig's Angina.

JOHN K., aged 12½ years, was admitted to the hospital Sunday, September 8, 1878, at about 10 P.M. On the preceding Thursday (before which time he had been quite well) a "small lump" appeared below the jaw on the left side. It increased in size until Saturday, and then appeared to be an ordinary abscess of the neck. On Sunday it remained much the same until 4 P.M. Then the patient began to complain of his tongue which was swelling. By 6 P.M. his tongue had reached about quadruple its normal size and it protruded from his mouth. On admission to the hospital at about 10 P.M. there was considerable swelling below the jaw on the left side and to a less extent on the right side. No fluctuation, but great œdema. Tongue much swollen, red and tense, and protruding between the teeth, preventing closure of the mouth, which is open to its full extent. Escape of much saliva. On the following day breathing more uneasy. Incision in neck at most likely place, but no pus reached. Toward evening, on account of great distress, the tongue was freely incised on each side of the median line with considerable relief in a short time. September 11th the condition had somewhat subsided. On raising the tip of the tongue pus can be seen issuing at a point where the mucous membrane is reflected from the tongue to the floor of the mouth, and a probe can be passed downward and backward for three or four inches. September 14th neck again incised and pus found. Convalescence soon followed.

This is probably as clear a clinical demonstration as one could find of the origin in a lymph gland, with periglandular extension to the cellular tissue, first in the submaxillary region, then to the floor of the mouth and tongue, and finally to the region of the larynx as shown by the increasing difficulty in breathing. The occurrence of the spontaneous opening under the tongue with a subsidence of symptoms, probably, had much to do with the recovery of the patient. It was the only one to be found among the writer's cases, in which the mouth was reported to be opened widely and the only one in which the tongue was markedly invaded. The opened mouth is probably to be explained by the fact that the floor of the mouth and the tongue were invaded before the submaxillary swelling had become too massive to prevent depression of the lower jaw, which in this case was demanded early for the accommodation of the early swelling in the mouth.

The writer's study of his cases does not show that the pathological changes occurring in the infected area differ materially from those which may be expected from any severe pyogenic infection occurring under similar anatomical conditions. Of the 106, spontaneous resolution was reported in 8. In 26 no pus was found. Two of these showed putrid foci. In 66 pus was found. In 12 of these the pus was

described as putrid, and in 5 gas was associated. In three gas without pus was reported, and in 3 more the process was spoken of as gangrenous. While this classification is, probably, more or less inaccurate—the pus might have been putrid and the fact not have been mentioned, and gangrene might have been present and the fact have been overlooked—it will demonstrate that, in all probability, the pathological changes present were the result of ordinary severe infections, as the streptococcic or staphylococcic. The bacteriological examinations which have been made in these cases, would then be seen to have agreed with the other pathological findings. The proximity to the alimentary tract will account for the frequency of gas and putrescence, while the intensity of the inflammatory process and the compression of the inflammatory swelling inside the jaw and under the tongue with the massive, hard, tense swelling externally, will explain the tendency to gangrene. It is probably no more frequent here than when such an infection occurs under the dense palmar fascia.

The question as to the advisability of retaining or rejecting the name, Ludwig's Angina, is one that probably will not be easily decided. While the process is pathologically identical whether it begins in the throat, in the mouth or in the neck, from the standpoint of prognosis and treatment, as already stated, a sharp distinction should be made between those beginning in the throat and those beginning in the neck. From the same standpoint those beginning with a cellulitis in the mouth by direct extension from the primary focus in the mouth (there were 8 of this type among the writer's cases) might be included with those originating in the submaxillary region. If the primary focus in these cases is exposed before the development of the submaxillary swelling prevents opening of the mouth, it can be thoroughly disinfected and the process, probably, arrested early. We might speak of these two varieties as sublingual phlegmons, one being primary the other secondary. But this would disregard the submaxillary cellulitis, which in the great majority

of cases would be the primary condition and then the most important because it is the one to be attacked surgically. We might speak of this class as cases of submaxillary cellulitis with extension to the mouth and throat. Ludwig's Angina would be more convenient and would be sufficient, since this is exactly the condition which Ludwig described. Delorme, who regarded it as essentially a primary sublingual phlegmon, argued for the retention of the name, Ludwig's Angina. The writer believes that the time has not yet arrived when we can conveniently discard it.

Clinical Course.—While the etiology and pathology of this condition has not been established, the clinical picture as given by Ludwig has, probably, never been questioned. He recognized the fact that various grades of severity may be met with, but presented the clinical course of the severest type, in order to emphasize the symptoms more forcibly, and to facilitate the diagnosis. From his study of the subject, the writer has been led to the conclusion that Ludwig's picture, while it may accurately describe the average case of his time, will not answer so well for that of the present. That is to say, the gangrenous or putrefactive conditions are not met with so frequently nor do they reach the advanced stage when present, which seems to have been the rule in his day. This change is due, probably to the fact that expectant treatment is now much less frequently employed. The progress of the infection is arrested earlier by more prompt incision and drainage. The irregular septic temperature, profuse sweats, delirium and progressively profound typhoid state, are by no means so common now. With few exceptions modern surgical treatment will arrest the progress of the infection or the patient will die in less than 10 to 12 days. Since his clinical picture appears to be the standard, and from time to time is given in more or less detail in journal articles, the writer wishes to present it here in order that he may apply to it briefly his own interpretation of the symptoms.

"The condition is ushered in with the usual symptoms

of a rheumatic or erysipelatous angina, *i.e.,* slight fever, repeated chills, headache, coated tongue, etc., sometimes with slight difficulty in swallowing. At the same time there develops a unilateral or bilateral hard swelling usually of the cellular tissue surrounding the submaxillary gland, sometimes of that about the subligual or parotid. Extension of the process occurs in all directions along the cellular tissue, toward the chin and the opposite side, and toward the larynx, and the parotid, forming a considerable swelling. The intermuscular tissue and even the muscles become involved. The sublingual tissues form a hard, congested swelling, arranged like a cushion just inside the inferior maxillary bone; and the tongue is pushed upward and backward. The mouth is opened with pain and difficulty. Movements of the jaw, swallowing and speaking are considerably disturbed. The skin is movable during this local stage (4 to 6 days), the general condition is little disturbed, and the fever moderate. Soon the skin becomes reddened, the sublingual swelling softens and at times shows crepitation. Occasionally fluctuation appears to be present as though pus were there. But this is not the case. Soon an opening occurs in the floor of the mouth discharging a thin grayish or reddish brown, offensive fluid, which more and more assumes in character the discharge of a putrefactive process. The constitutional symptoms now become more severe, *i.e.,* the fever is higher, sleep is disturbed, profuse sweats and delirium appear and the typhoid condition becomes more profound. Deglutition remains difficult, although the swelling becomes less tense, and suggests improvement. Dyspnoea sets in and increases, and probably indicates an affection of the nervous system rather than a mechanical obstruction of the respiratory tract. Perhaps this is due to effusion into the chest. The symptoms develop with alarming rapidity and are characteristic of a putrefactive typhoid process. Death from coma and lung paralysis occurs in 10 to 12 days from the commencement of the disease."

The following points he considers to be diagnostic:

"1. The insignificant inflammation of the throat, which often disappears entirely after the first few days, and which if it persists may be looked upon as superficial.

"2. The 'wood like' hardness of the swelling, which does not pit on pressure.

"3. The hard sublingual swelling, forming a ring just within the lower jaw, reddish or bluish in color.

"4. The sharp limitation of the indurated tissues which are surrounded by uninvolved healthy connective tissue. The slight involvement or more often lack of involvement of the glands although the inflammation attacks the connective tissue around the gland."

The writer believes that there will be nothing obscure in this clinical picture, if we take into account the anatomical facts to which he has already called attention, and the known facts concerning the usual rapidly spreading infection of the connective tissue. It is assumed that we are dealing with a case in which the infectious germs have gained entrance through some focus in the mouth and the first signs of cellulitis have appeared in the submaxillary region, where Ludwig located them. In the ordinary case of infection arising in this way, the germs pass by the lymphatic vessels to the glands, causing no trouble in the vessels. As soon as they reach the gland they begin to produce inflammatory changes and being confined the inflammatory material produces pain. If the infection is mild or moderate, it will probably remain limited within the capsule of the gland long enough to permit a localizing barrier of lymph to be prepared. In this way is developed the ordinary localized lymphadenitis which is so common in this region. Occasionally such a localized swelling will take on rapid growth and become diffuse, *i.e.,* the infection breaks through the barrier of lymph and spreads quickly along the cellular tissue. A localized osteo-myelitis, for example in the tibia, may break through the periosteum and set up an overlying cellulitis so rapidly as to confuse the diagnosis with that of erysipelas. Much more rarely than in the localizing cases and most characteristically in

streptococcic infections, the process extends from the gland to the cellular tissue so rapidly that its glandular origin is overlooked. The fever, chills, headache and early difficulty in swallowing may be accounted for by this inflammation, or it might be due to the preliminary angina present in some cases. The characteristic extensive swelling of the neck is due to extension along its cellular tissue. The superficial fascia offers no hindrance to it in any direction, while the connective tissue in the submaxillary fossa is abundant and lax and freely continuous with the same tissue in the retromaxillary and submental regions. In Ludwig's description and in almost all the reported cases the sublingual swelling and elevation of the tongue are referred to after the submaxillary swelling has been mentioned, which is to be explained by extension through the opening in the floor of the mouth, already described. The submaxillary swelling is hard so that its extension should be hard also. The skin is at first movable and not inflamed because the process begins deeply in the lymph gland and invades the adjacent connective tissue with great thickening or swelling of the latter before it reaches the skin, which is inflamed later. The invasion of the floor of the mouth by the inflammation and its extension to the pharynx and larynx in the writer's opinion, will satisfactorily explain the troublesome dysphagia and the dyspnœa. Its early invasion of the larynx and in some cases of the lungs will explain the rapidity and certainty with which an unchecked case goes on to a fatal termination.

The following description of Ludwig's Angina, taken from Poulsen, is that given by Boehler more than twenty years ago, and was based upon a study of 35 cases. It is repeated here because of its brevity and simplicity, because it will be of value for comparison with Ludwig's more detailed account of the clinical course, and because, as the writer views it, it is nothing more than the description of a submaxillary lymphadenitis with periglandular extension along the connective tissue, due to a virulent, probably, pyogenic infection.

"Under febrile and slight disturbances in swallowing there develops in an otherwise healthy person, in the region of the submaxillary gland, of one or both sides, an indurated, in the beginning indolent, somewhat movable tumor, which appears to proceed from the connective tissue around the submaxillary salivary gland. The overlying skin is natural and movable. The swelling which is at first the size of a hen's egg, extends more and more over the side of the neck reaching as far down as the sternum. There is an infiltration of the connective tissue which surrounds the muscles of the neck, and extends to the alveololingual sulcus, the soft palate and the pharynx. The tongue from the great swelling in the floor of mouth is elevated and pushed to the opposite side. Generally as the process extends farther, the skin of the submaxillary region becomes oedematous and dark red. Perforation occurs in the oral cavity, and in the submaxillary region with the escape of brown fetid pus. The breathing becomes laborious, and the patient dies in a state of septic intoxication."

Autopsies.—Of the writer's 104 collected cases, autopsies were reported in connection with 25. In only 16 of these is there a description, direct or indirect, of the condition of the larynx. Those in which such reference was found are briefly reviewed here. Thirteen showed positive laryngeal involvement, two were negative, and in one (Zillner), the writer would infer from the vague reference to the involvement of the mucous membrane, that oedema of the glottis was present. Of Cartonli's case it was said that the larynx, trachea, pharynx and oesophagus were not damaged. This patient on the day of his death had symptoms of pneumonia with orthopnoea, so that it is more than probable that death resulted from respiratory failure. The blackish condition of the subcutaneous tissue with ichorous pus oozing from its cut surfaces, was said to have extended into the tissues above the hyoid bone and under the jaw and as far as the posterior surface of the pharynx, destroying the surrounding tissues. This would have brought it so close to the larynx that the

question naturally arises as to whether the condition of the pharynx and larynx was determined from their outer surfaces through an external incision, or whether the internal surface was exposed freely and the mucous membrane directly inspected. Of Macaigne and Vanvert's case it was said that the larynx was sound and that the aryo-epiglottic folds were not oedematous. Yet in this case dyspnoea developed on the second day, about 24 hours after the onset of the condition, and the patient died a few hours later, although his general condition did not seem to be very grave. In the writer's judgment, the clinical side of the case points strongly towards oedema of the glottis. Death could hardly have been due to septic intoxication. If oedema of the glottis were present in these two cases or if these were excluded from this group, the strongest kind of a case would be made out for invasion of the respiratory tract, more especially of the larynx, as the essential cause of death in the typical Ludwig's Angina.

CASE I.—HEIN.—Man, 32 years, military officer, robust constitution. Admitted to the hospital, Aug. 15, 1823, for an indurated submaxillary swelling of the left side, which had begun some days before in the region of the submaxillary gland. Movements of tongue limited and painful. Deglutition disturbed from beginning. Could swallow only liquids. In the bucco-pharyngeal cavity, no redness nor inflammation. Respiration normal. Later swelling reached clavicle. Increased difficulty in respiration. At end of some days, softening in lowest part. Incisions here give fetid purulent liquid. No relief to patient. Death from asphyxia, Aug. 28th, the 13th day of the stay in the hospital.

Autopsy.—Some fibres of gangrenous cellular tissue in abscess. Surrounding tissues form a putrid mass which communicates at the level of the angle of the jaw, with pharynx. Complete mortification of muscles above and below hyoid bone, and muscles of larynx nearly completely destroyed. Mucous membrane of larynx and trachea dark colored, like gangrene. Ventricles of Morgagni contained thickened, grayish black mucosites. Other organs normal.

CASE II.—HEYFELDER.—Female, 37 years, pale, cachectic and gouty for 7 years. Towards end of August, 1837, exposed to repeated chills, which were followed on Aug. 30 by moderate fever, heat, lumbago and acute pain in right side of neck. Aug. 31, swelling in region of right submaxillary gland, was the size of a goose's egg. Movements of head disturbed. Deglutition painful. Tonsils swollen, but not inflamed. Isthmus of fauces red. Sept. 1, swelling much greater, and of "woody" hardness. Mouth opens with acute suffering. Tongue projects posteriorly. Speech and deglutition difficult but not painful. Examination of posterior part of mouth impossible. Tumor under tongue. Very considerable prostration. Sept. 3, everything much worse, and patient very weak. Cannot expectorate abundant mucus which collects in mouth. Jaws separate only a few lines. Deglutition nearly impossible. Delirium. Sept. 5, suppuration and crepitation on palpation. Deglutition and opening of mouth better, but general condition worse. Puncture at a soft spot, with escape of abundant fetid pus. Sept. 6, median incision from chin to hyoid, with escape of fetid pus, gas, and gangrenous debris. Sept. 8, coma. Sept. 9, death.

Autopsy.—Affected region a dark foul mass. Salivary glands pale bluish at periphery, normal in their depth. Cervical portions of vagus and recurrent nerves were a dirty red. Muscles had lost their relations in consequence of the gangrene of the cellular tissue which surrounded them.

Mucous membrane of tongue, pharynx and nose, slightly inflamed and covered with grayish mucus. Tonsils healthy. Mucosa of larynx and trachea presented a livid appearance and was covered with foul adherent mucus. Small abscesses were disseminated in the inferior lobes of both lungs. Heart soft and flabby. Turbid fluid in pericardium. Liver, spleen and right kidney softened.

CASE III.—BERMAN.—Female, 18 years, habitual good health. Complained of bad second left molar on Aug. 26, 1838. Aug. 27, on same side, appeared a hard, parotid swelling. Alveolo-dental periostitis, opposite the carious tooth. Aug. 28, abscess opened itself on external side of diseased tooth and discharged a mass of fetid pus. External swelling does not diminish, and it is extremely hard. Sublingual swelling forms a hard ring around the tongue, which is pushed up against the roof of the mouth. Voice harsh and muffled. Considerable dyspnœa. Aug. 29, spontaneous opening under the tongue with escape of fetid pus mixed with blood. External inflammation progresses to opposite angle of jaw and to sternum. Deglutition and respiration very troublesome. Aug. 30, bad night. Delirium. Considerable dyspnœa. Swelling invades greater part of thorax. Aug. 31, prostration increases. Extreme dyspnœa. Deglutition impossible. Sept. 14, stupor develops, finally coma and death.

Autopsy.—36 hours after death. Complete mortification of muscles from chin to sternum. Impossible to recognize their structure. Voluminous cellular debris and a considerable mass of fetid pus.

Epiglottis destroyed. Mucous membrane of larynx and trachea swollen and covered by viscid mucus. Mucous membrane of pharynx and œsophagus is blackish in color. On internal surface of inferior maxilla is a fistula, communicating with the gangrenous focus.

CASE IV.—ZILLNER.—M. B., 30 years, insane for 3 years. As the result of a cold there developed a hard non-painful swelling of the left cheek. About seventh day, chills, agitation and delirium. Ninth day, spontaneous opening in the mouth near angle of jaw, discharging abun-

dant sanious pus. Swelling continued to spread anteriorly and toward the clavicle. Thirteenth day, second spontaneous opening externally below angle of jaw. Fourteenth day, death.

Autopsy.—Crepitation on pressure over the whole swollen region. Overlying skin bluish red. Subcutaneous tissue granular, while hard and resistant. In the suprahyoid region, below the floor of the mouth, all the organs, mucous membrane, cellular tissue and muscles are transformed into an extremely putrid mass. Periosteum destroyed and inferior maxilla denuded, in the greater part of its extent. In the place of the submaxillary and sublingual glands there is a large cavity filled with pus, its walls being made up of connective tissue. The sheath of the sterno-mastoid is filled with pus.

Case V.—Finger.—Woman, 29 years, presented a Ludwig's Angina, on the twelfth day of a typhus fever. In the morning the sublingual gland and the surrounding cellular tissue were much swollen. Tongue considerably infiltrated and pushed against the roof of the mouth by the prominent swelling in the floor. Respiration much disturbed, causing fear of an œdema of the glottis. Patient died in the evening, presenting all the symptoms of suffocation.

Autopsy.—Cellular tissue around the submaxillary and sublingual glands on both sides of the neck, infiltrated with a yellowish purulent serum, and much swollen. Muscles of the velum palati, mucous membrane of the pharynx and larynx and all the corresponding half of the tongue are much swollen and infiltrated with a pale purulent serous fluid. The infiltration descends as low as the sternum.

Case VI.—Doig.—R., 22 years, soldier. Has had swelling of the neck for some days. When admitted, Feb. 4, 1876, he had a painful swelling extending over the whole of the left side of the neck. Parotid, submaxillary and sublingual glands of the same side, much swollen and very painful. On the right side the submaxillary gland is equally indurated, but is not of the same size as on the left side. Floor of mouth elevated and tongue pushed upward. Hypersalivary secretion. Considerable disturbance of deglutition. Intense dyspnœa. Insomnia. Paroxysmal anxiety. Skin over swelling normal, except that it is a little œdematous near angle of jaw. Puncture here, no pus. Death by asphyxia, Feb. 8th.

Autopsy.—43 hours after death. All the left side of the neck, from inferior maxilla to clavicle, is a semiliquid, extremely fetid mass. All the tissues of this region, glands, muscles and cellular tissue, are nearly completely destroyed. The portion of the jaw, adjacent to the destroyed submaxillary gland, is denuded of its periosteum. In the buccal cavity, one finds the mucosa, epiglottis and vocal cords, swollen, red and covered with mucus. There are ulcers of the tonsils.

Case VII.—Cartonli.—Man, 50 years. Admitted to hospital on evening of October 4th, 1879. Hard right submaxillary swelling. Speaks and opens his mouth with difficulty. It was learned from his companions, that on the evening before, he was feeling well and that the swelling had not been there for more than two days. Oct. 5th, temperature in morning

and evening was 39.5. Trismus present. Deglutition impossible. Esophageal sound introduced into stomach to relieve dysphagia but did not meet with any obstacle. Abundant salivation. Lungs congested. Oct. 6th, temperature 39.5. Delirious during night. During day appeared symptoms of pneumonia with orthopnœa. Tongue protrudes between teeth. Death during night.

Autopsy.—Left lung hepatized. Right submaxillary region hard and œdematous. Skin cyanosed. Subcutaneous tissue blackish. Small quantity of ichorous pus oozed from cut surfaces. This condition of the tissues extended more or less into the tissues above the hyoid bone and under the lower jaw. The submaxillary gland is hypertrophied, sclerosed, grayish, and on section gives issue to an ichorous material. The ichorous infiltration extended deeply internally and below to the posterior surface of the pharynx, destroying the surrounding tissues. Larynx, trachea, pharynx and esophagus not damaged.

Case VIII.—Baker.—J. A., 25 years. Admitted to hospital, Feb. 6, 1862. When first seen, he was suffering from "swelled neck and great difficulty in breathing." Great swelling on left side of neck and smaller swelling on right side, extending toward median line. Skin dark purple, very brawny and here and there is boggy. Higher up under the chin is an indistinct sense of fluctuation. Mouth open. Tongue against the roof of the mouth, and of normal size, and consistency. Mucous membrane of floor of mouth elevated to level of free edges of lower teeth. Fauces cannot be seen. Dyspncœa began night before admission, when he could not breathe without being propped up in bed. Soon after admission, he was found not to be breathing. Immediate tracheotomy. Pulse stopped.

Autopsy.—Seropurulent, necrotic infiltration of cellular tissue and muscles. On opening trachea, the rima glottidis is found nearly closed, with effusion of semipurulent matter into submucous tissue. This extended to the epiglottis. Glandulæ concatenatæ, submaxillary and parotid glands, much enlarged.

Case IX.—Bickersteth.—Man, 40 years. On admission, speech very difficult and indistinct. Breathing embarrassed. Great swelling beneath jaw. Floor of mouth raised and tongue pushed upward and backward against roof of mouth. Examination of fauces impossible. Surgeon notified and came immediately, but patient died a few minutes before he arrived at the hospital. Patient had been seized with rigors and severe pain in submaxillary region three days before admission. Shortly afterwards, there was swelling from the lower jaw to the sternum. Skin was normal in consistency, color, and mobility, but was tense.

Autopsy.—Shortly after death, a puncture with a tenotomy knife was made in the floor of the mouth, when a small quantity of air and some sero-sanious fluid escaped. All the connective tissue around the trachea and between the muscles is infiltrated with a seropurulent fluid, extending upward to the root of the tongue and downward into the anterior mediastinum. The submucous cellular tissue is similarly affected, producing anteriorly, sublingual tension, and posteriorly, œdema glottidis and general œdematous laryngitis.

Case X.—Michel.—Man, 38 years. Admitted to hospital, Dec. 2d, and died same day. Vigorous health. Duration of disease, four days. Suprahyoid, median and lateral swelling as high as ear, more marked on right side. No fluctuation. Constriction of jaws. Swelling of buccal floor. Marked dysphagia. Dyspnœa marked also, but no threatening of suffocation. Abundant foul saliva. Speech embarrassed. Temperature at 4 P.M. 39. Origin in carious tooth, causing a submaxillary swelling, which rapidly increased. At 6 P.M. median suprahyoid incision, only blood escaping. Patient seemed slightly relieved, but at 11 P.M. he died suddenly of suffocation.

Autopsy.—24 hours after death. Only the larynx and adjacent organs removed. Cellular tissue gangrenous only on right side. Coffee-cup-full of phlegmonous pus in right retromaxillary region, where the lymph glands are very large. Submaxillary and sublingual glands congested and slightly indurated. Pharyngeal mucosa red and slightly thickened in its retrolaryngeal portion. Tonsils slightly enlarged with some points of intraglandular suppuration. In cutting through the anterior wall of the pharynx there is seen a whitish, soft tremulous œdema of the supraglottic portion of the larynx. The aryteno-epiglottic folds, the superior vocal cords and the epiglottis are double their normal size. The uvula is hypertrophied and œdematous.

Case XI.—Gibson.—Man, 49 years. Came to out-patient department with swelling of neck below lower jaw, which began at noon of the previous day, when there was observed a scab on the right side of the neck below the jaw. At that time the swelling was enormous, extending to the chest and as high as the zygoma. Is a heavy drinker and had been drunk the evening before the trouble began. Perceptible enlargement of the salivary glands. No lymphatic glands could be felt. Skin normal in color. No pain on firm palpation at any part of the swelling. Floor of mouth considerably thickened, but tongue was not swollen or raised to any perceptible extent. Slight dyspnœa. Just beneath the jaw on the right side is a scab, ¾ in. in breadth with a pustular margin, very like a variola but having no areola. Did not feel ill enough to wish to stay in hospital, but on being warned, he consented to remain. Admitted about 1 P.M. At about 3 P.M. of the same day, he suddenly became unconscious and intensely dyspnœic. Tracheotomy done immediately, and artificial respiration carried out. Recovered and respiration became regular and rhythmical. Large tracheotomy tube introduced. A little later a median incision was made from the chin to the hyoid bone, dividing the structures almost to the floor of the mouth. Thin serous discharge. No pus. Crepitation under the skin of the chest. At 10 P.M. dysphagia, but no difficulty of respiration. Next day, 9. A.M., good night, increased swelling toward the chest. No fluctuation. No pain. 11 A.M., very feeble. Dyspnœa and considerable cyanosis of lips and face. 2.40 P.M., rapidly becoming livid and respiration more rapid. Gradually became comatose and died at 3.15 P.M.

Autopsy.—Emphysema detected from eyelids to nipple. Well marked œdema glottidis, the œdematous tissue partly resembling that found elsewhere, though perhaps not quite so firm. Infarctions found in lungs. Bacteriological examination showed no specific pathogenic organism. (It was of this case that Lockwood said that by different methods he later found the streptococcus in the tissues.)

Case XII.—Poulsen.—At midday, day before yesterday, patient observed a swelling of right side of his face and difficulty in swallowing. The swelling extended farther and farther toward the submental and parotid regions. To-day (3rd day), mouth can be opened only slightly, just enough to permit the introduction of a finger, with which the isthmus of the fauces can be felt to be free. Floor of mouth considerably swollen on both sides. Swelling œdematous but non-fluctuating. No carious teeth. When the beard was being shaved, suddenly he became dyspnœic. Sat upright in bed and died in a few minutes. This was 3 hours after admission, *i.e.*, 2 days after the beginning of the attack.

Autopsy.—Both parotid regions and upper part of neck considerably swollen. Some œdema of subcutaneous tissue. Swelling most marked in submental and submaxillary regions. Foul, grayish, rather thick fluid between the muscles passing from lower jaw to hyoid bone. Salivary gland somewhat thickened and its tissue to a slight extent infiltrated with this fluid. The glandular tissue seems unchanged. Same infiltration but with a clearer fluid invades the connective tissue of the neck. In the submaxillary salivary gland the infiltration of the connective tissue is somewhat more marked, and has the appearance of connective tissue pus and an inclination to abscess formation. On the right side the gangrenous pus infiltration extends between the sternomastoid and sternohyoid and sternothyroid muscles to just above the thyroid gland. It also extends along the blood vessels to the lower part of the thyroid gland. On the left side the pus infiltration extended only a little below the submaxillary gland. The lymph glands on both sides were somewhat swollen, but without pus. The pus infiltration did not extend to the tongue nor to the sublingual gland. Tonsils not swollen.

Enormous œdema of the uvula, and of the mucous membrane of the pharynx, particularly in the laryngo-pharyngeal sinus and especially in the aryteno-epiglottic folds. There was œdema also on the anterior surface of the epiglottis, a little on the posterior surface. Very marked œdema on the sides of the larynx, even as far as the vocal cords, especially on the left side, where the upper surface of the left cord was very prominent. In the left parotid gland there was œdema, and foci of pus infiltration in the connective tissue.

Case XIII.—Poulsen.—Man, 54 years, presents himself with a virulent phlegmonous swelling involving the left cheek, left retromaxillary region, and the lateral region of the neck. Some swelling of the eye-lids. Can scarcely swallow, and is hoarse, which symptoms are of a few days duration. No real difficulty in respiration. Mouth can be opened only slightly. Numerous stumps of teeth present. Fauces cannot be inspected. As far as one can palpate there is found considerable swelling of the left side. Epiglottis cannot be reached. Patient says that condition has developed in last three days. Before that he had suffered for a half day

from difficulty in swallowing. Temperature 40.5°. Under chloroform, incision made in submaxillary region parallel to jaw. Finger worked into a soft stinking pus infiltration of the connective tissues, as high as the mylohyoid muscles and posteriorly opposite the parotid as deep as the pharyngeal wall. At no time did pus flow out, but there escaped from the cavity a putrid offensive odor. Irrigation, iodoform packing. Following day, temperature 41°/39.2°. Respiration freer, and swelling subsided. Still a firm infiltration in submaxillary region. Gauze removed and found to be putrid and stinking. Another incision made downward. No denudation of maxilla. Patient collapsed. Toward evening, temperature went up to 41.1°. No dyspnœa.

Autopsy.—Diagnosis: Gangrenous phlegmon of submaxillary region. Pericœsophageal and laryngeal phlegmon. Hyperplasia lienis. In the larynx: considerable swelling and infiltration, especially on the left side. The swelling has a dusky, gangrenous appearance, as the infiltration itself, through an incision shows a dusky, gangrenous tissue. Above the left vocal cord is an abscess the size of a pea, with thick yellowish pus, in which were found numerous micrococci of various kinds, especially long chains. The greatest infiltration is found in the intermuscular connective tissue on the left side of the trachea and larynx, close to the internal jugular vein. The infection, here, extends to the left tonsil and the upper surface is the seat of gangrenous ulceration. The process extended downward around the œsophagus, where almost to the heart, was found a thick rather firm, dusky infiltration of the connective tissue, between the mucosa and the muscularis upon the posterior and left side. In the left submaxillary region, the edges of the incision were almost black and gave a very offensive odor. (It is interesting in this case to observe the difference between the clinical and post-mortem evidence of involvement of the larynx.)

Case XIV.—Ombredanne and Keim.—Man, 26 years. Dec. 29th, at 9 P.M., patient arrived at hospital, nearly asphyxiated. It was necessary to carry him. The swelling began on the 26th, at the same time as an inflammation about a carious tooth. Dec. 28th, dysphagia developed and already dyspnœa was present. Patient continued, nevertheless, to work. Respiration became more and more difficult. On admission, 29th, there was considerable suprahyoid, hard swelling, predominating on the left side. No fluctuation. Floor of the mouth slightly elevated and tongue swollen. Mouth full of mucus. Pulse 152. Submaxillary incision made 6 cm. long; 3 to 4 grammes of fetid pus escaped with the blood. While on operating table, respiration became more difficult and stopped. Tracheotomy and artificial respiration revived him. Dec. 30th, right side of neck more swollen and is enormous. Temperature 39.2°, pulse 140. Patient calm. Swallows liquids. Dec. 31, incision posterior to angle of jaw on left side. No pus, only blood. Temperature 39°, pulse 161. General condition worse. From first incision bloody serum and bubbles of gas can be expressed. Jan. 1, swelling invades base of neck and thorax, where crepitation can be felt. Jan. 3, swelling occupies whole thorax. Jan. 4, incision in right side of neck, evacuates some

drops of pus with blood and gas. Death at 7 P.M., with an intense dyspnœa. Face cyanosed. Tracheotomy tube is always in place and working freely.

Autopsy.—Pleural cavities contain considerable quantity of bloody liquid, and at the fissures were fibrinous deposits. Lungs engorged with blood and serum. Nevertheless, they crepitated under the finger. A portion of the tongue, floor of the mouth and soft parts of the neck were removed. The whole of the floor of the mouth, especially at the angle of the jaw, was transformed into a gangrenous mass. The muscles of the tongue are absolutely preserved and retain their red color, but the pus has infiltrated in front of the trachea to behind the sternum, where there was an extensive discoloration. The carotid glands were in full suppuration.

The epiglottis was œdematous, turgescent and curved like a horse shoe; the two ends touching each other. The aryteno-epiglottic folds were equally infiltrated, especially the right which was nearly a centimetre thick. The glottis and trachea were red and injected. No subcutaneous emphysema around the trachea wound. Bacteriological examination showed: streptococci, and staphylococci, the latter predominating. Injection of the pus into one of the lower animals gave rise to an ordinary phlegmon without the development of gas.

Case XV.—Macaigne and Vanverts.—L., 62 years. Entered hospital, March 12, 1896. Previous health good. Found on waking on morning of March 11, that his neck was swollen and painful in the suprahyoid region. Yet he worked all that day. At 5 o'clock he was compelled to go to bed from fatigue, chills and high fever. At 9 P.M., March 12, swelling was considerably increased. The lesion is deeply seated because the superficial layers of tissue move easily on it. Skin normal. Swelling of "woody" hardness. Floor of mouth swollen and indurated. Mucous membrane red. Tongue pushed upward and backward. Carious teeth. Respiration difficult because of narrowing of isthmus of fauces by elevation of tongue. Dyspnœa increased on slightest effort. Speech difficult. Some constriction of jaws. Pulse rapid. Temperature 38°. General condition does not seem very grave. At 11 A.M., a few whiffs of chloroform were given and a long median incision made. This was deepened to the mucosa of the buccal floor. Neither a serous nor a purulent collection found. Abundant blood escaped. Dressing applied and patient taken back to bed. A quarter of an hour later he was dead.

Autopsy.—46 hours after death. Inspection showed that no pus collection had escaped the bistoury. Larynx sound. Aryo-epiglottic folds are not œdematous. Lungs are normal. All the organs are congested, but present no other lesion. (The writer questions the post-mortem report of a sound larynx in this case. Death in less than 36 hours was too rapid for septic intoxication. The patient worked nearly the whole of the first day. Temperature was only 38° (100⅖ F.), and general condition said to be not very grave, at end of the first day. Floor of mouth invaded by inflammation. Speech difficult and dyspnœa on first day, increased on slightest effort. Sudden death.)

CASE XVI.—BIEDERT AND ROBERTSON.—Male, 22 years, admitted for typhoid fever of two weeks duration. One week later, the typhoid being moderately severe, patient began to complain of some dyspnœa. A swelling developed very rapidly on the left side of the neck, just below the angle of the jaw, hard and tender. Dyspnœa increased and examination of throat showed an œdema of the whole of the pharynx and larynx and epiglottis, the larynx being almost entirely closed. Six hours after the onset the dyspnœa had become so severe that tracheotomy became imperative. This relieved him. Temperature 103°. External swelling continued to increase very rapidly and was very tender. About ten hours after the onset he died suddenly. Cause of death not clear.

Autopsy.—Four hours after death. Typhoid ulcers in small bowel, particularly near the termination of the ilium. Tissues of the neck very œdematous and swollen, but no evidence of breaking down. Mucous membrane of larynx, particularly about the left vocal cord and epiglottis, was œdematous, greatly swollen and almost purple in color.

Bacteriological Examination.—Cultures taken from spleen by Dr. Ghriskey showed the Eberth bacillus. Unfortunately no cultures were taken from the cervical region, but a microscopical examination of the swollen laryngeal tissues showed a pure streptococcus infection.

A further study of the 104 collected cases, and the writer's two, gave the following results.

Age.—The phlegmon was present at birth in one. In another it began on the 6th day; in a third at 3 weeks; and in a fourth at 6 months. There were 7 cases between the ages of 1 and 10 years; 6 between 10 and 20 years; 33 between 20 and 30 years; 15 between 30 and 40 years; 12 between 40 and 50 years; 10 between 50 and 60 years; and 5 between 60 and 70 years. In 9 cases the only reference to age, was that the patients were adults, and in 5 there was no reference at all to age. The greatest number, therefore, occurred between 20 and 30 years. Poulsen's 251 submaxillary abscesses, showed about the same proportion in this period. Between 16 and 30 years, he regarded as the age of carious teeth.

Sex.—Of the 106 cases, there were 76 males, and 20 females, while in 10 cases the sex was not mentioned. Males being more exposed to changes in the atmosphere are, probably, more frequently the subjects of angina; while among the poorer classes they, probably, give less attention to their

teeth and are, therefore, more frequently sufferers from dental caries, than females.

General Health.—Murchison reported an epidemic of Ludwig's Angina in the Hebrides, and found that previously impaired health was associated with most of the cases. He reported none of his cases, individually, so that none is included in the writer's list. His view has received little or no support in the literature. Of the writer's 106, there were only 15 cases in which the general health, previous to the beginning of Ludwig's Angina, was considered to be in any way impaired. Two of these were gouty; one insane; 5 alcoholic; one albumenuric, alcoholic and diabetic; one worn out by privation and long walking; one was in the early stage of secondary syphilis; one, probably, had measles just preceding the beginning of Ludwig's Angina; in one the trouble developed during typhus fever; and in 2 during typhoid fever.

There were 21 cases in which the general health was said to be good; and 10 others in which, from the vigorous occupations followed by the patients and the absence of any reference to previous health, it may be assumed that it was not essentially impaired. In 60 more there was no mention of the general health previous to the attack, or of anything which would indicate that it might have been impaired. The natural inference is that in these it was normal. In 90 of the 106, or 85 per cent. of the cases, therefore, it may be assumed that previous to the beginning of Ludwig's Angina, the general health was practically normal.

Primary Focus of Infection.—Dental caries was noted in 36 cases, in 4 the wisdom teeth being involved. So far as reference to special teeth is concerned, they were always molar or wisdom teeth, *i.e.*, those nearest to the submaxillary region. Angina was present in 11 cases, and in these there were no references to carious teeth. In one there was a wound of the mucous membrane just posterior to the incisor teeth, which had been broken by the kick of a horse. In one there was a wound of the chin. In another there had

developed infection in the wound made by cutting a tongue tie. In one of the writer's cases the infection began in a similar wound resulting from a gun shot injury, producing a fracture of the lower jaw. In two cases there were ulcers on the side of the neck. In two others, otitis media seemed to be the primary focus. In one the trouble began with a peritonsillar abscess, and in one secondary syphilis was present, probably, giving lesions in the mouth and on the skin, which would account for mixed infection of the lymphatic glands. In 49 cases there was no mention of a primary focus other than the cellulitis in the neck.

Swelling in floor of mouth.—In 81 cases this symptom was noted. In 8 one is left in doubt as to its presence by the description of the case, while in 17 no reference is made to it. From the associated symptoms the writer would infer that it was present in most of these 25 cases, if not in all. With the jaws so close together that one can not see the floor of the mouth, without special efforts, and the finger can be introduced only with difficulty, this symptom might easily be overlooked.

Difficult speech.—Disturbed speech was mentioned only in 38 cases. In 4 the patients were too young for speech. The absence of mention of this symptom in so many cases is a matter of little importance since it is not a valuable symptom. Speech must be more or less disturbed, whether the case be one of localized submaxillary cellulitis or true Ludwig's Angina, from the failure to open the mouth and the limitation of the movements of the tongue, due to the fixation of the suprahyoid muscles going to it.

Dysphagia.—This symptom was mentioned only in 68 cases, but was said to be absent in only one. Like disturbed speech it must have been present in many more, and for the same reasons.

Dyspnoea.—This symptom was present in 81 cases, was not mentioned in 20 and in three was specifically said to be absent. In two others there were syncopal attacks.

The writer has tried to show that Ludwig's Angina,

probably, kills in the great majority of cases by invasion of the respiratory tract, first of the larynx and later in some cases of the lungs. The fact that dyspnoea could be overlooked in 20 cases and could have been reported absent in 3 cases, out of a total of 106, implies that in a fair percentage of cases death occurs without oedema of the larynx. The writer, however, has become convinced during his study of this subject that not all cases of oedema of the larynx give prominent and positive symptoms upon which the diagnosis can be easily made.

In the first place the two cases showing syncopal attacks, without any mention of dyspnoea were suspiciously like cases of laryngeal involvement. Death from syncope occurred in Nelaton's case in which the submaxillary swelling was enormous and became bilateral, and the floor of the mouth was involved. In one of Huguet and DeBovis' cases, syncopal attacks occurred in a new born infant, but there was in this, as in Nelaton's, no mention of dyspnoea. There was sublingual swelling also. That dangerous involvement of the larynx may occur without the development of dyspnoea is shown by Poulsen's case (see autopsy case No. XIII). There was said to be no dyspnoea in this case, and yet the autopsy showed considerable swelling and infiltration of the larynx, especially on the left side, dusky and gangrenous in appearance. Above the left vocal cord was an abscess the size of a pea, and the process extended downward around the oesophagus in the submucous tissue almost to the heart. If dyspnoea can be absent in a case of this kind, then it is probable that oedema of the larynx existed in many of the 20 cases, in which dyspnoea was not mentioned.

Parker, writing on tracheotomy in laryngeal diphtheria, says: "Membraneous laryngitis begins in one of two ways, primarily in the larynx and by extension to the larynx. In the former the chief symptoms are those of suffocation outweighing and hiding all others. In the latter the laryngeal symptoms are preceded by those of depression and blood poisoning. As a rule this spread (to the larynx) is *very*

gradual and *very insidious*. In consequence of the antecedent blood poisoning, but chiefly of the very gradual onset of the disease, the body becomes reconciled to its deprivation of oxygen; hence the suffocative symptoms, which are so prominent and so distressing in the other variety, are less marked, indeed often absent in this. Sometimes medical practitioners have themselves underestimated the gravity of the disease on account of this apparent absence of discomfort in their patients."

J. Solis Cohen, writing on the symptoms of oedema of the larynx, says: "Acute oedema of the larynx occurs so suddenly at times that the subject perishes without any premonitory symptoms whatever. Van Swieten mentions a case, of death with sudden change in the voice, while dining. Morgagni mentions a similar case, in a physician, who suddenly became hoarse, and died at once. Porter knew of two young men found dead from oedema in the morning, without any complaint having been made by them the night previous. Ruehle mentions a young man with swollen tonsils and overheated by dancing, found dead in the morning from oedema which had suffocated him without awakening him; and likewise the case of a servant girl, slightly hoarse, who went out lightly clad in the morning, and was suffocated while going up stairs on her return. Roger, while an interne at Hôtel-Dieu, was summoned to an attendant in an adjoining ward, who died of suffocation before he could be reached; and yet there had been no complaint save of a sore throat, so slight as not to interrupt the man's work in the hospital. These instances of sudden death certainly seem to indicate a sudden occlusion of the glottis from spasm of its constrictors, rather than a mechanical death from serous effusion. It is quite probable that the oedematous condition may have existed for some hours or days undetected and unsuspected, and that some sudden inspiration of dust or of saliva, has produced an immediately fatal spasm." The writer believes that there is abundant proof in the preceding statements, to show that a patient may die from oedema of the larynx in Ludwig's

Angina, without recognition by the attending physician of the laryngeal condition.

Diagnosis.—The first essential in making a diagnosis of any pathological condition is to have a definite conception of what that condition is. The writer's idea of it has been so fully set forth, already, that little more need be said on the subject. It may be permitted him to repeat that what Ludwig described was a virulent cellulitis beginning in the submaxillary region, rapidly spreading to the adjacent connective tissue of the neck and then into the floor of the mouth and pharynx; in consequence of which the patient's life is threatened; partly from septic intoxication, but chiefly from invasion of the respiratory tract, *i.e.*, the larynx, primarily, and in some cases the lungs, secondarily. Those cases in which the phlegmonous process begins in the throat in the immediate vicinity of the larynx have been purposely excluded, for reasons already given. Those beginning in the floor of the mouth are not so easily disposed of; and these the writer is inclined to include with those beginning in the submaxillary region and invading the floor of the mouth, secondarily. Those beginning in the floor of the mouth practically always extend to the submaxillary region, secondarily; as may those beginning in the throat, but the latter not so constantly nor so quickly for obvious reasons. It will thus be seen that a Ludwig's Angina is not actually present until the sublingual phlegmon has developed. Its diagnosis, therefore, depends upon the recognition of the latter condition.

While in a case of submaxillary cellulitis still localized to the tissues of the neck, it would be folly to wait for the signs of Ludwig's Angina to develop, it is vital to be competent to recognize this condition when it is already present. With rare exceptions the teeth will be forced so close together that a finger can be introduced only with difficulty. In most cases, however, it can be introduced and by it the swollen, indurated and inflamed floor of the mouth can be felt. In some cases it can be seen without any effort to expose it, and in most cases by separating the lips and teeth as far as possi-

ble. The tongue will be elevated and may protrude between the teeth. Pain and a feeling of fulness will be experienced by the patient within the mouth. Disturbance of speech and deglutition will be more marked than in a case of simple submaxillary cellulitis. Those symptoms and more particularly dyspnoea all speak for a Ludwig's Angina.

About the only attempt at a differential diagnosis that the writer found in literature was that of Leterrier, although his ideas have been repeated by a few other writers. He said that osteoperiostitis of the inferior maxilla, adenophlegmon and hygroma of Fleischman's bursa, are the only affections with which one can confound a sublingual phlegmon. An osteoperiostitis of the lower jaw is generally a local inflammation and is usually the result of a carious tooth. In most cases which the writer has observed it involved the external portion of the jaw. If it involved the internal surface of the jaw, it would probably still be localized; but if it gave a rapidly spreading cellulitis of the floor of the mouth which is adjacent to it, it would then be, essentially, a Ludwig's Angina, because a sublingual phlegmon, when there would be no occasion for a differential diagnosis. If distinctly localized, there would be none of the characteristic symptoms of Ludwig's Angina, and this condition would not be suggested. But such cases should receive prompt attention, *i.e.,* they should be disinfected and drained at once to prevent a possible extension to the floor of the mouth, which must always be a dangerous condition.

In an adenophlegmon, Leterrier says, one finds small rounded masses, painful and distinctly separated from each other; he further says that a sublingual phlegmon gives a single mass with special characteristics and occupying at least a part of the submaxillary region. That an adenophlegmon can give an extensive single mass in the submaxillary region, the writer believes, is a matter of common knowledge and hardly needs discussion. The cases of Ludwig's Angina, beginning as small localized swellings, to which the writer has already referred, are in all probability, instances of this

kind. An hygroma of Fleischman's bursa, which is situated under the tongue, could hardly be confused with a Ludwig's Angina, since it is not inflammatory, and is distinctly localized and movable on the surrounding tissues.

The only condition, which in the writer's opinion, could be confused with a Ludwig's Angina, is a localized submaxillary cellulitis, or rather an extensive cellulitis in this region which has not invaded the mouth or pharynx. Indeed, it is only this condition which has been confused in the literature with Ludwig's Angina, and the writer has found it necessary to exclude a number of these cases which were reported as Ludwig's Anginas. Gasser, for example, reported four cases, in all of which the streptococcus was found. The writer excluded three, because he could see nothing more than submaxillary cellulitis in them. The extension to the floor of the mouth and pharynx is what determines a Ludwig's Angina in these cases, and working upon this basis, the diagnosis should be comparatively easy. That the term, Ludwig's Angina is still useful in describing this extension, the writer believes.

Prognosis.—The prognosis of a sublingual phlegmon, which is the essential condition in a Ludwig's Angina, in the writer's opinion, will vary according to the virulency of the infection and, therefore according to the rapidity with which the mouth and pharynx are crowded and the larynx reached by the inflammatory process. There is also the lesser danger of septic intoxication which, of course, varies according to the virulency. Pyaemic deposits from invasion of the blood vessels, especally the veins, by the infection have resulted in a few cases, but neither this nor the septic intoxication is, probably, any more to be feared in Ludwig's Angina than in similar infections of other parts of the neck or body. Above all the prognosis will depend upon the promptness with which the condition is recognized and upon the thoroughness of treatment.

In a considerable number of cases, although free incision was made no pus was found and in these death usually re-

sulted. Where pus was found early a cure usually followed, although in some other foci existed and a fatal termination was the result. The writer believes that some of these as well as some of the cases in which no pus was found by incision, are to be explained by the fact that one or more lymphatic glands being involved, the foci of infection were still intraglandular in some or all of them, and the incision missed them. Similar periglandular cellulitis is common in the inguinal region, where it is the rule not only to evacuate the pus, but to shell out all inflamed glands, for we have learned by experience that the inflammation in these cases will frequently continue after a considerable abscess has been thoroughly opened and properly drained. The well known tendency of streptococcus to develop serous rather than purulent exudation in some cases, as in erysipelas, will explain the absence of pus in many. Of the writer's 106 cases, 43 died and 63 recovered. The more recent cases, however, have given much better results than the earlier, as in Ludwig's time, because of earlier recognition and more prompt and thorough treatment. There can be little doubt that a better appreciation of the pathology of this condition will lead to better results in the future.

Treatment.—Antitoxines are not our only hope for the future in those cases, as Semon says. Prompt surgical interference will, probably, always be of first importance. There has been considerable difference of opinion as to what this should consist of. Although spontaneous openings when they have occurred, have almost invariably been found in the mouth, incisions there have not given satisfactory results, and it is generally conceded that the external incision is best. Surgical experience has taught that incisions should first of all lay open freely the focus from which the infection is spreading. The focus in these cases is not the preliminary insignificant lesion, tonsillitis, carious tooth, etc., which, however, should receive suitable attention; but the lymphatic gland in the submaxillary region, or the infected wound, ulcer, phlegmonous inflammation about a tooth, etc., from

which the infection is extending directly to the adjacent connective tissue. If this focus is on the surface within the mouth an effort should be made properly to expose and disinfect it, as with pure carbolic acid. Care must be observed, of course, to prevent the caustic from doing damage to surrounding parts. When this was done in one of the writer's cases, prompt subsidence of the inflammation and a cure followed. If such a focus is in the pharynx, it will be practically impossible to reach it and, as in Semon's cases, treatment will be of little avail. Spontaneous resolution with or without spontaneous opening, or an effective antitoxine are about all we can hope for.

When, however, the cellulitis originates in the submaxillary region, the focus is, probably, in one or more of the lymphatic glands. It is in these cases that the best results have been obtained. This is, undoubtedly, due to the fact that the inflamed area, from the beginning, is better exposed for recognition and treatment. The dangerous invasion of the floor of the mouth and the pharynx is a later development, thus giving more time for arresting its progress before it reaches the larynx or lungs. Delorme reported that in one of his cases the submaxillary salivary gland was exposed without the location of pus, and that the prolongation of the incision towards the median line and through the mylohyoid muscle, located pus. There is room for question whether in such a case the salivary or a swollen lymphatic gland was exposed. The incision of necessity is a deep one in the presence of the massive, indurated exudate, which is always present. Hemorrhage is free and retraction of the edges of the wound difficult. A swollen lymphatic gland may readily be of the same size as the normal salivary gland, and under these circumstances in the absence of an opportunity to scrutinize its structure carefully the one might easily be mistaken for the other. The writer believes that in Delorme's case the pus first developed where the swelling was first observed, in the submaxillary region, and if the submaxillary salivary gland had been freely exposed by the

incision it would have located it. If, however, an outlying lymphatic gland were exposed the pus collection might have been overlooked until its extension through the opening in the floor of the mouth had been reached by the extension of the incision forward and through the mylohyoid muscle.

The best incision is undoubtedly the one which Delorme selected, *i.e.,* over the submaxillary gland and parallel with the lower jaw. The frequency of the spontaneous openings in the floor of the mouth and the fact that a number of writers have reported that they did not find pus until the mylohyoid muscle had been divided, indicate that this muscle should be freely divided, at least unless frank suppuration is found before it is reached. The dangers of an external incision increase as we approach the angle of the jaw, where the largest blood vessels of the neck and head as well as the largest branches going to the face are located. They are all, practically, posterior to this gland. The facial vein lies over it and the artery under it. The gland lies in front of the angle of the jaw, and from the anterior border of the masseter muscle where the pulsation of the facial artery can be felt as it crosses the border of the jaw, forward to the symphysis there are no blood vessels large enough to be feared. The anterior part of the submaxillary incision, therefore, should give little or no trouble, while with a little care it can be safely extended backward far enough to expose the region of the submaxillary gland freely. The mylohyoid muscle may be penetrated with the knife, finger, or a grooved director. The finger should be passed upward in the wound until only mucous membrane intervenes between it and the mouth.

Many writers have employed the median incision from the chin to the hyoid bone. It is safer than the submaxillary incision, since there are no blood vessels in the median raphe, or only small ones. If Ludwig's angina were merely a sublingual inflammation, *i.e.,* beginning in and essentially localized to the sublingual tissues, as Delorme maintained, one would expect that Delorme would select the median incision

to evacuate the pus, as it is the ideal incision for an abscess located under the tongue. Yet he employed the submaxillary incision in all his cases, and this incision has been called by a few French writers following his paper, the Delorme incision. The median incision carried through the mylohyoid muscle may find pus in the floor of the mouth in the typical case of Ludwig's angina, but only after it has extended along the floor of the mouth from the submaxillary region.

The point of greatest importance, however, in connection with this whole subject, is to recognize the dangerous possibilities of every submaxillary cellulitis, and to open the infected area promptly and efficiently. If we have waited too long or our efforts have not been effectual, and asphyxia is threatened, a tracheotomy must be done at once with the hope that the process may soon subside and recovery follow. Intubation is out of the question, at least in most cases, because the mouth can not be opened and the tongue is pushed up. Artificial respiration may be necessary to tide the patient over the crisis. The question of the anaesthetic is an important one. Local anaesthesia has been employed several times and Davis emphasizes its value. When the dyspnoea is marked the added burden of a general anaesthetic is not a trifling one. It would seem that local anaesthesia should then receive the first consideration.

Conclusions.—The condition known as Ludwig's Angina, is a rapidly spreading cellulitis, beginning in the region of the submaxillary salivary gland as a perilymphadenitis, and extending to the floor of the mouth and pharynx. The primary focus usually is some neighboring surface lesion as a carious tooth, tonsillitis or ulcer in the mouth.

The infecting organism is, usually, the streptococcus, alone or mixed with other organisms, as the staphylococcus, pneumococcus, or bacillus of malignant oedema; but it may be the staphylococcus alone or any organism capable of producing a rapidly spreading cellulitis.

Death results from invasion of the larynx in most cases. In a considerable number the lungs are also involved. The

associated septic intoxication is, probably, no more severe than that which results from streptococcus infections of the same grade in other parts of the body.

The opening in the muscular buccopharyngeal wall, through which the submaxillary salivary gland projects into the floor of the mouth, is the path by which the submaxillary infection invades the mouth and pharynx.

Any rapidly spreading cellulitis in the floor of the mouth is a menace to the life of the patient, as the anatomical conditions there, favor the early involvement of the larynx.

It is this invasion of the floor of the mouth and the pharynx which determines the alarming symptoms characteristic of Ludwig's Angina. It is evident, therefore, that a cellulitis of a given grade of severity, beginning in the floor of the mouth, is more dangerous than one beginning in the submaxillary fossa, since the larynx will be more early and surely invaded. The opportunities for recognizing and checking the danger are, therefore, correspondingly lessened. For the same reasons, the most dangerous cases are those in which the phlegmonous process begins in the pharynx or in the larynx, the danger being greatly increased in these because, even if recognized immediately, the parts can not be inspected or properly incised and disinfected.

The pathological changes occurring in the infected area do not differ, materially, from those which may be expected from any severe pyogenic infection, occurring under similar anatomical conditions. The proximity of the alimentary tract explains the frequency of gas and putrescence, in many cases in which gangrene was not reported; while the intensity of the inflammatory process and the compression of the inflammatory material, inside the jaw and under the tongue accounts for the frequency of gangrene.

The condition occurs with sufficient frequency and is sufficiently constant in its clinical course, to deserve a place as a morbid entity. No name is at the same time so brief and so comprehensive as that of Ludwig's Angina. Those cases in which the cellulitis originates in the floor of the mouth, may be included with advantage among the Ludwig's Anginas. Those in which the phlegmonous process begins in the throat, should form a separate group, from the standpoint of prognosis and treatment.

Modern surgical treatment has reduced, considerably, the number of cases in which irregular septic temperature, profuse sweats, delirium and a progressively profound typhoid state, occur.

Incisions in the floor of the mouth may be advisable in a few cases for the relief of excessive swelling, but they have rarely given satisfactory results. The median suprahyoid incision, while the safest of the external incisions, does not expose the usual primary seat of infection and should not be selected, except to evacuate an evident purulent collection in the submental region. The submaxillary incision, *i.e.,* over the submaxillary triangle and parallel with the lower border of the jaw, will, probably, locate an existing pus collection in the greater number of cases. If frank suppuration is not found before, the mylohyoid muscles should be divided and the sublingual tissues exposed.

On account of the added irritation of a general anaesthetic to an already dangerously inflamed larynx, local anaesthesia will in all probability prove to be the more valuable means of controlling the pain during the making of the incision.

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DR. G. G. DAVIS said that this was an intricate subject and one with many points needing elucidation. The pathology is intimately associated with the treatment. The disease is quite a fatal one, the mortality is still quite large. There seems to be no absolute accepted line of treatment: Dr. Thomas' paper points out a line of treatment. If the disease kills by interfering with the breathing, then the line of treatment should be to obviate as much as possible the edema of the glottis and the encroachment upon the air passages. If, however, infection is the lethal agent, then the treatment should be directed to that cause. Dr. Thomas spoke of 92 out of 106 cases beginning external to the mouth and this brings up the cause of the infection beginning external to the mouth, probably in the submaxillary or retromaxillary region. It is very hard to see what should cause a primary infection of that region. Dr. Davis personally believes that the infection begins most often in the mouth and travels to the other tissues. He called attention to the statement made by Dr. Thomas that one author stated that the infection travelled to the lymphatic glands in the submaxillary region, being conveyed by the lymphatics from the primary focus in the mouth. Dr. Davis does not believe it is a question of the lymphatic nodes. Inflammation of the submaxillary lymphatic nodes and of the retromaxillary lymphatics along the large vessels can as a rule be outlined by the sense of touch. The involvement of lymphatic nodes is usually more or less limited. This disease to Dr. Davis' mind pursues an entirely different course. Instead of producing discrete lymphatic enlargement we practically never see discrete inflammatory enlargement of the lymphatics. There is a widespread, board like, inflammation in which all evidence of lymphatic nodes is obscured and there is no outline of any nodes. He believes the disease propagates itself by direct continuity of the cellular tissue.

It is hard to point out an absolute cause in all cases. In several cases which Dr. Davis has seen he believes the cause of the infection to have been in the teeth. He called attention to the specimen presented by Dr. Thomas showing the connection between the mouth and the throat. It is obvious that if a person has an ulceration of the root of the teeth, and particularly if there is pus around a decayed tooth, it involves the submaxillary gland because this gland lies quite close to it, and if it simply

follows the submaxillary gland down it goes right out of the mouth into the neck. It is extremely difficult to state definitely that the trouble originated submaxillarily and not intra-buccal.

As regards the character of the inflammation Dr. Davis believes it is generally admitted from a bacteriologic standpoint that several kinds of bacteria give rise to this disease; in other words, not only has the streptococcus been found in a large number of cases, but in several of the cases the disease has been found to contain, so to speak, only microorganisms which are of a single type, not streptococcal: for instance, pure pneumococcus cultures, and the staphylococcus, besides other bacteria have been found.

There is a question as to what extent is there sepsis and to what extent is there suffocation as relative lethal agents in this disease. There have been cases in which there was absolutely no indication of the slightest obstruction with respiration in which death ensued, which could only have been caused by infection.

Dr. Davis does not accept the temperature as a guide for septic infection. He stated that in some of the worst cases of diphtheria the temperature is low, while in other parts of the body, the appendix for instance, the infection can be very marked and the temperature can be low. One of the first things that strikes the physician in many of these cases of Ludwig's Angina is the depression of the patient. Some patients have the great swelling with no depression whatever, while others have a terrible amount of depression. Sometimes the pus is both free and offensive. Dr. Davis has seen two or three cases where the swelling has broken alongside the alveolus close to the bone. With regard to the making of incisions his favorite one is directly in the median line, as through this incision the finger can be put right through into the mouth, and the serum also drains freely into it.

He believes the disease is a local one, and that it often kills by infection, although a certain proportion of the cases are accompanied by respiratory symptoms. In these cases the larynx is gradually choked off, and then the patient goes around until something causes complete obstruction, when naturally he dies. There are other cases which pass through a typical pyemic condition with chills, fevers, sweats, temperature 104° to 105°, who

die absolutely of sepsis without any respiratory difficulty whatever.

Dr. Davis believes the line of treatment to be pursued is that which would direct against any local septic trouble; he considers free incisions perfectly justifiable in bad cases, in fact one reaching almost from below the ear posteriorly to near the symphysis anteriorly.

Dr. W. Joseph Hearn called attention to the difficulty of etherizing patients suffering from Ludwig's Angina. In three cases which he had the opportunity of seeing there was great difficulty in this direction. In every case the patient was nearly suffocated. He was present at one operation where the surgeon had hardly got the patient half under ether when he was obliged to do a tracheotomy to keep the patient from suffocation. In one case of his own he attempted to give ether, and the man became cyanosed. Dr. Hearn therefore discarded the anesthetic and made free incisions as in ordinary cellulitis: this patient recovered. Dr. Hearn presumes from the difficulty in administering ether that the pharynx and larynx must be involved.

Dr. Charles F. Nassau stated that his experience with this condition was limited to two cases, although he also had the opportunity of observing a third that was under Dr. DaCosta's care at the St. Joseph's Hospital: this patient died.

It is Dr. Nassau's belief that the patients who get well are those in whom suppuration has been established. In one of his cases the condition followed during convalescence from scarlet fever; cover slips were made and there was found to be a streptococcus infection. In both his cases the operation was done on account of the extremely rapid spread of the infection outwards and over the chest; in both the infections probably occurred through the tonsil as both patients complained of a tonsillitis a few days previous. In one of his cases this tonsillitis cleared up to some extent and then this infection began, slightly at first, occupying at least three or four days in its development; the patient did not have much fever nor pain, but when seen by Dr. Nassau she was in a good deal of pain; she took ether very well. The other patient, not only on account of her extremely ill condition but particularly on account of the place where she was, was operated upon under cocaine anesthesia. This merely saved her the pain of the skin incision. In neither of his cases did he

find any pus; the nearest approach to it was in the second case, where behind the sheath of the common carotid a few flakes of lymph were found, possibly the beginnings of suppuration.

Dr. Nassau believes in the very widest and largest possible opening, even by the tearing up of tissues if this is found necessary. He believes that where the infection simply travels without suppuration the patient has a splendid opportunity of being carried off by the infection. He argues that sometimes one organism or one infection can be replaced by another; for instance, in an infection of the Fallopian tube which was probably of gonorrhœal origin, there may be an acute flare-up, and at operation no gonorrheic organism found, it having been replaced by the streptococcus or some other organism of suppuration. In the same way there may be a peritonitis from, say, the colon bacillus, and at autopsy one may find only streptococcus as the fatal cause. Therefore one organism will kill another. This is the basis of what treatment Dr. Nassau has given other than incision. His idea was to bring about suppuration as quickly as possible and to get the wound infected with something else. He does not consider it good treatment to keep these wounds too clean, but that a chance should be given for suppuration.

Dr. W. M. L. Coplin (by invitation) stated that he considered this subject of special interest to pathologists. For twelve years he has been directly interested in it. To call the condition cellulitis may be the truth but it is not the whole truth; it is really a myositis. It is peculiar in its distribution along the course of the muscles, and the change that takes place in the muscle fibres. If one will carefully examine these muscle fibres one will find that within the perimysium there is an extending exudate with the usual progressive myolysis occurring in various types of muscle inflammation, and an accumulation of numerous leucocytes within the muscle. He thinks one of the conspicuous features in cases of Ludwig's angina is the immunity of the lymph system. He has one specimen, a complete evisceration of the cervical region, in which the lymph nodes were examined microscopically, and showed practically no infiltration, one knows of course that where an inflammatory condition involves the primitive lymphatics there is almost invariably a leucocytic invasion of the lymph nodes. In two of the cases in which Dr. Coplin made complete sections of the neck he secured the glands and was

amazed by the escape of the glands from this process. With regard to the submaxillary and the sublingual salivary glands, he has a specimen from a case which has been reported, in which these glands are bare and section shows that they practically escaped infiltration. The condition in one case in which it was impossible to make a complete dissection of the neck certainly began as a paramygdalitis. Sir Felix Semon refers to one or two cases beginning with what we would now call paramygdalitis. In this case the tonsil was almost completely dissected out by the extending necrosis but on section the organ is but slightly involved, again illustrating the fact that the lymphatics may escape. With regard to the type of infection it is Dr. Coplin's opinion that it is etiologically a polymicrobic process. It is not a disease that should be given a distinct pathological position; because of its symptomatology, largely determined by the peculiar anatomy of the neck, it might be regarded as a clinical entity.

To return to the phenomenon observed in the muscle. The myochrome disappears early, giving the muscles a washed meat appearance. Dr. Coplin has seen muscles of the body of the tongue almost the color of the white meat of chicken. The muscle change resembles, possibly superficially, that peculiar disease known as the infectious myositis of Japan. The washed meat appearance is a very striking manifestation of infection travelling widespread through the muscle without focal necrosis. If one recalls the capillary injections of muscles in which a muscle fibre is seen festooned by the most elaborate capillary circulation, like vines around a column, one can understand that an infection gaining sufficient headway to sweep like fire through that kind of a circulatory field, yields its toxin directly to the circulating blood, hence must cause great depression; even with a limited area of infection the systemic phenomena would be largely dependent upon the toxicogenesis of the invading organism.

Dr. Coplin would look at the suggestion made by Dr. Nassau that where suppuration occurs the patients would be better, from just the other side. It seems to him that the explanation of these cases is that the attack made by the antibodies is such as to secure a focusing of the infection and establish a necrosis in that area of limitation; that where the individual is unable to resist the infection it travels with such rapidity that we do not see a marked

accumulation of leucocytes. That in these cases where suppuration does not occur there is just as much disintegration and destruction of the myochrome as in cases where suppuration does occur, but there is an immeasurably less abundance of leucocytes, and a less accumulation of antibodies.

Dr. Coplin was greatly interested in the effect of the disease upon the organs of respiration. In one case which he had the opportunity to examine in very great detail, there was a clearly defined streptococcal bronchitis, while between the intralobular spaces one could see the lines of an interstitial pulmonary lymphangitis. Delicate yellowish lines traced over the incised surface of the organ and extended toward the pulmonary lymph nodes, and in this very case there was, in the peribronchial lymph nodes, no cellular infiltration.

In some of these cases there is a respiratory difficulty behind the respiratory obstruction of the larynx, just as we occasionally see in puerperal sepsis, in erysipelas, and in that peculiar disease, Brinton's disease, the absorption of toxic material and the induction of advanced suppurative interstitial pneumonia. Dr. Coplin believes this is in some cases mistaken for capillary bronchitis, which presents a very similar clinical picture.

With regard to the atrium of the infecting organism Dr. Coplin does not consider this of much importance, and believes that it has little material influence on the pathology of the lesion.

COINCIDENT ABDOMINAL LESIONS.

CASES: (I) *Appendicitis with ruptured extra-uterine pregnancy.* (II) *Appendicitis, pregnancy and ureteral calculus.* (III) *Dermoid cyst of ovary, pregnancy and gallstones.* (IV) *Tuberculosis of ovary and appendix with floating kidney.*

DR. GEO. ERETY SHOEMAKER said that the subject of combined operations or of operations for different lesions present at the same time, was one of interest and importance, frequently calling for the exercise of judgment. A number of years ago he read a paper before the Academy of Surgery advocating the removal of the appendix, if not normal, in all suitable cases when the abdomen was opened for other purposes. The proposition was received with little respect at that time, but in the evolution of surgical opinion has since become the practice of many good abdominal operators. When operating for other abdominal conditions, examination of the appendix, in all patients not in immediate danger from shock or exhaustion, and where the fear of spreading septic material from another focus does not deter, will result in demonstrating in at least 25 per cent. of cases evidences of sub-acute or chronic disorder of the appendix. In his last 400 abdominal operations not undertaken for appendicitis alone, the appendix was removed in 88, or 22 per cent. Some of these disorders involve the organ only from without and can do harm chiefly by interference with drainage, through angulation from contraction of the meso-appendix or of surrounding adhesions. Other cases show evidences of intrinsic disease of the appendix in various stages of development. This is particularly true of chronic pelvic inflammation with definite lesions of other viscera, especially tubercular.

It may be difficult before operation to separate the appendiceal from the other inflammatory conditions present. Interesting papers have been presented on the topic of referred pain leading to obscurity in diagnosis between appendicitis and kidney or gall bladder disease chiefly. His object here was to draw renewed attention to the fact that even when one definite and important lesion is demonstrated and removed at operation the surgeon should not stop, particularly in chronic cases, until he determines that other organs are not involved. Dr. Mayo has recently spoken of the systematic examination of the gall bladder from the lower incision. This of course can only be done when the incision is large enough to admit the hand and wrist, and should be omitted when dealing with pelvic infections. It does not by any means follow that the second lesion should be operated upon at the same sitting. Indeed, it might be a serious error, to attempt to deal with a badly adherent and inflamed gall bladder, the same day that an acute appendicitis required operation, or vice versa. A bad hysterectomy may tax the patient's resources, and the removal of an adherent appendix might bring the colon bacillus risk into an otherwise clean field. Quiescent inflammatory conditions of moderate severity in strong patients may, however, be attacked at the same sitting, especially if in the same general locality. A movable kidney which is bad enough to cause trouble may be anchored at the same time that a chronic appendicitis is cured by appendectomy.

In gynecological work it is constantly found that the same

patient presents several conditions each of which causes trouble. Hemorrhage requiring the curette; laceration of cervix and perineum requiring repair; bleeding and prolapsed hemorrhoids requiring operation; chronic salpingitis and appendicitis requiring conservative operation. These may all be dealt with at the same sitting only if the inflammatory processes are quiescent. If they are active the operations must be done in two groups, and the more serious should be done first. He had a patient now convalescing in whom all of these conditions were operated upon at the same time.

The patient must not be kept too long under ether and after the abdomen is opened, no work on another part should be done. Minor procedures, such as repair of lacerations, should be carried out first, as these cause no definite strain, and the patient's danger begins only when the abdomen is opened. Of course gloves and instruments are changed when the field is changed to the abdomen. He reported the following instances of combined lesions of important type:

I. *Extra-uterine pregnancy associated with appendicitis.* C., 41 years. Not previously pregnant for 14 years. Menses irregular and apt to be profuse for nine months. No periods missed, but the last one, which began six weeks before examination, had been a week late, and bleeding had continued ever since. The rupture of the left pregnant tube had occurred two weeks before with sharp pain followed by fainting and perspiration. The ovum was still in the tube in a tiny unruptured sac of fluid. Pregnancy was probably not over six weeks old. There had been much rectal bleeding for several months, temperature had never been found by her physician to be over 100 when taken. Symptoms had been so mixed including bleeding from bowel and vagina, severe pain in left abdomen chiefly and abdominal soreness and chronic indigestion, that attention had never been definitely fixed by her physician upon the appendix region and an attack of moderate severity had doubtless passed over before the ruptured extra-uterine pregnancy occurred.

When referred to him in his office, the diagnosis of ruptured extra-uterine pregnancy was made and operation advised and performed the same day. The left tube was ruptured near the attachment of the broad ligament, many ounces of free blood and clot found in the peritoneal cavity. Tube removed leaving corresponding ovary. Examination of the appendix showed a hard meso half an inch thick, the appendix walls dusky red, hard, thick and rigid, the mucous coat purple, no pus; removal. Diagnosis: Decided sub-acute appendicitis without perforation. Ruptured left pregnant fallopian tube and intra-abdominal hemorrhage. It is interesting to note that in the four months which have elapsed since the operation the troublesome chronic indigestion present for years has disappeared.

II. *Coincident acute appendicitis: pregnancy and ureteral calculus with nephritis.* E. G. A patient is now in the Presbyterian Hospital where three prominent conditions had to be considered. *First,* pregnancy at four and a half months, with a very high right uterine cornu. *Second,* severe pain with tenderness behind and about right kidney, much blood in the urine, abundant dark granular and other casts, the pain passing down the course of the right ureter to right vulva. *Third,* an acute right sided abdominal inflammation with temperature to 103°, chills and a septic look. Leucocytosis 25,000.

This case was cleared up: first by the passing on the day of admission of a sharp pointed crystal with the urine with relief of kidney pain; second, by laparotomy and removal of appendix, the abdomen containing about two ounces of free turbid fluid, no adhesions, peritoneum deeply congested in right abdomen; third, by the use of large quantities of water by mouth and salt solution by rectum to overcome the nephritis. The pregnancy was undisturbed, the child lives. The gauze drainage has now been removed and the wound is healed. The general condition good except for nephritis.

III. *Coincident dermoid cyst of ovary, pregnancy and gall-stones.* J. C., 35 years old, 6 children. Applies (*a*) because of severe pain in gall bladder region for one month, through to shoulder. Constant distress also in epigastrium. No vomiting, no jaundice, no putty-colored stools. Only similar attack followed a confinement two years before. Examination shows (*a*) a tender, small gall bladder. (*b*) A rounded tumor four inches long, adherent in pelvis with much soreness and pain about it. (*c*) Pregnant two months. Perineum and cervix much lacerated.

As an adherent tumor overlying a pregnant uterus was a greater present menace than the sub-acutely inflamed gall bladder,

the abdominal incision was made low down and a dermoid cyst of the left ovary four inches by three by two and a half firmly adherent was removed without rupture. It contained bone an inch long and cholesterin. The appendix was quiescent but showed old inflammatory changes. It was removed through the same incision. The gall blader was examined through the lower incision and found to be tightly contracted around two large gall stones into an hour glass shape. There was no fluid. Operation on the gall bladder was postponed until after delivery, in the absence of dangerous symptoms. Recovery followed from the dermoid operation and appendectomy, the woman was delivered at term seven months later. She was seen a few days ago, and as she still complains of the gall bladder soreness she is to have an operation as soon as her child is old enough to wean.

IV. *Tuberculosis of ovary and appendix. Movable kidney.* M. E. Single, 27 years. Attack called appendicitis four years before and a second two months before; ever since which walking and jarring hurt the right lower quadrant and up behind the kidney. Loss of weight 13 pounds, now 105. For two months an inflammatory swelling on 7th rib in front. Pain in right upper abdomen at times severe and apparently due to a very movable kidney which varies in size, now presenting a fusiform swelling which is movable and can be displaced upward as far as the umbilicus. The appendix is tender, the tubes and ovaries are fixed. The patient was bright, cheerful and intelligent; keenly desired relief. Urine normal.

To overcome the pain crises in the right kidney region, as the fusiform swelling was probably an early hydronephrosis, the kidney was anchored. The appendix was exposed through a gridiron incision. The peritoneum nearby was sparsely studded with small tubercles; no fluid, no adhesions. In the meso appendix a cheesy nodule size of grain of corn. Appendix sub-acute catarrhal inflammation, removed with cheesy meso: stump buried. Through the gridiron incision the tubes were felt to be diseased. It was therefore closed and a small median incision made, through which by catgut ligation, the right tube was resected and the left removed at the cornu. One-third of the left ovary was removed. The tubes formed closed sacs imbedded in adhesions. No drainage.

Convalescence extremely smooth. Wounds healed primarily.

Several days later under local anesthesia a fusiform yellowish flocculent mass of material looking like coagulated lymph was removed from the periosteum of the 7th rib, leaving a smooth glistening cavity which promptly healed with packing. Pathological report of Dr. Steele: Tuberculosis of ovary, giant cells and typical areas of infiltration. Cells of larger type found in tubercles. No giant cells or caseation found in tubes.

These operations were done two years ago. Patient seen recently. Scars sound. No abdominal symptoms. Menstruation regular and painless. Walks well and works without distress. Kidney in place, no trouble since. No disease or tenderness in tubal or ovarian regions discoverable on examination of pelvis. Lungs negative. Weight same as before operation, 105. Considers that operations were of enormous benefit to her and claims to be gaining in general health, though still slender and rather pale.