ON THE PROBABLE PATHOLOGICAL IDENTITY
OF THE VARIOUS FORMS OF
ACUTE SEPTIC INFLAMMATIONS OF
THE THROAT AND NECK,
HITHERTO DESCRIBED AS
ACUTE ŒDEMA OF THE LARYNX, ŒDEMATOUS LARYNGITIS,
ERYSIPELAS OF THE PHARYNX AND LARYNX, PHLEGMON
OF THE PHARYNX AND LARYNX, AND ANGINA
LUDOVICI.

BY
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It is with considerable hesitation that I venture to submit the following paper to the judgment of the Royal Medical and Chirurgical Society. Its thesis—namely, that the various forms of acute septic inflammations of the throat and neck hitherto described under the names of acute œdema of the larynx, œdematous laryngitis, phlegmon of the pharynx and larynx, erysipelas of the pharynx and larynx, and angina Ludovici, are in all probability pathologically identical, and merely represent different degrees of severity of one and the same septic process due to invasion of the throat and neck by various pathogenic micro-organisms—is far-reaching, and nobody

could be more fully conscious than I am, that it can at the present moment only be finally proved by a harmonious combination of clinical, pathological, and bacteriological evidence.

Now I have at the very outset of my remarks to confess that my material, although, I think, singularly complete from the clinical point of view, is somewhat deficient from the aspect of pathological anatomy, and, furthermore, that my bacteriological evidence, so far as my *personal* experience goes, unfortunately is conspicuous by its absence. Under these circumstances, the question why I had not waited, before venturing to submit my ideas to this Society, until I had completed my proofs from every point of view, is so obvious that I feel anxious to answer it somewhat fully before entering upon my subject proper, the more so as I thereby may fairly hope to disarm to some extent hostile criticism.

In the first place, then, let me say that the affections of which the following paper treats, particularly their more serious forms, are, fortunately, so rare that even with exceptional opportunities no single observer is likely to ever command a really large material, and that often even in an extensive specialistic practice years pass without a single case of that nature coming under observation. As a matter of fact I have in the whole course of nearly twenty years' specialistic hospital and private practice, altogether only observed fourteen cases of acute infective inflammations of the pharynx and larynx. Yet this material, small as it may seem to anybody who has not studied the literature of the subject, is, I believe, an exceptionally large one so far as the experience of a single observer is concerned.

Further, I may fairly claim that I have taken to heart Horace's words, "*Nonum prematur in annum*," and have not rushed into print with undue haste. The very first severe case of this sort which I ever observed (this was in 1882, *i. e.* fully thirteen years ago) at once raised the question in my mind whether the present classification of the various forms of acute septic inflammations of the organs of

the throat and neck was scientifically tenable. In every subsequent case which came under my notice I have tried hard to get a bacteriological investigation, but in one single case only, the last one that came under my observation, this opportunity was given to me. I had to wait eleven years for it, and then the evidence obtained from it was unfortunately purely negative. Two years have passed since without a new case of the kind having come under my observation. Under such circumstances it is obvious that it is no good waiting any longer. Possibly again many years may pass before I have the chance of observing another case of the kind, and even then, supposing that I were fortunate enough to get the opportunity of a bacteriological examination, its evidence might again be negative; whilst numbers of such cases may meanwhile pass through the hands of other observers, to whom it might not occur to study the interesting question to which I wish to draw attention unless some incitement were given thereto.

But, apart from these more negative arguments, I have some positive reasons to adduce which seem to justify my present undertaking. My clinical material, although small in numbers, has, as mentioned before, by good fortune been so singularly complete, that from the clinical point of view alone I hope I shall be able to prove that my ideas on this whole subject are more than mere idle speculations.

Again, whilst my own bacteriological investigations have failed, another observer, Dr. Jordan, of Heidelberg, has been more fortunate, and I shall be able to show, in the conclusion of this paper, from quotations of his results and statements, that my ideas on the subject in reality represent an application of the present general views of bacteriological authorities to the special forms of septic inflammations of the throat and neck.

And, finally, I may be permitted to say, that whilst my own inclinations entirely go towards establishing, if possible, new views on the basis of well-ascertained and

incontrovertible *facts*, yet the value of what Professor Huxley has well called a "working hypothesis" ought not to be underrated. I venture to hope that the ideas on the probable identity of the various acute infectious inflammations of the throat and neck, which I shall have the honour to submit to the Society, may be thoroughly tested by subsequent observers, and that their correctness may be even more fully established than I am able to do with the material now at my disposal.

Having thus, I hope, justified my undertaking, I will at once go *in medias res*, and begin by showing from a few recent utterances of competent observers, taken almost at random, the confusion which at present exists in the nomenclature of the various forms of acute septic inflammation of the throat and neck.

Von Ziemssen¹ adopts Bouillaud's terminology in calling "laryngitis phlegmonosa" all those laryngeal inflammations which mainly concern the submucous connective tissue, without, however, being exclusively limited to this. The sub-headings of his chapter are "Œdema Laryngis. Abscessus Laryngis," and he adds that, in order to "simplify" his description, he had admitted into it those forms as well of laryngeal œdema which were of non-inflammatory origin. Hence we find in the paragraph dealing with the etiology such heterogeneous causes as inflammation of the mucous membrane, diphtheria, chemical and thermic irritation, burns and scalds, foreign bodies, mechanical insults, perichondritis, acute infectious diseases (pyæmia, septicæmia, ulcerative endocarditis, typhus, smallpox, scarlatina, measles, erysipelas), again acute and chronic nephritis, malarial cachexia, amyloid degeneration of the kidneys, cardiac affections, emphysema and cirrhosis of the lungs, compression of the vessels of the neck by tumours of the thyroid gland, enlargement of the lymphatic and salivary glands, tumours of the neck, aneurism of the aorta, &c.

¹ 'Cyclopædia of Special Pathology and Therapy,' 1876, vol. iv, 1, pp. 311 *et seq.*

Sir Morell Mackenzie¹ adopts a similar arrangement. He, however, gives his chapter in question the title of "Œdematous Laryngitis" with the synonyms "Laryngitis phlegmonosa. Laryngitis submucosa purulenta," and completely separates from it the erysipelatous form, which is described in a separate chapter as "Erysipelas of the Pharynx and Larynx." The division into inflammatory and non-inflammatory forms of laryngeal œdema is even less distinctly drawn in Sir Morell's work than in von Ziemssen's chapter, but he deserves great credit for having first of all expressed his belief² that in nearly all the instances of so-called "simple inflammation" the disease is due to blood-poisoning.

A very different division is adopted by J. Solis-Cohen.³ This author devotes a separate chapter to each—"Phlegmonous Laryngitis," "Erysipelatous Laryngitis," and "Œdema of the Larynx," and expressly states that phlegmonous laryngitis, though often attended by phenomena of œdema, is to be discriminated on the one hand from serous œdema of the larynx, and on the other from erysipelatous laryngitis, with which it was not unfrequently confounded.

In strict opposition, again, to this last statement, we find Massei,⁴ in a very interesting monograph on "Primary Erysipelas of the Larynx," which has exercised considerable influence upon the present views in this whole question, expressing his opinion as follows (l. c., p. 29):—"My own belief I may summarise to the effect that erysipelas of the larynx frequently is nothing else than the process called "laryngitis phlegmonosa," that this disease may occur in a sporadic as well as in an epidemic form, and that it represents a primary, independent, therapeutically not invincible process." In view of this unequivocal statement

¹ 'Diseases of the Throat and Nose,' 1880, vol. i, pp. 272 and 195.

² Ibid., p. 274.

³ 'Diseases of the Throat and Nasal Passages,' 1879, pp. 433 *et seq.*

⁴ "Ueber das primäre Erysipel des Kehlkopfs," 1886; 'Deutsche Uebersetzung von Vincenz Meyer,' p. 29.

it is rather bewildering to find in the same author's text-book,¹ not only subdivisions made into "Œdema," "Epiglottitis," "Laryngitis phlegmonosa," and erysipelatous forms, but the latter again subdivided into "Laryngitis erysipelatosa" and "Primary Erysipelas of the Larynx."

Matters, however, became even more complicated when, in 1888, Senator² described before the Medical Society of Berlin what in his introductory remarks he called a "little known," and in the conclusion of his paper boldly a "new"³ disease. To this he gave the name of "Acute infectious phlegmon of the pharynx," and characterised it as "a diffuse purulent inflammation in the deeper tissues of the pharyngeal mucous membrane, which thence is propagated to the larynx and the glands, and secondarily invades also other organs. The disease has attacked (viz. in the author's experience) persons previously in full health, and without any etiological factor being demonstrable."

The reading of Senator's paper was followed by an animated and very instructive discussion,⁴ in the course of which the late Paul Guttman strongly contested the view that the disease in question was a new one, and maintained that the cases which Senator had brought forward in support of his statements could be well interpreted as belonging to the class of erysipelatous inflammations of the

¹ "Pathologie und Therapie des Rachens, der Nasenhöhlen und des Kehlkopfs," 'Deutsche Uebersetzung von Emanuel Fink,' 1892, vol. ii, pp. 200—211, 237, 241.

² 'Berliner klinische Wochenschrift,' 1888, No. 5, 30th January.

³ British observers will of course be reminded by Professor Senator's description, of the late Dr. Carrington's and Dr. Hale White's cases of "Phlegmonous pharyngitis" ('Trans. of the Clinical Society of London,' vol. xviii, 1885, p. 164), which are simply prototypes of the affection described by Professor Senator. In the discussion which followed their communication in the Clinical Society ('Brit. Med. Journ.,' 1885, vol. i, p. 382) I for the first time stated the views which have gradually ripened into the present paper, and I have since twice referred to the subject, once in 1890 in the discussion which took place on the question in the Section of Laryngology of the Tenth International Congress at Berlin, and once in the 'Internat. Centralblatt für Laryngologie,' 1893, vol. x, p. 55.

⁴ 'Berliner klinische Wochenschrift,' 1888, No. 6, p. 112, *et seq.*

upper air-passages. Virchow declared that on the whole he agreed with Guttman's views, and that he, too, could not exactly define the mutual relationship of erysipelatous and diffuse phlegmonous affections. Sonnenburg stated that such phlegmonous affections as those described by Senator had long been known to surgeons. Senator himself, however, stoutly maintained that the disease described by him had nothing whatever to do with erysipelas, and was also essentially different from the forms of phlegmon mentioned by Sonnenburg.

Just as Massei's monograph had been followed by a host of publications on erysipelas of the larynx, thus now Senator's communication became the starting-point of quite a literature on acute infectious phlegmon of the pharynx, and thus the acute cedematous and phlegmonous inflammation of the upper air-passages, which only twelve years earlier had been described as more or less belonging together by v. Ziemssen, were now split up into (1) idiopathic or essential inflammatory cedema of the larynx ; (2) erysipelas of the larynx ; (3) acute infectious phlegmon of the pharynx and larynx ; (4) phlegmonous pharyngitis from other causes—not to speak of such forms as Strübing's angio-neurotic cedema, and the inflammatory cedema occasionally caused by some drugs, such as iodine preparations, which had also meanwhile been described. Most text-books of laryngology published since then, and the discussion on acute infectious inflammation of the pharynx and larynx which took place in the Section of Laryngology of the Tenth International Medical Congress¹ (Berlin, 1890), vividly reflect the uncertainty and confusion thus produced ; it is, however, unnecessary to quote further opinions, first, because I think that the illustrations so far given suffice to show the present unsatisfactory state of the question, and, secondly, because quite recently a very full historical and critical retrospect of the whole matter has been given by Dr. A. Kuttner of

¹ 'Verhandlungen des Xten Internationalen Medicinischen Congresses, Berlin,' 1890, vol. iv, Abtheilung xii, p. 173, *et seq.*

Berlin in an excellent monograph, 'Laryngeal Œdema and Submucous Laryngitis,'"¹ which has also been published in somewhat abbreviated form in Virchow's 'Archiv.'² Dr. Kuttner's views on the whole question are, I am glad to say, so very similar to my own that I could only repeat his description in other words, and I therefore venture to refer those specially interested in the subject to his paper.

My own attention was prominently drawn to the question by a case which I observed in 1882, and which will be referred to further on as Case 14. Although at the time I described it under the title "A Case of Erysipelas of the Larynx, Pyæmia, Death,"³ I was fully aware, as shown in my concluding remarks, that it was impossible to decide whether the disease started in the cellular tissue of the neck, in the tonsils, or primarily in the larynx. Nor did I conceal the fact from myself that the case might have been regarded, especially if not seen at its very onset, very justifiably by other observers as an instance of Ludwig's angina, the dyspnoea being considered as a mere result of the infiltration of the cellular tissues of the neck, and the laryngeal œdema, if ascertained at all by means of the laryngoscope, being looked upon as simply due to stasis. Finally, I also clearly saw that if a pathological anatomist had performed the post-mortem examination without any knowledge of the clinical course of the illness, his diagnosis might easily have been that of deep-seated phlegmon of the neck with metastasis in several joints.

Anyhow I learned from this case how difficult it sometimes was, in spite of the definite classification of acute septic inflammations of the throat and neck then existing, to accurately define in a given case, such as the one described by me, to which exactly of the admitted forms

¹ 'Larynxœdem und submucöse Laryngitis,' Berlin, Georg Reimer, 1895.

² 'Archiv für pathologische Anatomie, &c.,' vol. cxxxix, Heft 1, 1895, p. 117, *et seq.*

³ 'St. Thomas's Hospital Reports,' vol. xii, 1882, p. 114.

it belonged in reality, and my attention having thus been rivetted at an early period of my observations of such cases on this question, I have ever since tried to judge the cases which have subsequently come under my observation from an absolutely unprejudiced point of view. The result of this has been an ever-growing conviction *that the various forms of acute septic inflammation of the throat and neck, hitherto considered as so many essentially different diseases, are in reality pathologically identical, that they merely represent degrees varying in virulence of one and the same process, that the question of their primary localisation and subsequent development depends in all probability upon accidental breaches of the protecting surface through which the pathogenic micro-organism which causes the subsequent events finds an entrance, and that it is absolutely impossible to draw at any point a definite line of demarcation between the purely local and the more complicated, or between the œdematous and the suppurative forms.*

Whether this conviction—from the clinical point of view at any rate—is justified or not must be decided from the following short reports of the cases which have come under my observation from 1882 to 1893. If they were arranged in chronological order I feel sure that it would be difficult, if not impossible, to understand how I had arrived at my belief. But if given, as they will be, *in order of ascending severity*, the justifiability of my views will become apparent, I think, to every unbiassed reader. I have only to add that the fact that in most of these cases I saw the patients on one or two occasions only as a consultant must be the excuse for regrettable gaps concerning important points on which some further light is urgently desirable. The few instances in which I had the opportunity of following the cases up more thoroughly are given in full.

CASE 1. *Acute œdematous inflammation of the palate and pharynx; recovery.*—Baron B—, Secretary of the Russian Embassy in Paris, came to me on August 27th, 1882,

complaining of repeated shivering fits, general malaise, great dysphagia, and violent pain in the throat. He had been for a few days under the treatment of Dr. Forsbrook, who had diagnosed œdematous inflammation of the throat and had given him aconite and saline aperients. At the beginning of his illness he was very feverish.

When I saw him he was already better, though there was still very considerable œdematous inflammation of the palate and pharynx, which extended down towards the commencement of the arytaeno-epiglottidean folds, but curiously enough had not affected the latter; the larynx in fact was perfectly normal. No enlargement of the cervical lymphatic glands, no fever, pulse 80, weak. I gave him quinine, and ice internally and externally. The œdematous inflammation disappeared in a few days.

CASE 2. *Acute isolated œdematous inflammation of the epiglottis; recovery.*—I had attended Mr. O. S—, æt. 43, in March, 1882, for slight bronchial catarrh, and had ascertained that his pharynx and larynx were perfectly normal, when two days after his last visit he returned, on March 31st, on account of violent dysphagia, which, without any known cause, had developed within the last two days. The onset of the illness had been attended by slight shivering followed by rise of temperature, and the patient felt very low and ill. On examination *enormous* isolated œdema of the epiglottis was seen. This part was at least four times as large as it had been before, brilliantly red, smooth, and shiny; the rest of the pharynx and larynx were normal, and there was no enlargement of the cervical glands, no dyspnoea, voice normal. Pulse 106, small, easily compressible. Temperature 100·6°. No cause whatever could be elicited for the attack. I ordered ice internally and externally, quinine, absolute rest, and cold and fluid diet. On the next day there was a distinct diminution of the œdema with relief of the dysphagia, and two days afterwards matters had almost returned to the normal. I have not seen the patient since.

CASE 3. *Acute œdematous inflammation of the posterior wall of the larynx, with slight infiltration of the left side of the neck ; recovery.*—Mrs. McS—, æt. 52, was sent to me on June 21st, 1884, by Mr. Johnson Smith, of Greenwich. She had fallen ill about five to six days previously with shivering, febrility, sore throat, and some external swelling of the neck below the left angle of the lower jaw, which shortly afterwards was followed by dysphagia. There had been no difficulty in respiration and only slight hoarseness. She had already commenced to improve before she consulted me. When I saw her, her temperature was normal, pulse 82, very weak, and there was some slight infiltration of the mucous membrane over the aryttænoid cartilages, in the interaryttænoid fold, and on the œsophageal aspect of the cricoid plate. The neck was not tender on pressure, and no tumefaction could be felt on the left side. Under the use of ice externally and internally, and bromide of potassium, recovery took place within a few days.

CASE 4. *Acute œdematous tonsillitis, pharyngitis, and epiglottitis ; recovery.*—On March, 3rd, 1888, I was asked by my friend and colleague, Dr. Bristowe, to see with him a relative of his, Mr. S. B—, æt. 26, who had fallen ill five days previously with shivering followed by rise of temperature and sore throat, particularly on the right side. He had been exposed, he spontaneously stated, to sewer gas exhalations. In the first few days of his illness the temperature had been on the average 103°. Then improvement had taken place. Since the 10th, however, the pain in the throat had become aggravated, and he had been almost completely unable to take any food. In the night previous to the consultation difficulty of breathing had set in, followed by great weakness of the voice and very troublesome muco-purulent secretion from the throat, which could only be discharged with much difficulty. On examination the right half of the pharynx, including the tonsil, was seen to be very much swollen,

œdematous, and bluish red. The left half, including the uvula, was less tumefied, but also somewhat swollen and congested. The epiglottis was very œdematous, greatly enlarged, of a brilliant red colour, and quite rigid. Of the interior of the larynx it could only be seen that it was bathed in muco-purulent secretion; no infiltration was visible or appreciable by touch on the outside of the neck. On pressure of the right side of the neck near the larynx there was tenderness, but not acute. The tongue was covered with a thick white fur. The face looked very feverish, and had at the same time an apathetic expression. The temperature was 101.5° , the pulse 120, weak, easily compressible. There was no cough. The patient was fully conscious. No affection of heart or lungs.

Dr. Bristowe agreed with me in considering the affection as due to septic influences. Ice internally and externally, quinine, cold fluid nourishment, &c., were ordered.

On the next day the patient was much better. The temperature was 99.6° , the pulse 86. He could swallow much more easily. The pharyngeal swelling was much diminished, and had lost its bluish discolouration, and the œdema of the epiglottis had also become considerably diminished. Under continuation of the same treatment the patient got quite well within a few days without any further incident.

CASE 5. *Acute œdematous inflammation of epiglottis and left half of larynx; slight infiltration of left half of neck in a diabetic patient; recovery.*—On December 25th, 1887, I was asked by Dr. Milson to see with him Mr. P—, æt. 47, who had been suffering from diabetes, gout, and albuminuria. There had been a suspicion of renal calculi, and there was a history of syphilis. The patient had had on the 23rd and 24th several shivering fits without any known cause (the drains were stated to be in good order), followed by feverishness, pain in the left side of the neck

close to the larynx, difficulty in swallowing, and since the morning of the 25th also hoarseness. There was no dyspnoea, but an increased amount of albumen in the urine. In the course of the 25th the indisposition increased, and when I saw the patient at 5 p.m. his temperature was 102° , pulse 116, hard and resistant; heart and lungs normal. There was some infiltration in the neighbourhood of the left angle of the jaw, which was tender on pressure. The pharynx was normal, the epiglottis much congested, and together with the left ventricular band and arytaenoid cartilage greatly swollen and slightly cedematous. The movements of the vocal cords were normal. Ice internally and externally and quinine were ordered.

On the 27th, when I saw the patient again, I heard that under this treatment improvement had taken place in the two previous days. On the 27th the temperature was normal, pulse 84. The external infiltration had nearly disappeared, and the laryngeal swelling was much diminished. On the 28th, however, again sudden pain in the throat with difficulty of swallowing was experienced, which lasted about an hour and a half, and then disappeared. When I saw the patient at 5.30 in the afternoon nothing abnormal could be ascertained in the throat or externally, but the temperature was 101° , and the pulse again 96. The next day everything was normal again, and the patient made a good recovery.

A few days later I received a letter from the patient, in which he informed me in confirmation of the correctness of my suspicion (I had expressed a strong belief that he had been exposed to septic influences) that an examination of the house in which his new offices were situated had disclosed the fact that the drain under it was an old brick barrel-drain, containing a considerable deposit of sewage in direct communication with the sewer, and with openings and rat-holes into the basement story, from which an encased rain-water pipe ran up through his room, the casing forming a duct for the air in addition to

a soil-pipe to a w.-c. in the next room. He further stated that he had only taken possession a few days before the onset of the throat attack. It may be observed here that the patient was an eminent architect himself.

CASE 6. *Left-sided tonsillitis; left-sided cedematous inflammation of the pharynx; inflammatory oedema of the epiglottis and of the whole left half of larynx; brawny infiltration of left half of neck; recovery.*—On December 2nd, 1887, I was requested by Mr. Argles to see with him Mrs. W—, æt. 30, who had always enjoyed the best of health, but had fallen ill about a week previously with sore throat. According to Dr. Argles' statements the onset of the disease had been of the nature of undoubted tonsillitis on the left side. For nearly a week apparent improvements and aggravations had alternated. On the evening of the day previous to the consultation, however, the pain, so far complained of as being situated in the neighbourhood of the left tonsil, and the dysphagia, which was localised in the same region, had suddenly travelled further down to the level of the larynx, and a brawny infiltration of the whole left half of the neck from the angle of the jaw to the clavicle had occurred. There had been no rigor or shivering fit so far as could be ascertained, but the patient had been feverish throughout, and the temperature on the morning of December 2nd was 100°, the pulse 120, soft and easily compressible. The drains of the house were stated to be in the best of order. Inquiries as to whether the patient had met with any external or internal injury shortly before the onset of the illness had a negative result.

On examination the whole of the left half of the pharynx was found to be greatly swollen and congested, but nowhere ulcerated; the intensity of the inflammation increased in a downward direction. The epiglottis was enormously swollen, cedematous, brilliantly red, and shiny. The interior of the larynx could on this account not be seen. The voice was slightly hoarse, but there was no

dyspnœa. The dysphagia was very considerable; the infiltrated portions of the neck were, on the whole, little tender on pressure, mostly so close to the larynx. Lungs and heart healthy; no evidence of other complications. Ice was administered internally, and cold applied externally by means of Leiter's tube; five grains of quinine were given every four hours; and nourishing diet was ordered.

Under this treatment the external and internal swelling in the course of the next few days somewhat decreased, and no metastasis of any kind occurred. The temperature began to fall; it was on the evening of the 2nd, 100° , the pulse 104. In the morning of the 3rd temperature 98.4° , in the evening 101° , the pulse being 92.

On the morning of the 4th there was less difficulty in swallowing, and no dyspnœa; slight cough, temperature 99° , pulse 102. The infiltration extended down to half the length of the trachea, and was therefore less extensive than on the 2nd; its hardness also was less. The epiglottis was still much swollen, but not to the same extent as on the 2nd, nor was it so red. Temperature in the evening 100° .

On the 5th further improvement had taken place; temperature in the morning 98° , pulse 96. The external infiltration had much diminished; there was hardly any tenderness on pressure. The patient could swallow better, and the cough which had troubled her during the last day had also diminished. The epiglottis was again less swollen and red. In the evening, however, the temperature again rose to 101.2° , and the cough became more troublesome. Some rhonchi were heard all over the chest.

On the morning of the 6th the temperature was 99° , the pulse 96. The external infiltration was unchanged; internally the swelling of the epiglottis had slightly diminished on the right side, but was unchanged on the left. In the evening the temperature was 98.6° .

On the evening of the 7th the temperature was 97.8° , pulse 92. The patient felt well. The external infiltra-

tion had diminished. The pharynx looked quite normal. The tumefaction of the epiglottis had further diminished also on the left side, and it was now possible to look into the larynx itself and to see that the whole left half was very much swollen. There was much less dysphagia; the cough was still troublesome, but seemed less so than on the previous day. The patient slept well. An iodide of potassium ointment was ordered to be rubbed into the neck.

On the 8th very considerable improvement had taken place. The temperature had become quite normal, the pulse 84 and of good quality. The external infiltration had very considerably decreased. There were no pains, no cough, no dysphagia, and good sleep. The epiglottis was again less swollen; its left half, however, and the left arytaenoid cartilage, were still very oedematous, though somewhat less than on the previous day.

Further improvement had taken place on the 10th. The temperature had remained normal since the 8th, the pulse 80 and of good quality. Externally there was only a small remnant left of the infiltration, corresponding to the outline of the thyroid cartilage. Internally the epiglottis had now entirely returned to its normal dimension, the mucous membrane over the left arytaenoid cartilage was still much infiltrated, but quite pale. The patient got up on that day.

On the 15th the external swelling had entirely disappeared. The left arytaenoid cartilage was still somewhat oedematous. On this day it was ascertained that through a defect in the drainage the sewage from the stables situated behind the house was directly evacuated into the house itself.

On the 20th, when I saw the patient for the last time, the last remnant of oedema could still be seen on the left arytaenoid cartilage, but in all other respects the patient was perfectly well. The urine had been examined throughout her illness, but neither albumen nor sugar had ever been found.

CASE 7. *Acute œdematous inflammation of posterior wall of the larynx; double patchy pneumonia; slight pericarditis; recovery.*—On April 29th, 1892, I was hurriedly sent for by Dr. Arthur Farr to see Mrs. J—, a lady aged about 70, who, after going to bed apparently well on the previous night, awoke in the morning at 6 o'clock with great dyspnœa, which further increased so much that about 11.30 I was telegraphed for to come and be prepared for tracheotomy. On arrival I found the patient propped up in bed, breathing with some but not excessive difficulty, very little stridor, and the voice quite normal. I was informed that she had had a similar attack, though not so bad, some years ago, and that the dyspnœa this time had been much worse, but had already decreased during the last two hours. On examination I found the pharynx perfectly normal, the vocal cords much congested, their posterior ends being close to one another, leaving only a small, elliptic slit for the passage of air, and the posterior wall of the larynx being very œdematous. Rapid up-and-down movements of larynx, no external swelling, no tenderness on pressure, no dysphagia. Pulse 130, very weak and easily compressible. Dr. Farr, who meanwhile had arrived, agreed with me that for the moment tracheotomy was unnecessary. Quinine and ice internally and application of Leiter's bag externally were ordered.

On a further consultation at 3 p.m. of the same day, the aspect of the pharynx and the laryngeal conditions were very much the same. There was no increase of dyspnœa, the pulse was still 130 and very weak and small, whilst the temperature, which had been taken three times since the morning, had always been found normal or slightly subnormal. On the right side of the sternum in the third intercostal space there was a small patch over which distinct fine crepitant râles could be heard, and slight pericardial friction was noticed over the apex. I expressed my conviction that the case was one of septic inflammation, and that the prognosis was serious.

June 30th.—The patient has passed a good night, and her breathing to-day is distinctly easier. The larynx is still congested, but there is much less œdema. The inflammation of the right lung had much extended, and at the time of examination involved the whole upper part of the right upper lobe, whilst also over the base of the left lung tubular breathing was noticeable. There was, however, no pleurisy and not much discomfort in breathing, the number of respirations being only 32. The temperature was gradually rising, and at the time of consultation was a little over 100°, pulse about 120. The pericardial friction at the apex was only very faintly audible, and there was no extension of it. There was no albumen and no sugar in the urine, but an abundance of phosphates.

On the following day I had a letter from Dr. Farr to say that the patient was rather better, that the physical signs in the chest had improved, that the respirations were 30, the pulse 110, of fair quality, and the temperature 100°. The throat felt comfortable. I afterwards heard from Dr. Farr that the patient had made an uninterrupted recovery.

CASE 8. *Acute cedematous inflammation of epiglottis and arytxenoid cartilages (? purulent infiltration) ; double patchy pneumonia ; right-sided pleurisy ; recovery.*—On May 5th, 1892, I was hurriedly sent for by Dr. Henry C. Martin to see with him Miss L—, æt. 14½, and on arrival heard from him the following history. The child, who had been staying with her parents in the country, had had a fortnight ago a sore throat with high temperature, which at that time appeared to be perfectly epidemic in the family. She had, however, entirely recovered from this, and had come to town on the previous day in order to go to a juvenile party. Whilst there she felt rather indisposed. On her return she had an attack of shivering in the carriage, and felt very cold afterwards. Next morning there was great difficulty and pain in swallowing, hoarse-

ness, gradual loss of voice, increasing dyspnoea. Temperature at 11, 104° , and at 12, 105° . Then Dr. Martin sent for me. On arrival I found her in bed, with very slight stridor, quick respiration (36), pulse between 120 and 130, regular and strong. There was absolute aphonia, considerable pain in throat, tenderness on pressure over larynx, but no brawny infiltration. Pharynx normal; epiglottis enormously swollen and cedematous, bright red. Arytæ-noid cartilages similarly swollen. No view of vocal cords obtained, but enough was seen to show that there was still room enough in the glottis not to necessitate immediate tracheotomy. The organs of chest and abdomen all healthy. I expressed at once the opinion that this was a septic inflammation, and prepared for further complications. A Leiter's bag of ice-water was ordered round the neck, six grains of quinine given every four hours, and twenty grains of bromide of potassium every four hours. At 6.45 o'clock the same evening, when we met again, the temperature had gradually gone down to 102° , the larynx was not so much swollen, but very tender outside, especially on the left side; in the third intercostal space on the right side, close to the sternum, a very limited patch was discovered where somewhat prolonged harsh expiration could be heard. Otherwise the chest was normal.

On May 5th at 8 a.m. I heard, on meeting Dr. Martin, that the patient had had on the whole a comfortable night, and had slept at intervals for some length of time. The breathing was decidedly better. She had been repeatedly sick after taking the quinine, which had consequently only been given in half doses during the night. The tongue was very furred, the voice a little stronger than before, the dyspnoea decidedly relieved. Respirations were about 22, temp. 101° , the urine contained neither albumen nor sugar, the bowels had not acted. There was no pain in the chest. On examination the larynx was seen to be less swollen than on the previous day, but the breath was foetid, and the neighbourhood of

the arytaenoid cartilages bathed in thin pus. On examination of the chest, the bronchial expiration at the place in front noted on the previous day was more pronounced. Additionally, there was slight dulness over the left apex, and considerable dulness over both bases with very deficient entry of air.

At 11 a.m. a violent rigor occurred, followed by rise of temperature to 104° . The patient complained of severe pain in the pit of the stomach. Dr. Martin ordered two grains of calomel and a turpentine-and-gruel enema. Temperature in the evening rose to 105° .

At 6.45 p.m. a consultation took place between Sir William Broadbent, Dr. Martin, and myself. The swelling of the larynx had again considerably decreased, and the tenderness outside had also decidedly diminished. Respirations were again 36, pulse 124, and the patient was decidedly weaker, complaining of pain across the chest. In front, over the spot mentioned before, distinct harsh expiration, and in the second intercostal space on the right side a limited patch of pleuritic friction, were noticed. Posteriorly, the inspiratory sounds diminished in intensity below the angles of the scapulæ. On the right side downwards from the scapula there was distinct tubular breathing and crepitation, whilst on the left side a limited patch of the same character just below the angle of the scapula was observed. We all agreed that patchy pneumonia had developed and that the prognosis was serious. It was decided to go on with the quinine, if necessary by the rectum, and to let the patient inhale every three hours some of Brin's oxygen. Stimulants also (iced champagne) were to be taken when necessary. The bowels had acted twice.

May 6th, 8 a.m.—The temperature had gradually fallen through the night to 99° . Pulse 96, weak but regular. The patient had had a tolerable night, but complained of pain in front of chest and at the bases of both lungs. She felt rather weak, but had taken milk, medicine, and stimulants freely. On examination the

larynx was seen so much better that the ice-bag could be discontinued. The voice, which already on the previous evening had come back to some extent, was only slightly hoarse, but there was still some slight soreness of the throat, which on the previous day had been successfully combated by sucking cocaine lozenges (one-tenth of a grain in each lozenge). The larynx externally was still, but only very slightly, tender on pressure. On examination of the chest there was distinct consolidation over both bases posteriorly from the angles of the scapulæ downwards, more so on right side. In front friction and occasional crepitation could be heard in the third intercostal space on the right side near the sternum, and also over the heart in a very limited area close to the left of the sternum. The general aspect of matters, however, was distinctly better. We decided to continue with the quinine and the stimulants, but to discontinue the ice-bag and to use, if necessary, hot poultices every three hours.

6.45 p.m.—The temperature has again risen to 102°, and the pulse to 114 in the course of the day, but without any further rigor. The patient on the whole feels comfortable, but complains of occasional pains across the chest. The voice is almost normal, the slight soreness of throat still present, the epiglottis still somewhat swollen, especially on the left side, the arytaenoid cartilages much less swollen. Vocal cords normal, their movements unimpeded. Pressure on larynx is still somewhat painfully felt, but distinctly less than before. The physical signs in the chest are very much as they were in the morning. The patient begins to suffer from the effects of quinine, and every second dose is reduced to one half.

10th.—In the course of the last four days nothing remarkable has happened, except that on Saturday morning there was a trace of albumen in the urine, which, however, soon disappeared, and has been replaced by a large quantity of phosphates. The temperature has gone

up in the afternoons (see the following Chart), but has nevertheless been steadily decreasing on the whole. The

CHART I.

congestion of the lungs clears up remarkably quickly. On Sunday morning there seemed to be some slight effusion in the right side, but this also is fast disappearing under the use of iodide of potassium and quinine. The larynx is practically normal, there is no pain, and the child feels in every respect much better.

11th.—During the last two days there has been again steady improvement, the temperature gradually falling until it reached the normal to-day, and the congestion of the bases clearing up. At present the right base is almost normal, and only the inspiratory murmur at the left base is still somewhat weak. The throat is perfectly normal. There has been a slight pain in the left shoulder-joint yesterday, but this has entirely disappeared this

morning. On pressure of the left side the larynx is still very slightly tender. Ordered Ung. Iod. one part to Ung. Pot. Iod. three parts, to rub in externally; and as soon as the temperature has become quite normal, to take phosphate of iron, arsenic, strychnia, and acid glycerine of pepsin.

This was the last I saw of the patient, and I afterwards heard from Dr. Martin that she had made a full and complete recovery.

CASE 9. *Acute inflammatory cedema of epiglottis and aryæno-epiglottic folds following transitory inflammation of pharynx; epileptiform convulsions; ataxic respiration; brawny induration of neck; albumen and sugar in urine; delirium; death.*—My next case has a special and very melancholy interest for this Society, inasmuch as its subject was one of its own Fellows, my late colleague, Mr. Francis Mason, F.R.C.S. In describing his case I shall follow in part the report given in the 'Lancet' of June 12th, 1886, by Drs. Ord and Buzzard, in part my own notes taken at the time.

Mr. Mason, who at the time of his demise was only 48 years old, and who had, with the exception of frequent attacks of tonsillitis, enjoyed remarkably good health, had attended on the afternoon of June 1st, 1886, a Staff Meeting at St. Thomas's Hospital at which I happened to be his neighbour. He told me on that occasion that he had never felt better in his life. The same evening, however, he complained of sore throat. The night was tolerably good, but next morning he did not feel very well and sent for Dr. Ord, who examined him and discovered some swelling and redness in the fauces just below the right tonsil, and, in the belief that probably a fresh attack of tonsillitis of a gouty character was impending, prescribed salicylate of sodium and bromide of potassium for him. Although no improvement took place, the patient did his work till 5 o'clock in the afternoon. At that time, however, he felt so ill that he returned

home and went to bed. Previous to this it is remarkable that he ordered his tracheotomy instruments to be placed in his bedroom in readiness. No rigor occurred at the outset of the illness. Almost immediately, however, after he had gone to bed an attack of dyspnoea of such violence followed that the patient thought he would be suffocated. Dr. Ord, who was immediately sent for, found considerable swelling of the epiglottis and sent for me. I arrived at 7.30 p.m. and found the patient sitting upright in bed, the face covered with cold perspiration, but not cyanotic. He was absolutely voiceless, and complained of great dysphagia, so that drinking, even of small quantities of water, caused violent pain in the throat, particularly on the right side at the level of the larynx; he also suffered from a peculiar dyspnoea, or, more correctly said, "hunger for air." There was at that time not the least laryngeal or tracheal stridor. The temperature was 103.5° , the pulse small, most rapid, *i. e.* 140 to 150 beats in the minute, and very compressible. The neck was tender on pressure on the right side near the larynx. External swelling, however, was hardly to be noticed, and only in considerable depth a certain hardness was felt to the extent of about one inch on the right side of the trachea. In the pharynx at that time absolutely nothing abnormal could be seen, neither swelling nor discolouration. The epiglottis, on the other hand, was changed into an enormous, vividly red-coloured, oedematous tumour, which did not permit of the interior of the larynx being inspected.

I at once told Dr. Ord that I was convinced this was a case of erysipelas of the larynx, and Dr. Ord agreed with this opinion. I energetically scarified the epiglottis with a guarded laryngeal knife, and suggested the application of a Leiter's tube with an ice-water current, sucking of small lumps of ice if possible, sulphate of quinine in five-grain doses every four hours, and an aperient. The scarification was followed by expectoration of a large quantity of dark blood, and the patient felt a little easier, but ex-

pressed his firm conviction that his illness would end fatally. The respiration retained its peculiar character. Shortly after the incision the pulse was still 140; the temperature, however, suddenly fell to 99.5°.

At 10 o'clock at night the swelling on the right side of the neck was much more marked, and there was some dusky discolouration of the skin corresponding to it. Watch was set, in which the assistant surgeons of St. Thomas's Hospital alternated. For some hours after the scarification the patient remained easier, but about 2.30 a.m. on the following morning he suddenly ceased to breathe. Mr. Pitts, who was watching him, at once performed tracheotomy under circumstances of great difficulty, only Mr. Mason's wife being present at the time. After the tube had been inserted there followed an attack of epileptiform convulsions. These presently passed off, and the patient recovered consciousness and was evidently greatly relieved, but the pulse remained very quick, ranging above 160, and the respiration was irregular, and, so to speak, ataxic, resembling, but not quite corresponding to, Cheyne-Stokes respiration. On the next morning the brawny induration of the neck extended already to both sides, and up to the death of the patient still further increased, without showing anywhere distinct signs of suppuration in the depths of the tissues. The dysphagia on Friday morning was as great as before. The epiglottis had slightly shrunk at that time, and the two spots where it was scarified the evening previously were distinctly visible. It could now also be seen that the arytæno-epiglottidean folds were enormously swollen. The interior of the larynx proper, however, was still invisible. After this it was impossible to make any further laryngoscopic examination. During the morning of this day, Friday the 4th, the pulse fell in quickness and rose in strength, but the breathing never became quite natural in rhythm. In the evening the improvement ceased and delirium set in. Early on Saturday morning Mr. Mason again appeared to revive, and to a casual observer a remarkable improvement might

have seemed to have taken place: his voice was perfectly clear, the dysphagia had entirely disappeared (our poor friend drank in the presence of all his colleagues assembled for consultation a glass of champagne, in order to show us that he could drink without any difficulty), and pressure on the neck was, in spite of the existing enormous swelling, quite painless, but even at that time his mind was evidently not clear, and the irregularity of the breathing and of the heart's action continued. The revival was transient, and he died at 1.50 on Saturday afternoon, the illness having thus from the very first symptom to the fatal issue occupied not more than sixty to seventy hours. The urine examined in the night of Thursday was loaded with albumen and also contained much sugar; chlorides were suppressed. Under the microscope there were seen granular and hyaline casts, and later on some blood-corpuscles. It is possible that there may have been some interstitial change in the kidneys, but probably the greater part of the albumen and of the sugar were present only as the result of the intensely acute septic attack. On Saturday morning the albumen had diminished very greatly, and the sugar reaction could hardly be obtained. There was at no time any evidence of disease of the lungs or of any of the serous membranes. No post-mortem examination took place.

CASE 10. *Acute œdematous inflammation of (?) pharynx, epiglottis, arytxeno-epiglottidean folds, and mucous membrane over arytxenoid cartilages; left-sided basic pneumonia; death.*—On May 31st, 1890, I was asked by Dr. M. H. Neale to see with him Mrs. A—, æt. 60, who on the previous evening had had a rigor, followed by great pain in the throat, complete aphagia, hoarseness, great dyspnœa, collapse, diarrhœa, and slight delirium. All this had, at the time when I saw the patient, slightly improved. There was no record of temperature, nor had the urine been examined for albumen and sugar. From the history of the attack it deserves to be mentioned that

the patient on the day previous to the occurrence of the rigor had kissed a child, which had been dead for several days, in its coffin.

On examination at 5.30 p.m. there was orthopnœa but no laryngeal dyspnœa, cyanosis of the lips, yellow sodden appearance of the face, moaning respiration, hoarseness but no aphonia, great dysphagia, mental powers slightly obscured. The temperature was 100.8° ; the pulse about 120, extremely irregular. There had been no repetition of the initial rigor.

Over the thorax everywhere rhonchi were heard, but there was no evidence of pneumonia, pleurisy, or pericarditis. The heart-sounds were dull and irregular. The mucous membrane of the pharynx appeared much wrinkled, but not swollen. The epiglottis, the arytæno-epiglottidean folds, and the mucous membrane over the arytænoid cartilages were enormously swollen, of a dull red, in part covered with tenacious muco-purulent secretion; the vocal cords normal; not much stenosis of the air-passages. Great tenderness on pressure on both sides of the neck, but nowhere any infiltration.

I gave a very serious prognosis, and advised the external and internal application of ice, quinine, nourishing fluid diet after previous cocainisation by means of a 20 per cent. spray, and, if necessary, tracheotomy.

On June 3rd I received the following letter from Dr. Neale:

"Mrs. A—, the patient you saw with me on Saturday afternoon, died last evening. After you saw her the pulse improved very much; it became regular for some hours. Then on Sunday the respirations increased, and there was marked tubular breathing at the base of the left lung. The temperature did not rise above 100.7° . The throat symptoms improved slightly, and once or twice on Sunday she swallowed without much trouble. The cocaine, however, did not act well, though I painted and sprayed her well with 15 per cent. solution. I am very sorry I could not examine the urine. Very little

was passed after you left, and always some fæces with it. She was too weak to use a catheter."

CASE 11. *Acute œdematous inflammation of tonsils, pharynx, and larynx; double basic pneumonia; death.*—This case is the most terrible of all I have seen, with regard to the rapidity of its course and its peculiar circumstances, and will justify, I think, my entering a little more fully upon its details.

On June 5th, 1888, a consultation had been arranged between Mr. T. S. Townsend and myself to take place at his house in the afternoon, in order to examine a lady who suffered from some affection of the singing voice. Knowing the nature of the case beforehand, I only took a laryngoscope and mirror with me. When I arrived at Mr. Townsend's house I found the patient only, and a hurried note from himself to the effect that he dared not leave a patient, Sir P. M—, who was suffering from acute laryngitis, and who, he feared, would require tracheotomy to be performed. He added that he wished me to come to Sir P. M—'s residence, which was near by, as soon as I had finished my examination of the lady's throat.

In compliance with his request I hurried to the patient's residence, and found him a strong, fine man of about 56, suffering from orthopnoea, bathed in perspiration, the face dusky, all superficial veins standing out. With him were Mr. Townsend and Mr. Arthur Durham, who had also been sent for. On inquiry I learned that the patient on the previous day had gone in the best of health to Eton to celebrate the 4th of June with his son, who was at school there. He had returned perfectly well, but had awoke early in the morning of the 5th with a violent rigor, followed by rise of temperature, which when taken early in the morning had already been 104°. Shortly after rapidly increasing difficulty of swallowing, which soon amounted to complete aphagia, had made its appearance. He had begun to perspire strongly, and had continued to do so ever since.

About 10 o'clock in the morning Mr. Townsend, who had been sent for, had found tonsillitis and œdema of the larynx on the left side and commencing pneumonia in both bases. By that time the patient could not even drink water. The hoarseness also rapidly increased, and soon amounted to complete aphonia. This in turn was quickly followed by difficulty in breathing, and at 3 o'clock there had been a severe attack of choking. The temperature at that time was 103°. When Mr. Durham, Mr. Townsend, and I examined the patient at 4.30 p.m., there was no longer any swelling of the left tonsil to be seen, but bluish discolouration of the whole pharynx. The larynx was also bluishly discoloured so far as visible (epiglottis and arytaenoid cartilages), enormously œdematous, and covered by frothy secretion. The patient constantly hawked and expectorated partly rusty, partly hæmorrhagic sputa. The neck externally was tender on pressure only over the region of the larynx itself, but no infiltration could anywhere be felt. The heart-sounds were dull but regular; the pulse 80, regular. The urine had not been examined. An examination of the bases of the lungs could not then be made on account of the serious condition of the patient. The dyspnœa at 4.30 was moderate, there was some stridor, but no extensive respiratory excursions of the larynx.

We all three agreed as to the septic character of the disease, but considered tracheotomy not immediately required, particularly in consideration of the serious prognosis anyhow. We advised ice internally, warm poultices round the neck (by Mr. Durham's wish, and nutritive enemata with quinine, as the patient could not swallow anything. Mr. Durham then left the house, and I was about to do the same, when Mr. Townsend requested me to explain to Lady M— (who he knew was much opposed to tracheotomy under any circumstances) how matters stood, and to ask her not to put any difficulties in his way, should the operation after all become unavoidable. I of course complied with his request, and spoke to Lady M— in an

adjoining room. During our conversation the patient had a sudden attack of asphyxia, and both pulse and respiration stopped. I was called in immediately, and, whilst the patient was moribund, performed tracheotomy with my penknife, no other instrument being at hand. I succeeded in rapidly opening the trachea, and held the edges of the wound asunder with bent hairpins, which the patient's daughters, who, as well as his wife, were in the room, supplied, Mr. Townsend meanwhile carrying out for fully fifteen minutes artificial respiration; but all was in vain, the patient was dead. The whole course of the disease, thus, from the initial rigor to the fatal end occupied no more than eleven hours. No post-mortem examination was made.

CASE 12. *Brawny infiltration of neck; congestion of pharynx; general inflammatory œdema of larynx; left-sided pleurisy and pneumonia; death.*—On November 24th, 1885, I saw, with Dr. Chepmell, Mrs. S—, æt. 60, a lady whose only son had just been killed in the Soudanese War, whose general health had greatly suffered since her bereavement, and who had spent all her time in visiting wounded and sick soldiers returned from the war in the Military Hospital. It was known that amongst them there were several septic cases. On November 20th she had a rigor, followed by pain in the throat, shortly afterwards by dysphagia, and finally by dyspnœa. When I saw her in the morning of the 24th there was very great dyspnœa, tracheal stridor, cough, and sero-purulent expectoration; the temperature was 101.5° , the pulse 120. There was great prostration, and the patient sweated profusely. Her face was of a dusky colour, the sensorium rather obscured. The entire neck presented brawny infiltration, but without much discolouration. The pharynx and larynx were greatly congested, the latter slightly œdematous all over. The breath-sounds were rough, but otherwise normal; no abnormal dulness anywhere over the thorax. A Leiter's tube was applied round the

neck, and ice was given internally, together with an expectorant; a croup tent was erected over the bed, and plenty of nourishment and stimulants administered. My prognosis was very serious. In the evening of the same day the breathing was slightly better, temperature, however, 102° , and pulse 128. The patient was very restless. Quinine (5 grains every four hours) was added to the medication. After a restless night the temperature, taken in the rectum, on the morning of the 25th, was 103° ; the pulse 132, very weak, slightly irregular. The patient complained of violent headache and frequent sickness. The breathing and the expectoration were easier, but there was distinct dulness both in front and posteriorly over the base of the left lung, where also crepitation and pleuritic friction were heard. The patient was evidently sinking. In the afternoon a consultation was held with Sir William Gull, who corroborated the bad prognosis I had given in the morning. The temperature then was 104° , and the patient very comatose. Death took place the same evening. No post-mortem examination was held.

CASE 13. *Acute œdematous inflammation of epiglottis, aryteno-epiglottic folds and mucous membrane over arytenoid cartilages; abscess over left arytenoid cartilage; brawny infiltration of neck; double pleurisy; delirium; attempted suicide; death.*—On May 11th, 1893, I was requested by my colleague, Dr. Payne, to whom I am much obliged for permission to make use of this case, to see in Arthur (small) Ward of St. Thomas's Hospital, T. B—, æt. 37, a porter at one of Messrs. Spiers and Pond's establishments, who had been admitted on the previous day on account of constriction and soreness of throat of one day's duration, with inability to eat or to drink. He had previously enjoyed good health, and had only once, three years before, suffered from bad sore throat and a submaxillary swelling, which had been incised in Westminster Hospital. His present illness began on May 9th with the symptoms mentioned. About twelve

hours later he had a shivering fit, with aggravation of the symptoms. He complained much of the offensive odours of the refuse which in his capacity as porter he had to carry about.

CHART II.

Temperature chart of Case 13 from admission until death.

On admission, at 10.30 p.m., the patient spoke with a low gruff voice, metallic in quality, coughed constantly, and showed evidence of respiratory obstruction.¹ His pharynx was normal, the epiglottis slightly red and swollen, the arytaenoid cartilages and the arytaeno-epiglottidean folds greatly swollen, deep red, and evidently acutely inflamed. The vocal cords were slightly red, and their abduction impaired. There was much muco-pus in the larynx. Externally a brawny inflammatory cedema extended from the lower jaw to the clavicle on the right

¹ For the notes of this case and of the post-mortem examination I am indebted to Mr. Box, Medical Registrar to St. Thomas's Hospital.

side. At 12.45 a.m., *i. e.* about two hours after admission, tracheotomy became necessary, and was successfully performed in spite of much lividity and of free venous hæmorrhage. At the same time the inflammatory swelling in the neck was incised, but only serum escaped. The temperature on admission had been $100\cdot6^{\circ}$, at 4 p.m. it was 100° , at 8 p.m. $99\cdot4^{\circ}$, at midnight $99\cdot6^{\circ}$. During the next twelve hours after tracheotomy the patient expectorated about one ounce of thin, dark coloured, but not offensive pus.

On May 11th at 10.30 a.m. his voice was much improved. In the larynx an abscess over the left arytaenoid cartilage had burst, and the swelling had largely subsided, so that the cords could be seen for their entire length; they moved well. Deglutition was still painful. The tracheotomy tube was removed, and the interior of the larynx painted with Tinct. Ferri Perchloridi. There was still much cervical swelling and tenderness, but no purulent discharge.

In the afternoon I was requested to see the patient, and made the following note: "pharynx free, epiglottis still thickened, the mucous membrane is corrugated and bright red, the swelling of the arytaenoids is hardly noticeable, the movements of the cords are free, much tenacious pus in larynx."

In spite of the apparent improvement and of the absence at that time of any thoracic or other complication, I expressed the belief that the case would take a very serious course. The temperature during the 11th was at 4 a.m. $99\cdot2^{\circ}$, at 8 a.m. $99\cdot6^{\circ}$, at noon $99\cdot8^{\circ}$, at 4 p.m. $100\cdot8^{\circ}$, at 8 p.m. $100\cdot6^{\circ}$, at midnight $100\cdot4^{\circ}$. On the 12th the temperature at 4 a.m. was 100° , at 8 a.m. $101\cdot6^{\circ}$, at noon $102\cdot6^{\circ}$, and at 4 p.m. 102° .

The next note is dated the 12th, 5 p.m.: Respiration 56, pulse 104, temp. 102° . The patient was then delirious. There was evidence of right pleurisy and (?) pneumonia. The patient attempted suicide by stabbing himself with scissors, and was very violent. At 8 p.m.

the temperature was 102·4°. Death occurred somewhat suddenly at 12.15 a.m.

The post-mortem examination, which has an additional melancholy interest in the fact that it was the last which was performed by my late lamented colleague, Dr. Hadden, is summarised as follows :

Recent pericarditis with a little turbid effusion. Heart normal. Recent lymph over both lower lung lobes and corresponding costal pleuræ. Lungs simply congested. Liver cloudy. Spleen large and soft. No pus in tissues of neck. Much thickening of pillars of fauces, epiglottis, and ary-epiglottidean folds. A small grey slough on the posterior extremity of the right arytæno-epiglottidean fold, covering a small abscess cavity and extending in the direction of the arytænoid cartilage. Interior of the larynx red, swollen, and covered with thick mucus. Much mucus in trachea and bronchi. A small slough involving mucous and submucous coat over a tracheal ring just below the tracheotomy wound (? pressure of tube). Uratic deposit in right great toe-joint; no pus in joints. No record of examination of brain.

I deeply regret that owing to a mistake I was not informed of the death of the patient nor of the date of the post-mortem examination, and therefore was not present at the latter. The specimens unfortunately were not preserved.

After the patient's death a culture was taken from the secretion of the tracheotomy wound on agar by the house physician, handed to Mr. Shattock, and at once placed in the incubator. A pale yellowish growth developed within the next few days, which on microscopic examination proved to be a *Staphylococcus*. Sub-cultures of this were made in broth, and the fresh cultures injected subcutaneously in the ears and backs of rabbits. No local or general reaction ensued. The experiment was repeated several times with different broth-cultures, but always with negative results. Finally a rabbit was

fed with a large quantity of broth culture, but with equally negative result.

CASE 14. *Brawny induration of neck; follicular tonsillitis; acute inflammatory œdema of epiglottis and left arytxenoid cartilage; deep-seated abscesses in neck; double pleurisy; peritonitis; superficial abscesses; death.*—This is the case from which, as repeatedly mentioned, my occupation with the subject practically dates, and which I have described in vol. xii of the ‘St. Thomas’s Hospital Reports.’

CHART III.

Temperature chart of Case 14 from admission until death.

It is therefore only necessary here briefly to repeat that the patient was a man æt. 31, who came on December 29th, 1882, to St. Thomas's Hospital with a swelling occupying the whole left upper half of his neck, which was tender on pressure, hot, cedematous, and of an erysipelalous redness. At the same time there was acute

follicular tonsillitis and enormous œdema and discolouration of the epiglottis and the left arytaenoid cartilage. The patient was taken into the hospital. He soon became delirious; his temperature varied between 105° and 100° ; pleurisy developed, and abscesses in the left side of his neck as well as on the hands; and he died on the 9th of January, 1893. An autopsy was made, and the post-mortem diagnosis was: pyæmia ex causa ignota, abscesses in neck, œdema of larynx, double pleurisy, peritonitis, superficial abscesses.

For further details I must refer readers to the original.

The foregoing series of cases represents my personal observations of acute septic inflammations of the throat and neck. If these observations had to be classified according to the nomenclature more or less generally adopted at present, I have no doubt that they would be referred to chapters which are kept entirely distinct from one another in most recent text-books. Case 1 would no doubt go down as an instance of "violent catarrhal inflammation of the palate and pharynx," Case 2 as acute "epiglottitis," Case 3 as "idiopathic œdematous laryngitis," Case 4 as "violent catarrhal inflammation of the tonsils, pharynx, and epiglottis;" although in this case already the likelihood of a septic origin would probably become apparent to many observers. In Case 5 again an experienced observer would no doubt suspect from the fact that one half of the larynx only was in a condition of œdematous infiltration, and that at the same time some slight infiltration of the left half of the neck existed, that the origin of the disease was septic. But differences of opinion might arise as to whether this case had better be called a case of septic laryngitis, of erysipelas of the larynx, or of Ludwig's angina with participation of the larynx. Difficulties of a similar nature present themselves in Case 6; but I think that most observers, if they had seen the patient for the first time at the period of the illness at which she came under my observation,

would not have hesitated to call it a case of erysipelas of the larynx. The next Cases, 7 and 8, would, I believe, be unhesitatingly classified by most members of the profession as instances of erysipelatous inflammation of the larynx with subsequent extension to the thoracic organs, although in Case 8 the fact that at one time the larynx was bathed in thin pus could not but raise the question whether this was a pure erysipelatous inflammation, or whether there was possibly at the same time a purulent infiltration of invisible parts of the upper air-passages. In Case 9, again, to any observer who had not watched the case from the beginning the difficulty of deciding whether it was a case of erysipelas of the larynx with subsequent extension to the cellular tissue of the neck and early and severe involvement of the central nervous system, or *vice versa*, whether it was a case of Ludwig's angina with subsequent extension to the larynx and implication of the highest nerve-centres—would have been very considerable, although in view of the fact that the development of matters fortunately was carefully watched by competent observers from the very onset leaves hardly any doubt that the former of the two alternatives corresponded to the actual facts. Cases 10 and 11, I think, would have been looked upon as instances of erysipelas of the larynx followed by pneumonia. In Case 12 again we meet with the difficulty—this time greater than ever—of deciding whether, according to the present nomenclature, it should be classified as an example of Ludwig's angina or of erysipelas of the larynx. Case 13 would probably be regarded by some as an example of erysipelas of the larynx, by others as one of phlegmonous laryngitis, and again by others as an example of Ludwig's angina; whilst in Case 14, finally, as already stated in a previous paragraph, good reasons could be adduced for classifying it under any of the headings—erysipelas of the larynx, Ludwig's angina, deep-seated phlegmon of the neck.

It will have been seen from this list that my personal observations do not appear to include a typical example

of the affections described by Carrington and Hale White and by Senator under the titles of "phlegmonous pharyngitis" or as "acute infective phlegmon of the pharynx," *i. e.* cases in which a diffuse purulent infiltration in the deeper tissues of the pharyngeal mucous membrane existed, which thence is propagated to the larynx and the glands, and secondarily also invades other organs. Apart, however, from the fact that the clinical appearances and the course of the disease in several of my more severe cases closely resemble the descriptions given by Senator himself of his own cases, it is quite possible that if a post-mortem examination had taken place in all my fatal cases, conditions similar to those described by the authors just named might have been found. My two last cases, at any rate, plainly show that an inflammation of the pharynx and larynx which clinically has all the appearances of erysipelas may be accompanied by formation of pus in the neighbourhood of the œdematous parts (in Case 13 in the larynx itself, in Case 14 between the muscles of the neck), and I think it will be generally conceded that no rational differentiation could be established on the sole strength of the fact that in one instance an apparent infiltration exists in the deeper tissues of the pharynx, and in the other in such close neighbourhood to that part as in my two last cases.

But all the difficulties mentioned in the last paragraph are, I venture to believe, of our own making, and will disappear when we have the courage to disabuse our minds of the artificiality of the present system, and to analyse, untrammelled by dogmatic teaching, the essential facts adduced in my observations from the point of view of an unprejudiced critic.

Now what are these facts?—We have before us a number of cases of acute inflammation of the throat and neck, attacking in most instances previously healthy persons, varying enormously in intensity, varying also in primary localisation and in the fact that in some of them the inflammation remained limited to the part or parts

originally attacked, whilst in others an extension occurred, either by contiguity or by metastasis to other organs, and, finally, varying in the character of the exudation, inasmuch as in most cases, so far as could be ascertained, it was of a serous, and in two cases at any rate of a partly purulent nature, whilst in one case (Case 13) a fibrinous exudation accompanied the pleurisy, which was one of the complications set up by the disease. But all these cases were during life characterised by symptoms which both subjectively and objectively not merely resembled one another, but which in reality formed a practically uninterrupted scale of increasing severity of one and the same process, namely, of a septic inflammation characterised by oedematous infiltration of the tissues attacked. It appears to me not merely the simplest, but also the most natural explanation, and one entirely in concord with the general state of our present knowledge of septic inflammations, that the differences in severity, extension of the process and nature of the exudation (*i. e.* whether serous, fibrinous, or purulent) observed in the different cases, depended upon various degrees of *virulence*, and probably also on the *quantity* of the pathogenic micro-organisms which in the individual instances caused the disease, whilst the accident of *primary localisation* was in all probability in each case determined by accidental breaches of surface forming the suitable portal for the entrance of these micro-organisms into the body. There appears to me not the least necessity for subdividing the cases—and thereby merely confusing the issue—into rigid conventional forms, which do not stand, as my series so plainly shows, the test of practical experience; and so strongly am I convinced of the unity of the different forms, that on the strength of my own observations, joined to Carrington and Hale White's and Senator's experiences, I venture to submit, in the following lines, a sketch of the clinical course of these septic inflammations of the throat and neck, which, though no doubt as yet not quite complete, may serve as a skeleton of the clinical and pathological

events, to be invested hereafter by future observers with more complete details.

I should describe the course of events in these cases as follows :

Acute septic inflammations of the throat and neck may be caused by the action of *various* micro-organisms (on this point more in conclusion of this paper), and vary in primary localisation, extent, and complications in accordance with the virulence and probably also the quantity of the pathogenic factors. They may, and do, attack previously healthy persons of both sexes, and apparently of all ages. Nothing definite is as yet known about the length, and indeed the existence, of an incubation stage, whilst prodromal symptoms, though apparently present in some cases and manifested by general malaise, headache, sore throat, and some febrility, may in other, and indeed in the worst cases, be entirely absent. The onset of the disease is usually very sudden, and is manifested by a rigor followed by quick rise of temperature ; but this rigor, too, may not be present, or may be only of the nature of slight shivering, or may occur after some of the actual symptoms of the affection have already made their appearance. Very rarely repeated rigors occur, and, if so, point to further complications. The course of the fever in these cases does not appear to follow any definite rule, and very probably depends upon the virulence of the septic infection in every individual case. In some cases, though of a severe type, it does not rise at all much over 100°, whilst in others it goes up to 105° and more. The only general conclusion so far admissible seems to point to the fact that the highest temperature in most cases is reached at the very onset of the disease, and that thereafter it gradually falls, although in the event of further complications taking place a renewed rise of temperature may accompany their onset. The pulse very rarely reaches excessive frequency, and during the acute stage of the disease as a rule varies between 110 and 130. It is characterised in most cases

by weakness and by being easily compressible. An irregularity of the pulse and of respiration appears to occur in very rare cases in which an early involvement of the central nervous system has taken place.

With regard to the subjective complaints, the first and almost general one is that of pain in the throat and great difficulty in swallowing, which within a few hours usually amounts to complete aphagia. This difficulty appears to be always present, whether the original seat of the septic inflammation be in the pharynx, the larynx, or the cellular tissue of the neck. It is, in those cases in which the larynx is involved or becomes so in the further progress of the inflammation, followed by hoarseness, loss of voice, and dyspnoea, which may or may not bear a laryngeal character and be accompanied by stridor. These symptoms are attended by a general feeling of great malaise and prostration. The aphagia lasts in some cases for hours, in others for a day or two, but in all cases in which recovery takes place is distinguished by remarkably quick subsidence, as are, indeed, all the other subjective symptoms complained of.

The objective symptoms vary, of course, in accordance with (*a*) the localisation and (*b*) the extension of the inflammatory process. In the great majority of cases it appears that the pharynx and particularly the tonsils are the parts first attacked, forming as they do the natural portal for the entrance of micro-organisms into the body. (This point will be more fully discussed further on.) Needless to say, however, it is quite possible that the pathogenic organisms may pass these parts and primarily settle in any other locality in the throat in which an accidental breach of surface favours their entry. Thus, in some cases the larynx—and here above all the epiglottis,—and in a few others the cellular tissue of the neck, will be the original seat of the septic inflammation.

In those cases in which the tonsils and pharynx are first affected these parts very quickly swell to a considerable degree. The inflammation of the tonsils does not

seem to be distinct in anything from ordinary follicular tonsillitis; if the posterior wall of the pharynx, however, be affected, the swelling is much more developed and of a much more oedematous character than in ordinary catarrhal inflammation, and the colour of the inflamed part, whilst in the majority of cases of a bright scarlet, in some instances shows a distinct bluish, sometimes almost dark blue hue. The swelling may remain limited to the pharynx, and after a day or two retrogressive changes, and finally recovery, may take place without the larynx ever becoming affected. If the swelling has been very considerable, the mucous membrane of the affected portion during the retrogressive changes shows a wrinkled appearance. In the very worst cases in which the pharynx has been the primary seat of the disease (cases such as have been described by Carrington and Hale White and by Senator), the whole brunt of the inflammation appears to fall upon that part, and in such cases (in which generally the disease ends in death), after a few days a diffuse purulent inflammation is found in the deeper tissues of the pharyngeal mucous membrane, the inflammation being propagated to the larynx and the glands and secondarily also invading other organs. Needless to say the septic inflammation may spread from the pharynx in other directions than downwards, and may for instance attack the nose, the naso-pharyngeal cavity, the external integument, or the brain. A case in which a phlegmonous pharyngitis was followed by purulent meningitis has recently been reported by von Stein.¹

In the great majority of cases, however, it is characteristic of the inflammation, when beginning in the pharynx, that it appears to completely leave this part after a few days or even hours and to extend further downwards to the larynx. The previous observations so far recorded all point in the direction that if the latter organ be attacked by septic inflammation, be it by propagation from the pharynx or be it primarily, the epiglottis

¹ 'Monatschrift für Ohrenheilkunde, &c.,' No. 10, 1894.

appears to be singled out prominently to bear the brunt. Next to this part of the larynx the arytaeno-epiglottidean folds and the mucous membrane over the arytaenoid cartilages appear to suffer, whilst not much can be stated as yet with regard to the conditions of the interior of the larynx, the swelling of the vestibule, as a rule, preventing inspection of the vocal cords. From the hoarseness and aphonia, however, attending in many instances the laryngeal inflammation, it is very likely that the interior of the larynx, though possibly to a lesser degree, is also involved in the inflammatory process. The appearance of the epiglottis, arytaeno-epiglottidean folds, and arytaenoid cartilages in these cases is characterised by enormous tumefaction, in consequence of which particularly the first-named part is enlarged to four times or even more its natural size, and presents itself as a semi-transparent scarlet or bluish-red roll, lying across the pharynx. Such dimensions does this swelling sometimes assume, that even the arytaeno-epiglottidean folds and the arytaenoid cartilages are quite concealed from view.

At this stage, in the great majority of cases, there is steadily increasing dyspnœa, aggravated not rarely by attacks of suffocation, which, if not in time relieved by tracheotomy, may end fatally. In very severe cases the exudation becomes partly purulent, and either a diffuse purulent infiltration of some parts of the larynx may be met with, or a distinct abscess may form, which may burst spontaneously, as in Case 13 of my list. In a number of cases the inflammation remains limited to the larynx; in other cases, however, it extends from this part to the neighbouring tissues of the neck, and causes an infiltration of these parts, which may concern either the immediate neighbourhood of the larynx only, or extend over the whole of one or even both sides of the neck. The skin of the neck in such cases is hot, and so tense that it is difficult to make out any details within the general infiltration. The parts are also, as a rule, very tender on pressure. The infiltration is hard, brawny, and its colour varies from slight redness to a

dusky violet. In the worst cases it extends from the lower jaw to the clavicles or even further down, and the outlines of the neck may be completely lost. An erysipelatous redness is found in such cases to extend even further than the infiltration itself. The exudation into the cellular tissue of the neck in most cases of this kind seems to be of a purely serous character. In other cases, however, either diffuse purulent infiltration is met with, or localised abscesses exist in the cedematous cellular tissue or between the muscles of the neck. In the very worst cases metastatic abscesses occur either in superficial parts or in joints.

Whilst the purulent variety of the septic inflammation usually leads to speedy death, cases of serous inflammation of the larynx and its neighbourhood may get well, however considerable the swelling has been, within a few days. Here, again, it is characteristic that the maximum of the inflammation usually is attained within a few hours from its very onset; and that in the cases in which recovery takes place, already in a day or two after the commencement considerable diminution of the swelling forms the rule. Within a few days nothing but a wrinkled appearance of the mucous membrane may give evidence of the enormous swelling which only shortly before formed the most conspicuous sign of the severe illness.

Often, however, the disease does not remain limited to the neck, but spreads—sometimes with incredible rapidity—to other parts. In addition to the lungs, the serous membranes particularly are liable to become attacked, and pleurisy, unilateral or double, pericarditis, peritonitis, and as already mentioned, meningitis, may be developed within a few days or even hours from the initial rigor. When the lungs are attacked, either basic or patchy pneumonia in one or both lungs shows their involvement in the pathological process. Not rarely several of these complications are met with in one and the same case. The exudation in the serous membranes may be either of a serous, fibrinous, or purulent character; hæmorrhagic effusions have not been met with in

my own observations. Even in cases complicated by pneumonia, pericarditis, and pleurisy, recovery is possible, and, if occurring, is remarkable for its quickness and completeness. In more severe cases, however, death ensues under signs of increasing coma and heart failure, and in the worse cases of this character the whole process from its beginning to the fatal end may not occupy more than ten to twelve hours.

In very rare cases it appears as if the whole brunt of the septic infection, apart from the parts first attacked, fell upon the central nervous system. In such cases epileptiform convulsions, delirium, irregularity of heart and pulse action, are amongst the earlier symptoms, and death may occur under signs of severe septic infection of the nervous system, without any complications developing in the chest, and after the local inflammation of the pharynx and larynx has already completely subsided.

In a final series of cases—those which have been hitherto described as cases of angina Ludovici—the primary localisation of the inflammation is the cellular tissue of the neck. The pathogenic micro-organism may penetrate into this part either through an external breach of surface or through a carious tooth, through the tonsils, the lymphatic glands of the tongue and pharynx, &c., &c. In these cases the brawny infiltration of the neck will be the first objective symptom observed, and the participation of the pharynx and larynx only occur at a somewhat later period. In other respects, however, the events observed are absolutely identical with those seen in the cases in which the pharynx or larynx were the parts first attacked.

The foregoing description may serve as a clinical sketch of our present knowledge of these septic inflammations of the throat and neck. It shows, I hope, that my conception of the unity of these various processes is not an arbitrary one, but one fully in accordance with the clinical and pathological facts which have come within my own knowledge.

Whilst thus convinced of the pathological identity of these various processes, I am, needless to say, fully aware that a number of important objections could be raised against my views, and I am myself conscious of the following four which demand an immediate reply :

1. That I had given no proof that the milder cases in my list were really of septic origin, and that the symptoms observed in them may well have been the result of a simple catarrhal inflammation.

2. That the different localisation in these cases, namely, whether originating in the pharynx, larynx, or cellular tissue of the neck, spoke against their being identical.

3. That the variations in the fever curve in the individual cases also seemed to point in the direction that this fever was caused by different, not by identical, processes.

4. And above all, that the fact of the exudation sometimes being of a serous and sometimes of a purulent character most powerfully combated the view that these inflammations were identical in nature.

1. With regard to the first, I must at once admit that I am not able to prove beyond doubt that the milder cases I have adduced are of undoubtedly septic character. A real proof could of course only be adduced in the shape of cultures of pathogenic micro-organisms being obtained from such slighter forms of cedematous inflammation as those observed in my first four cases. This I have not been able to do myself, and it must be left to future observers to adduce such stringent proofs. Already at present, however, I wish from the *clinical* point of view to state that the inflammation in these cases is unlike an ordinary catarrhal one. I am fully aware that an cedematous inflammation can be brought about by other causes than septic ones, that it may be the result of chemical, thermic, and traumatic irritation as well as that it may represent the acme of an intense catarrhal process, but in cases of the three former categories, if met with in

adults, one is almost always able to make out a definite cause for the occurrence of the acute œdematous inflammation; whilst in the instances in which a mere catarrhal inflammation leads to œdema, the latter forms as it were the *climax* of the inflammatory process, and is not reached until after some measureable time. In the cases here under consideration, however, the course of events is quite different. The process *commences*, without any demonstrable immediate cause, with the appearance of an intense œdematous inflammation, the maximum of which, as I have tried repeatedly to point out, usually is reached at its very onset; it further *singles out* certain parts of the upper air-passages, and is not general from its very beginning, as seen in ordinary catarrhal inflammations. Further, the *dimensions* of the œdematous swelling far surpass what is observed in an ordinary catarrhal inflammation; again, the attendant *constitutional* symptoms, shivering, rigor, febrility, and general feeling of malaise, point to a septic origin; and, finally, I wish to point out that even in the slightest of my own cases in several instances definite proof was obtained of the patients having been exposed to *septic* influences, such as sewer gas and drainage poisoning. I fully admit, however, as already stated before, that further bacteriological research must definitely decide whether the slightest forms of œdematous inflammation of the pharynx and larynx are, in reality, due to septic causes.

The second objection which might be raised against my view of the unity of these various processes, namely, that they must differ in kind because they differ in primary localisation, is one which I think it is not difficult to refute. This can best be done, it appears to me, by a simple reference to the analogous case of diphtheria. Diphtheria remains diphtheria, whether it primarily attacks the pharynx, the larynx, the nose, the bronchial tubes, the conjunctiva, the vulva, the rectum, or any wounded surface, and equally so whether it remains limited to the part first affected or whether it spreads to

other parts by contiguity or auto-infection. Obviously it will be most frequently primarily met with in the pharynx, because that part, from its situation, its function as the portal of respiration, and from the histological configuration of some of its constituents, notably of the tonsils, forms the most natural highway for the entrance of pathogenic micro-organisms. In cases, however, in which the pharynx happens to be healthy, whilst the larynx is in a catarrhal condition with some lesion of the epithelium, or in which for any other reason the latter has been injured, it is quite conceivable that these micro-organisms may pass through the pharynx and primarily settle in such parts in which favorable conditions for their further development offer themselves.

It appears to me that the question of the primary localisation of acute septic inflammations of the throat and neck is absolutely analogous to the one just discussed. From theoretical reasons as well as from the experience, not only of myself but also of other observers, the pharynx forms the starting-point of the septic inflammation in the vast majority of cases, and I wish here particularly to draw attention to the *rôle* played by the anatomical constitution of the tonsils, not only in this but also in other forms of infectious diseases. The epithelial covering of these glands shows gaps large enough to allow a transit of leucocytes, and Philipp Stoehr¹ has drawn attention to the fact that an enormous transit of such cells undoubtedly occurs in the tonsils, without the epithelial strands being actually destroyed. Obviously an anatomical arrangement of this kind fully suffices to explain the predilection with which infectious processes occurring in the mouth localise themselves primarily in the tonsils, and I believe that in a not far distant time these glands will also in a good many cases be held responsible for the entrance of the tubercle bacillus and possibly of other micro-organisms into the system. In

¹ 'Biologisches Centralblatt,' vol. iv, No. 12, und 'Sitzungsbericht der Physikalisch-medizinischen Gesellschaft zu Würzburg,' 1883, No. 6.

some papers recently published in Virchow's 'Archiv' by Emil Schlenker,¹ who worked with Dr. Hanau, and by Emil Krueckmann,² it was shown that tuberculosis of the cervical lymphatic glands almost always depends upon the invasion of these glands by the bacillus *from the tonsils*, and in no less than about 60 per cent. of cases of tuberculosis of the lungs examined on the post-mortem table by the last-named observer, tubercles were detected in the tonsils, similar results having been previously obtained by Strassmann and Dmochowski.

That diphtheria preferentially makes its first appearance on the tonsils is too well known a fact to be insisted upon at any length. In the series of cases, too, which I have adduced in this paper, it will have been observed that in a considerable proportion of the cases tonsillitis formed one of the initial symptoms of the septic inflammation.

Facts of this character go far, I think, to explain the different localisation of the septic inflammations of the throat and neck. In the majority of cases their first seat will be the pharynx, and the disease may either remain limited to that part or spread thence; in other cases, however, the micro-organisms may pass through gaps in the epithelial covers of the tonsils or the follicles at the base of the tongue or in the floor of the mouth, and find their way into the cellular tissue of the neck, where, of course, they may also penetrate by other breaches of surface, by carious teeth, &c. In the third class of cases they may with the inspiratory current of air be sucked through the pharynx and be directly inoculated into the larynx, which probably in such instances has become more vulnerable by a previous catarrhal inflammation or by some other cause leading to breaches of surface. Future observers

¹ "Untersuchungen über die Entstehung der Tuberkulose der Halsdrüsen, besonders über ihre Beziehungen zur Tuberkulose der Tonsillen," Virchow's 'Archiv,' vol. cxxxiv, pp. 161 *et seq.*, and 247 *et seq.*, 1893.

² "Ueber die Beziehungen der Tuberkulose der Halsdrüsen zu der Tonsillen," Virchow's 'Archiv,' vol. cxxxviii, p. 534, 1894.

will have to investigate why in such cases the brunt of the inflammation preferentially falls upon the epiglottis, a part in which the mucous membrane is closely attached to the perichondrium of the cartilage. Enough however, I think, has been shown to render it highly probable that the question of primary localisation after all depends only upon *accidental* circumstances of the individual case, and considering the identity of the clinical and pathological events occurring afterwards, I think it will be generally admitted that no real difference can be established between these septic processes on the mere strength of their having shown themselves in one case first in the pharynx, in another in the larynx, in the third in the cellular tissue of the neck.

To two points I wish to draw attention in conclusion of this part of my subject. It is remarkable that in the cases in which the process begins first in the pharynx, and in which extension occurs, this extension should so decidedly take place in a *downward* direction. It will have been observed that in all my cases the nose and naso-pharyngeal cavity remained unaffected, however intense the inflammation might have been in the close neighbourhood of those parts. I will, of course, not for one moment maintain that this is the universal rule. It is well enough known that erysipelas sometimes primarily appears in the nose, and thence spreads through the naso-pharyngeal cavity into the pharynx and further down, or that it extends from the nose to the external integument of the face; but anyhow it appears to me very remarkable that in all my cases nose and naso-pharynx should have so entirely escaped. The case mentioned previously, however, observed by von Stein, certainly shows that this is not an unexceptional rule, and that an originally pharyngeal septic inflammation may also extend in an upward direction.—Secondly, it may be thought by some rather revolutionary that I venture to include Ludwig's angina amongst those cases in which the septic inflammation originally beyond doubt started from the mucous mem-

brane of the upper air-passages. My reply to this is that the whole existence of this affection as a disease *sui generis* has been by no means fully established; that it was first described at a time (1836) when no examination of the larynx could be made, and that it is already admitted¹ that its ultimate cause consists in the immigration of micro-organisms through some spot of the mucous membrane of the mouth denuded of epithelium. It therefore appears to me not justified by the real facts, to artificially maintain a difference from the mere accident of the primary localisation in the cellular tissue of the neck, whilst the affection itself is pathologically identical with those taking place in the mucosa and submucosa of the respiratory mucous membranes.

The third objection to the view of the unity of the forms of septic inflammations of the throat and neck, namely, that the fever in the different forms takes a different course, is one that can be dismissed, I think, with very few words, and indeed I only mention it particularly because Massei lays great stress upon the fever curve as characteristic for the form of septic inflammation he has described as primary erysipelas of the larynx. It appears to me, however, that the height of the temperature and the fever curve in these cases will so entirely depend (*a*) upon the quantity and the virulence of the poison which has penetrated into the organism, (*b*) upon the question whether the resulting inflammation be of an œdematous or of a purulent or of a mixed character, and (*c*) possibly also upon the question in which tissues it was first localised, that one cannot expect the fever in these cases to take a simply typical form. I can again for the purpose of illustrating what I mean do no better than return to the analogous case of diphtheria. In this disease also the degree of fever varies most markedly—whilst there is never any tendency to extreme hyperpyrexia, the temperature in the severest and most malignant cases is

¹ See, for instance, Schech, 'Die Krankheiten der Mundhöhle, des Rachens und der Nase,' 3rd edition, 1889, p. 81.

often quite low.¹ Yet nobody has ever suggested, so far as I know, that the difference of temperature in various cases of this disease constitutes a difference of kind between them. Further observations, more accurate than I have unfortunately been able to make them in a good many of my cases, will have to show whether any general type of fever after all exists in these septic inflammations, but whatever the results of such an inquiry may be, they cannot influence, it appears to me, our general views as to the identity or non-identity of the individual forms.

The most important objection, however, to my view of the unity of these septic inflammations, and the one which is sure to be raised, is this, that in a number of cases the inflammation was of a purely cedematous, and in another class of an entirely purulent character, and that this fact decidedly pointed to these two classes being of different origin. A few years ago, indeed, this objection would have been simply fatal to my suggestions. It was then dogmatically taught and universally believed that there were two different pathogenic micro-organisms, the one the *Streptococcus pyogenes*, the other one the *Streptococcus erysipelatosus* (Fehleisen's), and that these two micro-organisms, though indistinguishable under the microscope and in pure cultures, yet were essentially different, inasmuch as the former always and without exception produced a purulent, the second equally unexceptionally an cedematous, form of inflammation.

Cases such as my two last would under this teaching of course have been referred to a *mixed* infection, which explanation is at any rate a very convenient one. In the course of the last few years, however, the bacteriological opinion concerning this question has very considerably veered round. Already in 1891 the specificity of Fehleisen's erysipelas coccus had become more and more doubtful, and its real identity with the *Streptococcus pyogenes* more probable. To fully prove this, however,

¹ Morell Mackenzie, 'Diseases of the Throat and Nose,' vol. i, 1880, p. 142.

two more points were wanting, namely, the demonstration in the human subject that the *Streptococcus pyogenes* could also produce erysipelas, and, secondly, the demonstration that also other pyogenic micro-organisms could produce the same result. This proof was definitely given at the meeting of the German Surgical Society in 1891 in an able paper "On the Ætiology of Erysipelas,"¹ read by Dr. Max Jordan, Professor Czerny's assistant at the Surgical Clinique of Heidelberg.

The author was in a position to fill the gap by the clinical observation and bacteriological investigation of two cases of erysipelas, with the result that the question of the nature of the erysipelas coccus was decided in the sense of its non-specificity.

Other facts recorded in literature and quoted by the author, taken in conjunction with the experiences gained in these two cases, enabled him to draw the two following conclusions :

1. Erysipelas ætiologically considered is not a specific disease ; usually it is caused by the *Streptococcus pyogenes*, but it may also be produced by the *Staphylococcus pyogenes aureus*.

2. The micro-organisms causing erysipelas most probably in every case enter into the circulation ; pyæmia following erysipelas is therefore of a primary character, and not due to a mixed infection.

These general conclusions, I need not say, go in exactly the same direction as the propositions of the present paper, and lend, I think, in themselves considerable force to my views on the special subject now under consideration.

But a much more powerful ally of these views has arisen lately in a second paper by Dr. Jordan, in which this author from the broadest conceivable basis dis-

¹ Langenbeck's 'Archiv für klinische Chirurgie,' vol. xlii, 1891, pp. 325 *et seq.* A *résumé* of this paper has been given by Dr. Jordan himself in the 'Centralblatt für Chirurgie,' vol. xviii, 1891, Appendix, pp. 77 and 78, and my description of his conclusions is a *verbatim* translation of this report.

cusses the nature of septic infections in general.¹ So important, indeed, is this paper with regard to the point I wish to prove, that I must be allowed to give a short *résumé* of those parts of its introduction in which the author in the most lucid and convincing manner summarises the present bacteriological views on the nature of inflammations in general, on the ætiology of suppuration, on bacteria as causes of purulent inflammation, on the mode of action of pyogenic cocci, and on the influence of the nature and condition of the tissues in which the inflammation occurs.

Dr. Jordan follows Cohnheim's teaching with regard to the pathology of inflammation, in starting from the thesis that the "inflammatory irritation" ("Entzündungsreiz") primarily attacks the blood-vessels, causes them to become dilated and produces an alteration of their walls, as a consequence of which augmented transudation and increased permeability for leucocytes occur; the emigration of the latter (diapedesis) is the cardinal symptom. The *intensity* of the inflammatory irritation is of importance in determining the character of the resulting exudation; hence we have to distinguish between serous, fibrinous or croupous, purulent and hæmorrhagic inflammations. From an analysis of experimental investigation concerning the question of suppuration, Jordan then comes—amongst others—to the following conclusions:

1. *No specific pus microbes exist.* Apart from the Staphylococci and Streptococci a large number of other bacteria exist which under certain circumstances can produce a purulent inflammation.

2. *The pyogenic cocci proper are capable of producing apart from purulent inflammation all other forms of inflammation.*

These points are now proven in detail; the author

¹ "Die acute Osteomyelitis und ihr Verhältniss zu den pyogenen Infectionen auf Grund klinisch-bacteriologischer Beobachtungen sowie des jetzigen Zustandes der Bacteriologie," 'Beiträge zur klinischen Chirurgie,' vol. x, Tübingen, 1893.

shows by quotations from the bacteriological work of the most prominent bacteriologists within the last decade that: (1) the *Staphylococcus aureus albus* and *citreus*, (2) the *Streptococcus pyogenes*, (3) the *Staphylococcus citreus albus* and *flavus*, (4) the *Micrococcus pyogenes tenuis*, (5) the *Micrococcus tetragenus*, (6) the *pneumococcus* (Fraenkel und Weichselbaum), (7) the *Bacillus pyogenes foetidus*, (8) the *Bacillus typhosus*, (9) the *Bacterium coli commune*, (10) the *Bacillus pyocyaneus*, can each of them alone produce a *purulent* inflammation. In special connection with the subject here under consideration his statement that nowadays the *identity* of the *Streptococcus pyogenes* with the *Streptococcus erysipelatosus* is *fully acknowledged* by the most prominent bacteriologists (Baumgarten, Fraenkel, Cornil, Babes, and others) is of special importance.

The second point, namely, that the action of the pyogenic cocci proper is not an *exclusively* pyogenic one, is then equally exhaustively proved. Jordan shows: (1) that the *Staphylococcus* may produce a purely *serous* inflammation: and (2) that the *Streptococcus* may produce the *most various* forms of inflammation, apart from the purulent one, namely, (a) serous, (b) fibrinous, and (c) necrotising inflammation.

In the following two paragraphs he discusses the mode of action of pyogenic cocci and their pathological dignity, and comes, from actually observed facts, to the conclusion that in a previously perfectly healthy organism, without any concomitant action of predisposing moments, small quantities of pyogenic cocci suffice to produce a purulent reaction of the tissues. This naturally leads to his discussing the conditions under which an infection can take place. He shows that the presence of pyogenic micro-organisms in the system does not lead with absolute necessity to the production of suppuration, and states that in order that the latter may occur two conditions are necessary, namely, first, that a certain *quantity* of germs be introduced, and, secondly, that they must possess a

certain *virulence*. Already Fehleisen had found that *small* doses of pyogenic microbes have no effect upon dogs and rabbits, whilst the injection of *large* doses leads to speedy death of the animals. Similar results were obtained by Watson Cheyne and Hermann. As to the question of virulence, this is a *variable* and not a constant quality ; it depends upon a number of different factors, particularly on the condition of *nutrition* and the *age* of the cultures of the micro-organisms.

Finally Jordan shows that the *nature* and the *condition* of the *tissues* invaded by pyogenic organisms may produce considerable differences in the nature of the resulting inflammation. The individual tissues show very different resistance to the invasion of various micro-organisms, and their resistance further varies if any alteration exists in them due to local or constitutional causes. Of particular interest in connection with the question here under consideration is the fact that, as Bujwid's experiments have shown, in cases of *diabetes* the power of resistance of the tissue elements becomes diminished. These experiments were made in order to investigate the cause of the frequency of abscesses and furuncles in diabetic persons, and throw an interesting side-light upon the fact that in several of my own cases sugar was found in the urine during the septic inflammation of the throat and neck, or that the disease itself occurred in persons previously diabetic.

It is obvious that these conclusions of Dr. Jordan's form the most complete and most satisfactory vindication of my position that I could possibly desire. Viewed in the light of his statements, my propositions, revolutionary as they may have appeared from the mere clinical point of view, become in fact a *simple clinical application of general bacteriological principles to a certain group of septic inflammations*. For if it be conceded—as it must be now, I think—that no pyogenic micro-organisms exist which have an exclusive and specific action in producing suppuration, and that various microbes are capable of producing, according to their virulence, their quantity, and the character of

the tissues in which they develop, *any* form of inflammation (*i. e.* serous, fibrinous, purulent, or hæmorrhagic), it is obvious that the *special* form of exudation met with in a given case can no longer be looked upon as the decisive criterion for the pathological classification of the individual case, and that a terminology based upon such non-existing differences must be given up as illogical and erroneous. This is what I have tried to establish in this paper from the purely clinical point of view, and it is most gratifying that the results of an independent clinical study should so entirely agree with the outcome of the accumulated bacteriological teaching of the present day.

I am of course fully aware that the final proof of the correctness of my views must still be adduced. Only when it has been shown that in the slightest as well as in the most severe forms of septic inflammations of the throat and neck pathogenic organisms are met with, cultures of which will in one case produce a serous, in another case a purulent inflammation, will the question have been ultimately solved. I am, however, fully convinced that this can only be a question of time; and I hope that I have succeeded in making it at any rate extremely probable that the various forms of septic inflammations of the throat and neck are in reality pathologically identical.

Should a general agreement to that effect be obtained, the next step will of course be a sweeping revision of our present nomenclature concerning œdematous affections of the larynx. The chapter "Œdema of the Larynx," commonly met with nowadays in text-books of laryngology either ought to disappear completely, as a serous transudation into the tissues of that organ is a symptom caused by the most heterogeneous affections, or at any rate it should be limited according to Kuttner's proposal to the *passive* forms of œdema, such as are met with in Bright's disease. On the other hand, no separate chapters ought to be accorded to "Inflammatory Œdema," "Erysipelas,"

and "Phlegmon of the Pharynx or Larynx," and "Angina Ludovici"; and the conditions hitherto described under these names should be summarised as different stages of one and the same pathological process under the one general heading, "Acute Septic Inflammations of the Throat and Neck."

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 133.)