CHAPTER III.


BY

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Introduction.

From a clinical point of view the influenza epidemic of 1918-19 resolves itself into two sharply distinguished parts, the first including the type of cases met with in June 1918; and the second, the quite different type, which abounded in the autumn (October) of 1918 and recrudesced in the spring (February) of 1919. In the June 1918 outbreak, although the cases were very numerous there was little or no mortality; there was little or no pulmonary complication; and the patients, though striken severely for the time, speedily recovered after so short an attack that it was widely spoken of as "influenza of the three-day fever type." It was in the October 1918 and in the February 1919 outbreaks that the high mortality from pulmonary and general septicæmic complications developed and gave such an entirely different clinical aspect to the disease; and it was in this part of the epidemic that the dreaded heliotrope cyanosis was so pronounced a feature of the fatal cases.

It is of importance, however, to emphasize the fact that the heliotrope cyanosed type of case, though it attracted chief notice during the influenza epidemic of 1918-19, was not a new phenomenon confined to this epidemic; it had already been met with and reported upon in connection with minor epidemics during 1916 and 1917, at which time the label given to these fatal cases was "purulent bronchitis," though I, personally, am strongly of the opinion that these smaller outbreaks in 1916 and 1917 with heliotrope cyanosis as a striking phenomenon of the worst cases, were of precisely the same nature as those of the enormously more widespread and serious "influenza" outbreaks of 1918 and 1919. (See below, p. 69.)

It is not possible to give exact figures as to the number of cases I myself saw. In the epidemic of June 1918 I had the opportunity to study some hundreds; but in the epidemic of the autumn of 1918 and the spring of 1919 the total number extended to many thousands; part in Guy's and other civilian
hospitals; part in military hospitals in the Aldershot Command; amongst Canadian Troops at Bramshott and Witley Camps; and amongst United States troops on board ship and in the hospitals at Liverpool and in the hospitals at Winchester and at Portsmouth. I also performed, or was present at, autopsies totalling over three figures.

A very striking point throughout was that wherever one met with the disease, and of whatever nationality the patient, the malady in each mainpart of the epidemic ran remarkably true to type; except for the rarer complications the clinical type of case, whether of the mild "three-day fever" type of June 1918, or of the much more serious type of October 1918 and February 1919, was much the same wherever one met with it. It will be convenient to deal first, and briefly, with the mild June type; and secondly and more fully with the graver autumn type.

The Clinical features of the relatively mild June 1918 type of Case.

In the midst of perfect health in a circumscribed community, such as a barracks, or a school, the first case of influenza would occur, and then within the next few hours or days a large proportion—and occasionally even every single individual of that community—would be striken down with the same type of febrile illness, the rate of spread from one to another being remarkable. The patient would be seized rapidly, or almost suddenly, with a sense of such prostration as to be utterly unable to carry on with what he might be doing; from sheer lassitude he would be obliged to lie down where he was, or crawl with difficulty back to bed so that barrack rooms which the day before had been full of bustle and life, would now be converted wholesale into one great sick room, the number of sick developing so rapidly that the hospitals were, within a day or two, so overfull that fresh admissions were impossible and the remainder of the sick had to be nursed and treated where they were.

The men's temperatures were raised to varying heights, generally about 103° or 104° F.; the pulse rates were less raised in proportion; the tongue was coated; the face flushed; the eyelids a little drooped as though the patient were but half awake; and in a very considerable proportion of these cases, either at the very beginning or within a few hours of the onset, there was huskiness of the voice and a tendency to hawking and throat-clearing, less often to actual cough. The throat was complained of as being sore so that it was difficult to swallow or to speak; frothy expectoration, not large in amount, was brought up rather from the mouth and pharynx and larynx than from the bronchial tubes. There was a varying degree of bright reddening of the posterior part of the palate, uvula,
fauces and pharynx without white spots or folliculi of pus; without exudation and often without any swelling of the tonsils or cervical glands. In some, on the contrary, the tonsils, besides being reddened looked swollen and enlarged and there might be tenderness on either side of the upper part of the neck below and behind the angle of the jaw, suggesting that the lymphatic glands here were inflamed, too, though palpable glandular enlargement was not as a rule a feature of these cases.

Headache, especially of that type in which the head does not ache so much when it is kept still, but aches badly when there is a change of posture such as when the head is rolled from side to side, or some effort of coughing is made, was prevalent in many cases; but in the main one would summarise the symptoms as being those of lassitude and general all-over aching, with fever, a coated tongue, loss of appetite, soreness of the throat, huskiness of the voice and headache.

Most patients slept well and wished simply to be left alone.

Gastro-intestinal symptoms were not pronounced; there was no dyspnoea; water and cooling drinks were all the patient asked for, and in this state he lay in bed the next day and the day following, during both of which the temperature would as a rule remain up, though tending to fall upon the third day. The pyrexia might continue longer than this, but in many it was already coming down to normal at the end of the second day, and most of it had become normal at the end of the third day and remained so thereafter. The patient by this time was feeling almost himself again; asking for food, wishing to get up, and complaining of little more than some remains of soreness of the throat and perhaps some huskiness of voice, though the latter was better than it had been during the preceding three days. Convalescence was rapid and the great majority of the patients were fit for their ordinary work again by the end of the week. Only in those who seemed already predisposed to bronchial catarrh as a result of being subject to emphysema, for example, or chronic bronchitis, tended to have any persistence of the bronchial infection.

Amongst those previously robust there was practically no mortality, though individuals taken by the influenza when already ill with something else may have had their end hastened by the intercurrent influenzal attack.

There was no albuminuria, no special tendency to infection of the accessory nasal sinuses; indeed no tendency to any particular complication at all. Hundreds of cases ran very much the same course simultaneously, one very much like another, and "three-day" influenza was the popular name generally given to the disease.

In regard to treatment, nothing special was indicated; rest in bed for three days and ordinary nursing without drugs led
to just as good results as active administration of medicines, such as aspirin, salicylates, quinine, or anything else.

The Clinical Features of the Gases in the severe Epidemic of the Autumn of 1918 and of the Spring of 1919.

Contrasted with the extensive and acute but non-fatal outbreak of June 1918, the world-wide "plague" of influenza of the following autumn and winter, with its millions of deaths, presented very different clinical characters; and in the multitude of severe cases one saw the constantly repeated picture of a dreadful malady which few physicians had seen the like before. Those who had experienced the minor epidemics of "purulent bronchitis with heliotrope cyanosis and fatal ending" that had occurred here and there in military camps in America, England and France during 1916 and 1917 had already become familiar with some of the worst features, especially the dreaded blueness, of what was probably the same malady under a different name; but now it was a question of seeing hundreds or even thousands of cases in districts in which the fatal "purulent bronchitis" had affected but a few.

Nevertheless, it is important to emphasize the fact that, although it was the "pneumonic" type of case that attracted so much attention, creating such consternation owing to its mortality, and thereby colouring the picture of the epidemic as a whole, these fatal "pneumonic" cases constituted but a minority of the whole. There were far more cases of ordinary straight-forward benign influenza than there were of "influenza-pneumonia"; but these benign cases were overshadowed by the grave ones; and there is a little danger, if one does not emphasize the fact, that future generations might gain the impression that the whole of the 1918-19 epidemic was of "pneumonic" and grave character. Broadly speaking, I should say that out of 1,000 individuals struck by the disease fully 800 had no more than an ordinary attack of uncomplicated "influenza," a little more severe perhaps than the "three-day fever" of June 1918, but not any worse than simple influenza as it may occur at any other time. It was the remaining 200 who were so much more seriously ill, with "pneumonic" symptoms added to those of simple influenza; and of these about 80 died. The most dreaded symptom was the heliotrope cyanosis; it developed in less than half of the pulmonary cases, but once it became definite the prognosis was so bad that I should say out of every 100 "blue" cases 95 died.

It is not within my province to discuss the bacteriology of the disease, nor to enter into the question of whether the disease was really influenza at all, or something else. A separate chapter deals with these points. I have, however, been present at many discussions of them, both public and private, and my personal conclusion is that the primary nature of the whole epidemic has been "influenza"; that Pfeiffer's bacillus has
not yet been deposed from its place as the causal organism of "influenza"; that it has in all probability been the primary organism in the 1918-19 epidemics; but that whereas Pfeiffer's bacillus alone may be responsible for the symptoms in the 800 mild cases, an additional organism—sometimes the pneumococcus, sometimes the streptococcus, sometimes Friedlander's pneumobacillus, sometimes a combine of two or more of these, and sometimes, perhaps, yet other organisms—has become virulently associated with Pfeiffer's bacillus in the 200 graver cases; the pulmonary and other complications and the high mortality from septico-toxamia being the result of the doubled or trebled infection and not of influenza solely.

Expressed in another way, too briefly no doubt to be strictly accurate, and yet helpful in interpreting the remarkable difference there was between the mild and the severe cases that were under observation simultaneously, I would describe the 800 as suffering from influenza only; the 200 as suffering from influenzo-pneumococcal or influenzo-streptococcal or influenzo-pneumo-streptococcal septicotoxæmia and not from influenza only.

It was, however, only when any given patient had absolutely recovered that one could relegate him with certainty to the "mild" category. Even the mildest case had to be regarded as potentially grave; no matter how benign the illness might appear to be at first the dreaded pulmonary complications and cyanosis might set in without any notice at all. A patient might have been ill a day or two with mild influenza and seem to be progressing well; in an hour or two the whole picture might change, and twenty-four hours later the patient might be dead. During the epidemic itself, therefore, every case had to be regarded as in grave danger; it is only when one looks back that the two big classes—800 out of every 1,000, mild and ordinary; 200 out of every 1,000 severe, pulmonary, grave—emerge clearly into view.

There is little need to enter into the clinical details of the milder cases; they were ordinary influenza cases running an ordinary influenza course. It is the "pneumonic" type of case that calls for a detailed account, and in what follows it is the "pneumonic" type that is being discussed. One point, however, calls for special mention, and that is that whether the case developed into the grave type, or remained benign, *epistaxis* was a phenomenally common early symptom. It is not possible to give statistics, for in the stress and dire overstrain of those strenuous days and nights no fall records were kept; but when special inquiry was made in scores of consecutive cases, some degree of nose-bleeding had occurred in over half; in most it had been but blood enough to redder a handkerchief, but in some it had been sufficiently severe to call for treatment for the nose-bleeding itself; and all the medical men one met were commenting on the commonness of this epistaxis. It is referred to again later.
The Special Features of the "Pneumonic" Gases.

The first point to emphasize is that although the pulmonary complications were spoken of almost universally as "pneumonia," the one thing they hardly ever were was ordinary croupous lobar pneumonia in the recognised sense of the term. The word "pneumonia," therefore, has been used in this article in inverted commas, because one needs some word to use for the pulmonary complications, and "pneumonia" is the term that was applied almost universally; it would be quite erroneous, however, to suppose that these cases were ordinary influenza plus croupous lobar pneumonia. The physical signs were most variable; but the occurrence of dullness, bronchial breathing, bronchophony, pectoriloquy, and crackling rales over the greater part or whole of one lobe was frequent enough to make those who saw no autopsies believe that there was real lobar pneumonia present; and many made the diagnosis of lobar pneumonia on the physical signs, and used the term in discussion as though they thought real croupous pneumonia was present. Only in the most exceptional cases was croupous lobar pneumonia found post-mortem; the "pneumonia" of these influenza cases was almost everything else but lobar pneumonia, unless lobar "pneumonia" is held to include any variety of lung inflammation that can consolidate a whole lobe. In more cases than not there was no really "lobar" consolidation, and some died with symptoms precisely similar to the others and yet without any consolidation of the lung at all. The "pneumonia" was an acute infective pulmonary inflammation in which such consolidation as resulted was due not to croupous lobar pneumonia of the classical sort, but to a conglomeration of changes which included bronchitis and peribronchitis, coagulative oedema, hæmorrhage, collapse, broncho-pneumonia, abscess formation, and compression by pleuritic effusion, totally different to anything ordinarily seen in the post-mortem room. Hence, in speaking of these cases as "pneumonic," it must be emphasized that the pulmonary inflammations implied were those peculiar to the epidemic, and not just croupous lobar pneumonia complicating influenza.

The "pneumonic" complication—often with physical signs of but a little bronchitis when more were anticipated—would develop at any period of the influenza attack; there was no rule. In most cases the patient had been ill for a day or two with ordinary simple influenza, not necessarily severer than that of his neighbours, when there was a rapid or sudden change for the worse, and the picture changed rapidly from that of influenza to that of a chest case; and the effects of the pulmonary changes were often so fulminating that death might ensue in 24, 36, or 48 hours, in such a way as to suggest that it was not due to the lung lesions themselves but rather to a generalised and very virulent microbic-toxæmia, or actual septicæmia.
On the other hand, there was often no preliminary "influenzal" period at all, the patient being attacked from the start in such a way that ordinary lobar pneumonia of virulent or even ultra-virulent type would be the diagnosis made by most of us if one saw the case singly and not in such an epidemic; at the beginning of the outbreak it was most difficult to persuade those who had not yet seen other cases that the condition was not ordinary pneumonia; but the autopsies showed just the same conditions as did the other types of cases, and seldom, if ever, true lobar pneumonia.

Again, the pulmonary complications were often later in their development, yet equally fatal. The patient might have had no symptoms other than those of ordinary influenza for nearly a week; his temperature might be falling steadily, or might have become normal, so that danger might be regarded as past, and yet the "pneumonic" complications might set in and carry off a man who seemed almost convalescent.

Less often, and yet not infrequently, the patient might be apparently quite convalescent from "influenza," ready to be discharged from hospital, and yet go down with "pneumonic" symptoms, and die.

All types were seen in abundance—the initial, the early, the later, and the latest.

At whatever stage the pulmonary complications set in the patient generally began to complain in some way of his chest, often of pain in one side or other of the thorax, or on both sides; frequently in front, in one or other anterior axillary line below the level of the nipple; almost as frequently at one or other base; or, again, down the front of the chest behind the sternum as though he were "all raw inside there." In practically every case there was also cough, not always severe, but sometimes in itself distressing, short, dry, and hacking to begin with; looser and associated with frothy or blood-stained or purulent sputum within a few hours, or the next day. Towards the end of a severe case coughing and expectoration would be entirely absent from sheer weakness of the sufferer and his inability to cough at all. The rate of breathing became accelerated out of all proportion to the physical signs; in the worst cases the respiration rate would rise to 40, 50, or even 60 to the minute, and yet without any particular evidence of respiratory distress; orthopnea was exceptional and although the patients were breathing so rapidly they seldom, if ever, complained of actual difficulty in breathing; it was rather a frequent breathing—a polypnoea or tachypnoea—than a true dyspnoea. The condition of the skin was not constant; it might be hot, dry, and pungent as in ordinary lobar pneumonia; quite as often the whole of the patient's body and limbs would be covered with profuse perspiration, the latter often resulting in sudamina and miliaria. A definite rigor might occur at the onset of the pulmonary complication, but more often there was
nothing in the way of a definite rigor to attract notice, though the temperature, already raised, might rise higher.

The pulse rate, though raised, was seldom unduly rapid, and it was a remarkable feature of a great majority of the cases that the condition of the pulse remained good almost to the last, falling only in articulo mortis.

The Heliotrope Cyanosis.

The facies, at first flushed and red, with a peculiar drooping of the eyelids giving a weary look, shown in Plate 1, might remain purely red throughout, but in nearly half the cases affected by the pulmonary complications the red tint rapidly changed to one of progressive cyanosis, depicted in Plates 2 and 3. It was] when this dreaded heliotrope cyanosis appeared that one knew that the prognosis had now altered so completely that the patient was almost certain to die; a small percentage of cases managed to recover, even after the cyanosis had developed, but the great majority succumbed, and it was amongst cases of this type that the great mortality of the epidemic occurred. There were, of course, cases which died without the cyanosis being pronounced, but in going round a large ward one could, without examining the patients at all beyond looking at their countenances, pick out those who were going to die with almost uniform certainty by reason of their colour alone. It was not by the temperature chart nor by the physical signs in the chest, nor by feeling the pulse that one could tell the serious cases so well as one could by their colour; the cyanotic tint might be definite in a patient who was complaining little, who was taking his liquid nourishment well, was taking an intelligent interest in his surroundings, answering questions promptly and clearly, and in no way—except by his colour—indicating that by the next day or the day after he would almost certainly be dead.

The plates reproduced here were taken from rather extreme cases and very often the degree of fatal heliotrope cyanosis fell a long way short of that depicted; but the illustrations were taken from actual cases, and there were hundreds as severe as these. Whatever the degree of cyanosis, however, it rendered the prognosis bad. It depended much upon what the original colour of the patient had been, what the amount of actual blueness of the face was; for example, a man who was naturally sallow and pale-faced would show little of the heliotrope colour in his face generally, but it would be obvious in the colour of his lips and ears. The naturally pale man tended to look rather ashen than heliotrope, but with a distinctive colour of the lips and ears that attracted at once. The plethoric man, on the other hand, would be seen to change from a frankly red countenance to a colour which, if one were to reproduce it in
painting would necessitate the admixture of more and more blue with the red until ultimately the whole face and particularly the lips and ears passed through the stages of dark red to purplish-red, to reddish-purple, to absolute purple; and then towards the very end—as shown in Plate 3—that which had been a purple face might become a pale, cyanosed, deathly countenance with purple lividity of lips and ears.

In some the cyanosis might be well marked before the patient had been ill 24 hours, and death occurred in some instances within this time from the onset.

In others, the duration might be 48 hours. In others again, the lividity came on more gradually, and the patient might remain alive for three, four, or five days, or even for a week, breathing 50 or 60 to the minute, not unconscious, not subjectively distressed, though objectively a dreadful picture; but the ending in over 90 per cent, of all the cases in which the cyanosis developed was progressively downhill towards death; the latter being preceded in many instances by delirium of a low type, associated with unconsciousness, though in some on the other hand consciousness was retained almost, if not quite, to the very last.

I have seen many cases of precisely the same type of heliotropic cyanosis as the result of what was then called "purulent bronchitis" (see *Lancet*, July 14th, 1917, p. 41), and my impression is that, though there were certain clinical differences, especially in regard to the character of the sputum, the "purulent bronchitis" cases of 1916 and 1917 were really sporadic instances of precisely the same malady as those of the epidemic of 1918-19; and it is of interest that when Dr. Eyre helped Major Abrahams and me by determining the bacteriology of these previous "purulent bronchitis" cases, he found that they also had a double infection—influenzo-pneumococcal in some places, influenza-streptococcal in others. The only other Condition in which I have seen similar facies with cyanosis has been "gassing"; but in gassed cases the patient has been in dire distress as well, whereas the influenzal "pneumonic" cases were in much less distress than were those who saw them.

For a long time the nature and causation of this peculiar heliotropic cyanosis was obscure. It was certainly not due to cardiac or circulatoriy failure, for the condition of the heart and pulse remained strikingly good, and although some observers have noted dilatation of the heart at autopsy in these cases, cardiac dilatation was, in my experience, quite the exception; again and again at the post-mortem examinations one was struck by the fact that the heart looked remarkably normal both in colour and in size; there may have been a little dilatation of the right side, sometimes, but certainly no more than is general in cases of death from acute conditions, and anything like extreme dilatation of even the right side of the heart was very exceptional. The
PLATE 1.—This illustrates an early case in which the facial colour is frankly red, and the patient might not appear ill were it not for the drooping of the upper eye-lids, giving a half-closed appearance to the eyes.
PLATE 2.—This illustrates a pronounced degree of the "heliotrope cyanosis." The patient is not in physical distress, but the prognosis is almost hopeless.
PLATE 3.—This illustrates another type of the cyanosis, in which the colour of the lips and ears arrests attention in contrast to the relative pallor of the face. The patient may yet live for twelve hours or more.
cyanosis was not relieved in the least by venesection, or by the administration of digitalis or other cardiac stimulants. At one time it was thought that there might be some peculiar chemical change in the blood leading to the formation of methaemoglobin, or even sulph-hæmoglobin, but repeated spectroscopic examination showed no abnormal blood pigment to be present. When, however, one had the opportunity of examining microscopical sections of the lungs, in which coagulative exudation both into the alveoli and into the interstitial tissues was often a very pronounced feature of the section, one realised that this albuminous exudate—quite different to that seen in ordinary pneumonic cases—was the probable cause of the cyanosis. The appearances in some lung sections were very similar to those of the extreme exudate that results from gassing, and layers of this albuminous fluid coming between the inspired air and the blood capillaries would necessarily interfere with the absorption of oxygen by the latter, an extreme degree of anoxhæmia being the result. The general conclusion was that the heliotrope cyanosis was due not to heart failure, nor to abnormal chemical changes in the blood, but to sheer anoxhæmia resulting from this widespread and extensive albuminous exudate into the alveoli and interstitial tissues of the lungs.

The Temperature Charts.

Short of reproducing many scores of actual temperature charts it would be impossible to show how variable the course of the pyrexia was in different cases otherwise of the same type. Facing page 76, ten charts from simultaneous cases are reproduced, five from cases which recovered and five from cases which died, and their variability speaks for itself. Sometimes the temperature dropped rapidly with speedy recovery, as in Chart 1, but Chart 6 shows how a patient may have little pyrexia, and it may seem to be falling comfortably by lysis, and yet the patient may die. Chart 2 shows termination of the illness by crisis with recovery. Chart 7, a similar sudden fall of the temperature followed by death. Chart 3, a fall by lysis, prolonged by irregular persistence for several days, ending in recovery. Chart 9, a fall by lysis in a severe case that seemed to be doing well, with subsequent rapid rise and death. Chart 10 shows a rapid fall on the third day of the disease as though the patient had terminated his illness by crisis, but the pyrexia flared up again, rising by steps to a second maximum about the 10th day, when a second apparent crisis occurred, and yet the illness continued, pyrexia recurring after the second apparent crisis, and followed by a rise after the second crisis and terminating in death. No two charts were quite alike; definite termination by crisis and recovery, as is shown in Chart 2 was very rare indeed; the chief thing which struck one on studying many hundreds of consecutive charts was that
there was nothing in the temperature chart itself to tell one whether the patient was doing well or badly.

The Respiration Rate.

The respiration rate, on the other hand, was a much more helpful guide than was the pyrexia; the rapidity of breathing in the fatal cases was even greater than that which one expects in ordinary lobar pneumonia; rates that were nearer 40 than 30 to the minute were very common, but in the worst cases the rapidity of breathing was generally over 40 and often 50 or even 60 to the minute, and this sometimes before the fatal cyanosis became evident. It was in almost all cases silent respiration without stridor and phenomenally without distress; though breathing so fast the patients themselves were seldom conscious of panting for breath; they did not complain of their breathing. They might complain of pains in their chest, or of cough, or of chest trouble, but they did not—at any rate in the great majority of cases—complain that they had difficulty in breathing or that they could not get their breath. They very seldom had orthopnea; most cases indeed lay flat down in bed and preferred not to be propped up. It was exceptional to find actual orthopnea, and then only as a rule when there was pre-existent emphysema or myocardial degeneration or valvular disease. This absence of real dyspnœa was remarked on repeatedly by observers from all parts, and led to the use of the terms polypnoea or tachypnoea, to distinguish the condition exhibited by these patients from real dyspnœa.

Cough.

Cough was not always a prominent feature, and often it was almost absent throughout even in those most gravely affected. It bore little relation to the extent of the physical signs in the lungs, but was most troublesome when there was evidence of much bronchitis, or again in the many patients who had a pleuritic friction rub. It was apt to occur in paroxysms which were sometimes very exhausting when there was little sputum. Towards the end of the grave cases cough, which might have been very troublesome indeed in the earlier stages, generally ceased, even though the lungs were full of rhonchi and rales from apex to base; probably because the patient had too little reserve of strength for the effort of coughing to be possible.

Sputum.

The sputum was very variable indeed. Cases in which large quantities—8 or 10 ounces—of almost pure pus were coughed up daily in the way which was so remarkable in the previous cases of so-called "purulent bronchitis" were met with here and there, but these were very exceptional indeed during the main epidemic.
CHARTS OF 5 CASES OF RECOVERY.
(Chapter 3)

Name: B Age: 40 Disease: INFLUENZA with PNEUMONIC SEPTICEMIA Result: DEATH

Name: CADET C Age: 21 Disease: INFLUENZA-PNEUMONIC SEPTICEMIA Result: DEATH

Name: SERGT L.M. Age: 32 Disease: INFLUENZA-PNEUMONIC SEPTICEMIA Result: DEATH

Name: PTE M Age: 37 Disease: INFLUENZA-PNEUMONIC SEPTICEMIA Result: DEATH

Name: PTE R Age: 19 Disease: INFLUENZA-PNEUMONIC SYNDROME Result: DEATH

CHARTS OF 5 FATAL CASES.
Some cases had hardly any sputum at all. Frothy mucoid or muco-purulent phlegm was much more common; it had no particular odour. Sometimes there was nothing but pure froth; again there might be froth only with some bouts of coughing; pellets or dollops of muco-pus at other times in the same patient; the individual expectorations remaining separated from one another in the disinfectant in the sputum jar. Or again, the sputum might be glairy and mucoid or stringy; or it might be simply purulent, the successive expectorations running together in the receptacle like pure pus. The colour was equally variable; sometimes the sputum was all white froth; or the froth might be tinged with blood streaks; or dollops of pure clotted blood might be coughed up separately from the froth; or there might be liquid red blood expectorated in a way recalling a moderate haemoptysis from phthisis; or the more purulent type of sputum might be tinged red or brown with altered blood, or it might be definitely glairy rusty sputum like that of ordinary lobar pneumonia; the same patient might bring up different types of sputum on successive days, and in the same ward successive patients, suffering apparently in a similar way, might be bringing up each a different type of sputum, so that no conclusion could be drawn from the appearances of the latter as to what degree of the malady the patient had.

Occasionally the colour attracted notice; instead of being white or very pale yellow, red, or brown, it was bright orange; or in other cases pale green, or greenish yellow, or bluish yellow, or greenish blue, or pale blue; apparently these peculiarities in colour were due to variations in the pigments produced by the micro-organisms present.

The Physical Signs.

The physical signs varied widely in different cases of similar type. As a general rule what struck one most was the frequent paucity of abnormal signs, when the degree of illness of the patient led one to expect many. Percussion and stethoscopic evidence of extensive consolidation was often conspicuous by its absence. Before one gained experience one thought the case was one in which this absence of the familiar signs of pneumonia was due to the consolidation being seated too deeply at the moment to be detected, and one expected dulness, bronchial breathing and pectoriloquy to manifest themselves the next day or the day after. In a small proportion of cases this proved to be so; and the unwary would regard the signs as indicating lobar pneumonia beyond doubt; but the autopsy findings would prove the lobar consolidation to be due to the conglomerate lesions mentioned previously, and not to true croupous pneumonia. In a very large number of cases, on the contrary, the classical signs of lobar consolidation never developed at all.
There might be a few scattered rhonchi over the front of the chest, and over the upper part of the lungs behind, changing from rhonchi to non-consonant rales as one passed down to the bases; but without dulness, bronchial breathing or pectoriloquy, even rales and rhonchi might be difficult to find in some cases. Again, there might be little more than deficient vesicular murmer at one or other base—the kind of condition leading one to say, "the consolidation is here and will show itself more definitely to-morrow"; and yet it did not. Again, amongst the scattered rhonchi and mucous rales one might come across a patch or two of rales that were almost or quite consonating, yet without bronchial breathing or pectoriloquy; such patches might persist, or develop further, but quite as often they became less obvious within an hour or two, or might disappear entirely. In not a few cases one might note definite bronchial breathing with crackling rales and pectoriloquy over a quite considerable area of a lower lobe at one time, and yet later on the same day the bronchial breathing and pectoriloquy might have disappeared entirely, giving place to ordinary vesicular murmur, rhonchi and non-consonant rales again; suggesting that there had been temporary collapse. On the other hand, especially in cases that survived several days, the areas, particularly in the lower lobes, in which crepitant rales could be heard, might increase until such rales would sometimes be audible all over both lower lobes, yet without bronchial breathing; the percussion note at the bases being impaired but not dull; or there might be definite dulness over one or both lower lobes with crepitant rales, bronchial breathing, bronchophony and pectoriloquy; or again basal dulness with absence of breath and voice sounds might be found on one or both sides, sometimes the result of massive collapse, sometimes of compression by pleuritic effusion. Whatever the other signs, pleural friction was to be heard in a large number of cases, though detection of the friction was often rendered difficult by the fact that patients tended to take rapid short shallow breaths, so that it was not always easy to get them to make an inspiration deep enough to render the friction audible. Such friction was most often to be found posteriorly or in the axilla, but it might occur anywhere from apex to base; the physical signs of consolidation, on the other hand, when they did develop at all, were almost always in the lower lobes, apical consolidation signs being remarkably uncommon, though met with in exceptional cases.

The extensiveness or otherwise of the physical signs, however, seemed to bear little relationship to the degree of illness of the patient; nor to his cyanosis. A man might be heliotrope blue with hardly any abnormal lung signs to be detected; or he might have signs suggesting consolidation of both lower lobes and not be blue at all. It was by the blueness and not by the physical signs that the prognosis could be measured best, and one saw quite a number of cases in which the patient went
the heliotrope colour and died without exhibiting any material departure from the normal as regards pulmonary physical signs.

**Herpes of the Face and Herpes of the Ears.**

Herpes facialis varied greatly in its incidence amongst different groups of cases. One might see several hundreds consecutively with none at all; and then come across a number with it. I should say that its occurrence was exceptional on the whole—less than 3 per cent.—and yet it might chance that one would occasionally find upwards of a quarter of the cases in a single ward presenting this symptom. When it did occur it was generally of just the same type as that seen in ordinary pneumonia, affecting the lip margins, chin, or alæ nasi; but in exceptional cases it was more like the type seen in spirochætosis icterohæmorrhagica, the massed vesicles and their inflamed bases extending all over the chin and lips and nostrils, out on to both cheeks, and down on to the neck, becoming hemorrhagic and covered with bloody scabs

The occurrence of herpes facialis, even of this exceptionally severe type, did not imply that the patient would not recover.

Several striking instances of bilateral herpes of the primæ were met with, both with or without herpes of the lips. The grouped vesicles developed on all parts of the prima, more often on the external surface and free margin than on the posterior surface, incommoding the patient mainly by rendering it painful for him to lie with either ear turned down to the pillow, but otherwise not related, apparently, to any other special feature of his case.

**Skin Rashes and Lesions.**

Apart from the herpes of lips and ears, and from the development of sudamina or miliaria in perspiring cases, pronounced skin lesions were exceptional.

General flushing, especially of the face and neck, was common; but in a very few cases did this flushing reach a degree that could be called definite generalised erythema. Now and then one met with a case, reddened sufficiently to rouse a suspicion of scarlatina; but the erythema differed from that of scarlet fever in that it was not punctate, and it involved the face and neck as well as the trunk and limbs.

Morbiliform eruptions were very rare, and when met with there was always a question as to whether the patient had not really got measles or German measles as well as influenza; so many thousands of individuals were ill together that the coincidence of measles or almost any other illness with the influenza was almost sure to be met with; one could not say, therefore,
that the influenza itself was the cause of the few morbiliform rashes seen.

Generalised purpura was remarkably rare; in so toxic an infection one was on the look-out for it, and expected it; but it was very rare indeed, even in the worst cases; though I saw it in four, all fatal. Less uncommon was a localized purpuric eruption on the legs, below the knees, though even this was rare. Its local occurrence was not necessarily followed by death. In two cases, after a shower of purpuric spots upon the feet and ankles, hämorrhagic bullae developed upon the sites of the original small petechias, associated with extensive oedema of the toes, feet, and ankles, great pain in the feet, and tenderness; the bullae subsequently ulcerated, and the patients, though surviving several days, ultimately died. This purpuric-vasculo-ulcerous state was bilateral and more or less symmetrical. I saw no others of this type, and I do not know the nature of this form of lesion, though I imagine it was vascular and, perhaps, the result either of multiple embolism or of multiple thrombosis of venules in the extremities.

Larger and smaller tender areas of erythema in various parts, not hämorrhagic, were complained of by many patients; as a rule they subsided spontaneously and they did not attract great notice; but looking back at them now it occurs to me that these may have been similar lesions of lesser degree.

\[ \text{Epistaxis, Haemoptysis, and other Haemorrhages.} \]

Epistaxis has been referred to already; it was strikingly common at the onset, usually slight, sometimes so severe as to last 24 hours or more. The fact that it was so common suggests that the naso-pharynx was an important focus from which the germs of the generalised malady were absorbed—a point debated later.

Haemoptysis was also common, though not so common as epistaxis. The latter occurred in the simple mild cases as much as in the severe; haemoptysis was a phenomenon of the "pneumonic" cases mainly. Occasionally, no doubt, it was due to the influenzal infection lighting up a latent tuberculous lesion; but quite apart from this, true haemoptysis, with the expectoration of bright red liquid blood in amounts varying from half a teaspoonful to several ounces was met with so often that it could not have been accidental, over and above which it occurred in cases in which subsequent autopsy showed no tuberculous focus to be present. Similar haemoptysis does occur in croupous pneumonia, but not with the frequency it did in this epidemic.

Apart from actual haemoptysis, blood-tinging of the sputum was common enough—as blood streaks; or general staining with bright red; or as a discolouration from altered blood—dull red, orange, rusty, brown, blackish-red, or almost tarry black; typical glairy rusty sputum was exceptional.
Hæmatemesis was observed many times; as a rule it was difficult to exclude the possibility of the blood from the stomach having been derived originally from the nose, swallowed and then vomited; but in a few cases it seemed almost certain that the blood was derived from the stomach itself—perhaps a diffuse oozing from an injected gastric mucosa. If blood was vomited at all it was generally of considerable amount—many ounces. There was no marked tendency to repetition of the hæmatemesis; the prognosis was not necessarily bad; no ulceration of the stomach was found in the fatal cases but acute congestion of the mucosa was common.

Bleeding per rectum was noticed in a few cases only. Dr. Lowe has recorded extensive ulceration of the colon in fatal influenza, so that the passage of blood per rectum is to be expected sometimes; but it was not commonly a symptom to attract notice. Whether occult blood was often present in the stools I cannot say; probably it was, if only because of the frequency of epistaxis and the liability of the nasal blood to be swallowed when the patient is recumbent. Pronounced melæna attributed to this cause was observed more than once.

Amongst women, a tendency to excessive uterine bleeding was exhibited by some when the menstruation came on during the attack; but there were many cases in which this tendency did not show itself at all; and although I have heard from others that spontaneous uterine bleeding occurred sometimes, unconnected with a menstrual period, this was not noticed in cases I saw myself.

Hæmaturia, sufficient to colour the urine with blood, was very exceptional. Nephritis was constant in the fatal cases, and albuminuria, suggesting nephritis was common in many that survived; red blood discs were sometimes found in the urine microscopically in such cases, but definite hæmaturia was rare.

I cannot say whether spontaneous retinal hæmorrhages occurred or not; there was little time or opportunity for retinoscopy during the epidemic and no obvious retinal changes were observed in the small number of cases thus examined.

Hemiplegia, followed by recovery, developed in one case during the acute influenzal attack apparently due to haemorrhage; there was insufficient evidence, however to prove that the influenzal attack was the sole and essential cause of the seizure.

Delirium and Coma.

Delirium and coma occurred often enough amongst the bad cases, but far more striking than their occurrence was their entire absence almost to the very end in so many instances. Big strong men, heliotrope blue and breathing 50 to the minute, obviously dying, would be fully conscious, talking rationally on almost any subject, relatively clear-headed to within half-an-hour of death; often not realising in the least how dire their condition was.
When delirium did occur it was seldom of the noisy, trembling, shouting-out type, though cases of this sort occurred too. More often it was of the low muttering restless type, the patient picking at the bed clothes and at objects round about him, with rambling talk or incoherence; getting out of bed, but readily getting back when asked to; rational for a few moments when spoken to, able to answer questions, and yet lapsing quickly into a restless semi-conscious state when left. Another type of case became totally unconscious hours or even days before the end, restless in his coma, with head thrown back, mouth half open, a ghastly sallow pallor of the cyanosed face, purple lips and ears—a dreadful sight.

It is noteworthy that the Cheyne Stokes type of breathing was very seldom met with even in those deeply comatose; perhaps because of the extreme anoxhæmia.

Subsultus Tendinum.

Subsultus tendinum was a marked phenomenon in many cases quite apart from delirium or coma. The patient might be rational enough to be talking about himself and he might even himself call attention to the way he could not keep his legs or arms or back muscles quiet when he wanted to. A leg or an arm or the abdominal muscles or the back would give an involuntary twitch or jerk; or the face or one shoulder, or some other part; the character of the condition approaching in a few instances almost to that of mild chorea. As a rule the prognosis proved bad, though the patient was by no means always in extremis when the symptom first developed.

Headache.

Headache was a pronounced symptom in nearly all the cases, the simple influenzal as well as the "pneumonic." Sometimes the whole head ached and throbbed; sometimes the head did not ache if the patient kept quite still, but swam and ached all over if it was turned quickly or if the patient sat up or coughed. Besides this generalised headache, however, and often in addition to it, there was complaint of special aching "at the back of the eyes"; or "inside the head in front," the patient generally putting his hand low down across the forehead to indicate its site. The more generalised headache was doubtless due to the toxæmic state, thus corresponding with the aching limbs and back; but some light on the different nature of the ache "inside the head behind the eyes" is thrown by the autopsy findings in the sphenoidal and ethmoidal air cells. In 22 consecutive cases in which these were opened up they were found to be obviously infected in 21; and in over half of these the sphenoidal air cells were not merely inflamed, but they contained definite turbid fluid or actual opaque pus. It seems probable
that infection of these sinuses is common, and this may be the cause of the peculiar headache "right at the back of the eyes" that so many influenza patients complain of, either at the beginning of the illness, or during it, or even after the acute phase has passed off.

*Nephritis.*

In all the autopsies that I did or saw—between 100 and 200 in all—there was no instance in which the kidneys were not definitely inflamed; the common lesion being that which gives the swollen blood-oozing kidney. It would be unlikely that every case developing this acute nephritis should die; doubtless many recover, some completely, some with residual renal defects. Few cases, however, presented the ordinary clinical picture of acute nephritis for there was no oedema to attract notice. It is probably too much to say that no case of generalised nephritic oedema occurred at all; but I saw none, even when acute nephritis was demonstrated post-mortem. This raises the old difficulty of trying to decide whether, when albuminuria without oedema occurred in the patients who recovered, the condition was to be labelled merely "febrile albuminuria" or actual "nephritis." Albuminuria at some stage of the malady was common; all the cases that died seemed to have acute nephritis; the inference seems to be that at least some of the albuminuria cases that survived have some degree of actual nephritis also. One has not had the opportunity of tracing these cases since, so that one does not know what the condition of their urine may be now; but one feels that there is at least the possibility that some, regarded as cured, may come under notice again as chronic tubal nephritis cases when a few years have elapsed. If they do so the possible origin of their kidney disease may be unknown and they will then fall into the category of Rose Bradford kidney cases.

There being no oedema at the time of the acute disease, the urine has not always been tested; when practitioners were all so overwhelmed with cases that it was sometimes days before they could get to visit the patient at all, there was no time to test urines as a routine procedure, and how common, therefore, albuminuria was one cannot say; but it was certainly common in the severer type of cases. It would be thought perhaps that microscopical examination of the centrifugalized deposit for renal tube casts would have settled the questions of whether the albuminuria was "nephritic" or merely "febrile"; but this is not so, for even when acute nephritis was demonstrated post-mortem the urine during life had generally shown few casts, if any. Broken down renal cells with red blood discs were observed far oftener than casts; the lesion was so acute that the cast-forming stage had not been reached; hence, absence of tube casts will not exclude acute nephritis in the living, and
enable one to label the albuminuria merely "febrile." Personally, I have the feeling that acute nephritis was common in these cases; that it generally recovered quickly; but that in some cases it may have damaged the kidneys to an extent we do not yet know, the existence of nephritis during the influenzal illness having passed unnoticed because there was no oedema to attract attention.

Rarer Complications.

Acute Subcutaneous Emphysema of the Chest Wall.

Every now and then, perhaps once in 500 cases, one met with a very striking symptom, namely, spontaneous emphysema of the chest wall, with widespread and characteristic crackling beneath the palpitating hand. I saw 12 instances of this and all of them died, but I have heard of one or two which recovered. The patients were all of the severe type, but not necessarily in the cyanotic or hopeless stage when the accident happened. The escape of air into the subcutaneous tissues occurred over the front of the chest first in nearly all, spreading thence for variable distances until neck, shoulders, chest, back, and abdomen might all be crackling in a similar way, much as when the same accident results from a broken rib.

Sometimes the escape comes on immediately after a severe spasm of coughing, but it has also developed without obvious relationship to any coughing bout; the patient, for instance, may wake up to find himself crackling when he touched his chest.

The pathology of the condition is not infection by gas-producing organisms; it is due to escape of gas from within the lung through ulcerated apertures in the two layers of pleura, the ulceration in its turn being due, in my belief, to infection from minute abscesses in the underlying lung close beneath the visceral pleura. At autopsy one frequently finds half-a-dozen or more tiny abscesses, each the size of a pea or thereabouts, aggregated together in the central part of a haemorrhagic and consolidated portion of lung; the pale abscesses, small though they are, contrasting markedly with the dark red consolidated lung in which they have formed, the infection being so acute and the abscesses so small, one of them now and then leads to an ulcerative puncture through both layers of pleura, the effect of which is to cause, not a pneumothorax but subcutaneous emphysema, just as a fractured rib causes not a pneumothorax as a rule but an escape of air into the subcutaneous tissues. Were the abscesses not so small, pneumothorax would be more likely perhaps. I have met with no case of spontaneous pneumothorax in this epidemic.
Hæmorrhage into, or Spontaneous Rupture of, Rectus Abdominis Muscle.

Quite a number of cases of spontaneous rupture of one or both rectus abdominis muscles have been met with, and in a still larger number the rectus abdominis muscle, short of actually rupturing, has been found at autopsy to be in a hæmorrhagic necrotic state such as precedes rupture. Almost invariably this lesion has affected only that quadrant of the muscle which is below the level of the umbilicus, and I have not met with similar hæmorrhage into, or rupture of, any other muscle, though I imagine that others besides the rectus abdominis must be affected sometimes. Possibly coughing efforts or some other mechanical reason is responsible for the rectus abdominis being most often involved; apparently the disorder is the result of vascular changes within the muscle—venous thrombosis it is thought. The affected muscle has much the appearance of the breast of a bird that has been badly shot at close quarters, the muscle being soft, pulpy, and infiltrated with dark extravasated blood. The lesion is not always obvious during life because the whole of the muscle sector is involved at once; one does not get, therefore, the drawing apart of the two normal ends with the depression in the centre, which is the characteristic sign of rupture of a healthy muscle.

If the patient is not already too ill to complain he tells one of acute pain in the lower part of the abdomen, either central between umbilicus and pubes, or to one or other side of this. In one such case, when the lesion affected the right rectus abdominis muscle only the pain was so severe, localised and associated with unilateral muscular rigidity over the right iliac fossa that it simulated acute appendicitis, and it was only after urgent operation had been performed that the error was found out. The appendix was normal; the right rectus abdominis muscle was crimson black from hæmorrhagic necrosis. This patient recovered completely; had it not been for the operation I should have had no idea of the real nature of his attack of acute and persistent pain, and I feel that the complication may have been even commoner than the ten or more cases in which I saw it demonstrated led me to suppose.

Jaundice.

Jaundice was quite uncommon, and when it was met with it was generally confined to a small percentage of cases in one particular district, other districts providing none. Its degree and type were similar to those of ordinary catarrhal jaundice; there was pallor of the stools and darkening of the urine, and the impression one got was that the jaundice was simple catarrhal-obstructive and not hæmolytic-toxic jaundice; the patients might do very well in spite of their jaundice, and although one might have expected to meet with hæmolytic-toxic jaundice of
grave omen in at least some cases of so serious a microbiotic disease, I met with none personally.

Parotitis.

Unilateral or bilateral parotid inflammation was not uncommon; it was associated with pain and tenderness over the swollen gland, but generally no reddening; it was quite exceptional for abscess formation in the gland requiring incision and drainage, to supervene. At first it was thought that the non-suppurating cases were instances of mumps coinciding with influenza, but this view was altered when the parotitis was found in cases that had had mumps before, and when, in case after case, the swelling was confined to the parotid glands, very seldom spreading to the submaxillary and sublingual glands as mumps generally does. Apparently the parotitis was secondary to infection of the ducts from the mouth, corresponding to the similar parotitis that used to be common after abdominal operations before the toilet of the mouth was attended to more carefully than it is now. The parotitis was commoner in the severer cases than in the milder, so that many of the patients exhibiting the mumps-like facies died; but intrinsically it did not appear to be a grave sign.

Pericarditis and Peritonitis.

Some observers reported pericarditis as a complication, but I saw no case of this at autopsy, and observed no case of pericarditic friction during life. It was remarkable that this should be so, seeing how common acute pleurisy was, and how virulent the general infection; many cases having living microorganisms in their bloodstream. Peritonitis was equally rare. I saw no case with acute general pneumococcal or streptococcal peritonitis.

Panophthalmitis; Cancrum Oris; Noma.

Panophthalmitis was seen twice; cancrum oris and noma not at all. It speaks volumes for the general efficiency of the nursing of the cases that these dread results of uncleaned eyes and mouth were conspicuous by their absence.

Meningitis.

Acute meningitis occurred in a small but definite number of cases, its incidence being sufficient to attract special notice.
The cases in which it occurred may be classed under three headings, viz.:—

(a) Meningococcal cases, not influenzal at all, but mistaken at first for instances of the prevailing epidemic.

(b) Meningococcal cases, in which the primary illness was influenza, the meningococcal meningitis developing during the course of the influenza or before convalescence from the latter was complete.

(c) Acute meningitis, not meningococcal, but due to one or other of the infecting organisms of the influenzal attack—Pfeiffer's bacillus, diplostreptococci, pneumococci or streptococci being recovered from the cerebrospinal fluid.

It was always difficult at the time to be sure into which of the three groups to put the cases; and there may always be doubt as to whether groups (a) and (6) are distinguishable at all; but one's impression, based on some scores of cases, is summarised above. The diagnosis was arrived at by lumbar puncture and bacteriological examination of the cerebrospinal fluid. Most of the meningitic cases proved to be meningococcal and it was only by studying the history of each and the charts and the course of the disease that one could see whether the disease was meningococcal meningitis from the start or whether there had been influenza first, meningococcal meningitis supervening during its course. That some were not meningococcal meningitis from the start seemed certain, because sometimes the patient had had typical influenza, with a subsidence of pyrexia and symptoms, and a partial convalescence before the meningitic symptoms developed. It is interesting moreover, that—although actual statistics are not available—one met with a larger number of these meningococcal meningitic cases amongst a given number of the influenza cases than one would expect to find even under conditions of crowding amongst an equal number of healthy individuals. For instance, in one hospital of 600 influenza patients four meningococcal meningitis cases developed in one week; in all of these there seemed little doubt that their initial illness had been influenza; and their previous history showed no common source from which they could all have derived the meningococcal infection. I saw over 20 instances of the association in all. The impression one formed was that the influenzal infection opened up the path for absorption of meningococci from the posterior nares and rendered carriers of meningococci more liable than usual to develop actual meningitis. I quite realise, however, that this is only an impression, without logical proof.

Apart, however, from meningococcal cases there were a few instances of acute fatal meningitis in which no meningococci were found, and the organism recovered from the cerebrospinal fluid was either Pfeiffer's bacillus or one of the...
infective cocci associated with the latter in the patients' complex influenzal attack.

*Otitis Media; Ethmoidal and Sphenoidal Sinus Infection.*

Earache was a fairly common symptom, especially in younger patients; and when the epidemic swept through a boys' school, for example, there was considerable liability for one or more of the boys to develop acute otitis media followed by otorrhoea either during the acute influenzal attack itself or before recovery from it was complete. Amongst grown-up people the proportion of acute otitis media and otorrhoea cases was less, though temporary deafness of one or both ears, with pain suggesting temporary catarrh of the middle ear and mastoid cells, was not uncommon.

Infection of ethmoidal and sphenoidal air cells was rarely recognised during life; but that it must have been almost common seems probable from the post-mortem findings. These regions were not opened up in every fatal case, but they were in 22 consecutively, and in only one of these did the interior and lining membrane of the sphenoidal air cells look normal; in the remaining 21, the lining membrane was congested and red; in six there was definite opaque pale yellow pus filling the air spaces; and in the remainder there was turbid fluid which in every case yielded on culture the same micro-organisms as were recovered from the lungs.

The sphenoidal air cells were more constantly inflamed than the ethmoidal, to judge from the naked eye appearances; the frontal air spaces less often still; bacteriologically, infection was almost constant. This seems to have an important bearing on the nature of the headaches which accompany influenza and which trouble some patients long after the influenza itself is past.

*Infective Endocarditis.*

Infective endocarditis was met with in no case post-mortem, and during the epidemic at any rate was not observed during life, but it is noteworthy that in the months which succeeded the epidemic, and indeed throughout the year 1919, one was constantly meeting with cases of the chronic type of infective endocarditis, whose origin was obscure. In many of these cases there was an old-standing valvular lesion of the heart, but in some there was no known previous heart disease; so chronic were many of these cases that some survived for many months after the diagnosis became clear as the result of the changes in the cardiac bruits, the progressive anæmia, the multiple emboli and the development of a palpable spleen, all associated with more or less pyrexia of long continued type.
Whereas in ordinary times it is exceptional to have more than one case of infective endocarditis under one's care at Guy's Hospital, during 1919 one has had as many as five patients in the ward suffering from this disease simultaneously, and altogether I have seen over 70 cases in the course of twelve months, in many of which in addition to the typical clinical picture the diagnosis was confirmed by autopsy.

It would be a very difficult thing indeed to prove that the occurrence of so many cases of infective endocarditis in the year following the acute influenzal epidemic was really in any way due to that epidemic, but one began to meet with the cases in December 1918 and already by March and April 1919 one had become convinced that there was something remarkable about them and one wondered whether these chronic months-lasting infective endocarditis patients might not be, as it were, an aftermath of the virulent infections of the acute influenzal epidemic types. In none of these cases did one find influenza bacilli in the blood, a form of streptococcus being the usual micro-organism recovered. The patients had generally been "seedy" for weeks or months before they had to take to their beds and the diagnosis of infective endocarditis became clear; and one wondered whether the infecting micro-organism had not settled upon the heart valves at the time when there was the world-wide virulence of infecting micro-organisms which, taking in the main the form of an acute influenzo-streptococcal, or influenzo-pneumococcal illness, might in certain individuals have led to some focal infection, of which subacute or chronic infective endocarditis might have been one. One does not know whether there was any similar frequency of infective endocarditis after the virulent influenza outbreaks of 1889, 1890 and 1891, but it will be interesting to watch whether, after any future acute influenza epidemics, a similar frequency of infective endocarditis shows itself during the twelve months which succeed that future acute epidemic.

*Localised Pus Formation.*

Acute pleuritic effusions, generally small in amount and yielding turbid fluid rather than definite pus, were quite common in the "pneumonic" cases; this turbid fluid contained X3olymorphonuclear cells microscopically and yielded cultures of pneumococci, diplo-streptococci, or streptococci bacteriologically, but it was not very common for these effusions to develop into definite empyemata. Many such effusions, though containing turbid fluid, excess of leucocytes and micro-organisms resolved spontaneously; some were found post-mortem in the fatal cases, but a few became definite empyemata, and it was noted as a rather remarkable feature of such cases that once definite pus developed in the chest, the patient ultimately did well, no matter how ill he might appear to be at the time.
The same seemed true of other cases in which localised collections of pus developed elsewhere than in the chest. In two or three instances, for example, successive subcutaneous abscesses, pyæmic in type, had to be opened and drained with recurrence of fresh abscesses for several weeks, and yet, after a time of great anxiety, the patients made a complete recovery. In another type of case—quite exceptional—subcutaneous suppuration developed at the site of saline injection. Almost without exception any patient in whom any local abscess or empyema or similar focal suppuration occurred, ultimately did well. To such extent did this impress more than one observer that deliberate attempts to produce a spontaneous abscess, not by injecting extraneous organisms but by focalising the patients' own organisms, were made in the belief that if such "fixation" abscesses could be produced, the patients would do as well as those in whom spontaneous suppuration occurred. When, however, one tried to produce suppuration in this way by trauma, or by the subcutaneous injection of irritants such as turpentine, one did not once succeed in obtaining a local "fixation" abscess; and yet it is a point that merits special emphasis, that whenever spontaneous suppuration did develop in these influenzal "pneumonic" patients, one could almost invariably assume that the patient was going to pull through and get well, even though his actual state at the time might otherwise look precarious.

**Age and Sex Incidence.**

No age and neither sex was free from the danger of infection, and the "pneumonic" type of influenza case was met with in infants, children, boys, girls, young adults, full-grown people, and the aged. One's opportunities, however, brought one much more into touch with those of military age than with either the very young or the very old, and one formed the impression that the incidence of the disease—unlike that of 1890-91—was considerably greater in those between the ages of 20 and 50 than in those below this age period, in addition to which it was people of these ages who were mostly aggregated together in camps and barracks, juveniles and old people being spread wider apart and therefore less liable than the military to develop the disease by direct contagion when the first case in the neighbourhood fell sick. Apart, however, from the age incidence of the disease there was a very definite age incidence of the fatalities, and it was a striking feature of the epidemic that strong, healthy adults, especially those between 20 and 40, and more particularly between 25 and 35 years of age, were those who were most liable to succumb to the dreaded "pneumonic" type of the infection. Stricken with the same disease at the same time, and from the same source, with pneumonic complications, the chances of a man of 55 pulling through seemed
better than the chances of a man of 25 or 30. In this respect the epidemic of 1918-19 appeared to be entirely different to epidemics of the past in which, as a rule, the deaths have been largely among the elderly or the very young.

The Route of Infection.

It seems likely that the route of infection was not invariably the same, but one received a strong impression that the nasopharynx and the nasal passages were a highly important focus from which the spread of the offending micro-organisms took place in a very considerable number of the cases. There is no absolute proof of this view perhaps, but there are several highly suggestive points which lead one to this opinion and, if correct, the view is of considerable importance in connection with the need for nasopharyngeal cleansing or antiseptic "toilet" as a preventive measure. Some of the points which would seem to indicate that the infection is at first localised in the nasopharynx, thence spreading to cause the more general disease, are as follows:

In a very large proportion of cases epistaxis (p. 80) was the initial symptom, and occasionally this epistaxis was in itself quite severe. The occurrence of such epistaxis, spontaneously, in so many individuals suggests that there must have been some common cause for a breach of the surface of the nasal mucosa; this breach occurring as a rule at the very commencement of the attack, or even before the patient realised that influenza was upon him. The likely common cause would seem to be acute congestion from a microbic invasion of the lining of the nose, in which case if the germs of the disease were the cause of the inflammation, there would be a ready path for themselves or their toxins to enter the blood stream and cause the acute disease.

In the next case the constancy with which acute infection of the sphenoidal air cells was found (p. 88) with actual pus in these cells in a considerable percentage of the fatal cases, would seem to afford strong evidence of there being posterior nasal infection tending to spread into the parts in this immediate neighbourhood.

The relative frequency of earache, otorrhoea (p. 88) and deafness indicating similar spread to the middle ear via the Eustachian tube, points in the same direction; whilst from the mouth the way the micro-organisms tended to spread into the ducts connected with it, was shown by the development of parotitis (p. 86).

Again—though perhaps less cogent as an argument—the way in which meningococcal meningitis (p. 86) tended to occur in a larger proportion of these influenzal cases than one would expect from its incidence in the population generally, led one to think that this might be due to the influenzal infection in
the nose opening up channels through which the meningococci latent in the posterior nares could obtain a path of entry to cause their attack upon the meninges.

When bacteriological examination of the posterior nares of the apparently healthy contact cases was made the same varieties of organism as abounded in the tissues of infected patients would be found, but this, perhaps, is not an important argument in favour of the posterior nares being the primary focus from which the disease would subsequently develop, because even amongst the healthy individuals in non-epidemic times so many varieties of bacteria are found in the posterior nares that their recovery in influenzal contact cases need not necessarily have indicated a departure from the normal. The above points, however, and especially, perhaps, the common epistaxis and the frequency of infection of the sphenoidal cells led one to think that the nasal passages, and especially the posterior nares, were an important nidus for organisms which, by further spread, produced the disease; and one could not help feeling that an important means of checking development of the disease in contact cases was to wash out the nose and mouth and the posterior nares as adequately as possible at least twice each day, using for the purpose a bland antiseptic, mild enough not to irritate the mucous membrane, and yet sufficient to assist in keeping these parts clean.

The whole question of prophylaxis is dealt with elsewhere by others, but I wish to emphasize my belief in the importance of the nose, the naso-pharynx, mouth and fauces in this connection and in their relation to the route of infection in many at least.

Treatment.

I do not propose to go into the question of treatment at any great length, for once the influenza attack in any individual case had developed into the "pneumonic" type, it appeared that, no matter what treatment was adopted, it was extremely difficult, if possible at all, to modify the course of the disease in the least; and it was borne in upon one with the strongest emphasis throughout the epidemic that the thing to pay attention to was prevention rather than cure.

In the average case best results were obtained if the patient, directly he felt ill at all, went straight to bed and stopped there; those who tried to keep about in spite of having the disease upon them not only ran unnecessary risks themselves but also did harm, even if they did so with the best intentions, because, by keeping up and about, they were sources from which others became infected.

The patient should go to bed at once, take a tumbler of hot whiskey and water, 15 or 20 grains of aspirin, and cover himself up with a sufficiency of blankets in the hope of breaking out
into a good perspiration. There seemed little to be gained by
avoiding any food at all; even milk was best avoided for 48
hours, provided that the patient during this time drank as much
fluid as he could manage, either as plain water or as barley-
water, lime juice, lemonade, weak tea, soda water or iced water.
Five pints a day would not be too much. If need be a simple
aperient, such as castor oil, would be given at the beginning,
and for the rest the treatment resolved itself into one of skilful
nursing without the administration of any drugs, unless there
were some symptom calling for individual treatment. Aspirin,
for example, to relieve the headache, or to give sleep; a sedative
mixture to relieve cough until such time as there is phlegm to
be brought up when the sedative mixture might be changed
for an expectorant one; a more potent hypnotic if there were
sleeplessness in spite of aspirin. If definite bronchitic catarrh
develops, leading to the dreaded "pneumonic" complications,
one would be only too thankful if one knew of anything which
would with any certainty check the disease process, but one
met with nothing that was in the least degree successful in this
respect. In a few cases, say a hundred or so consecutively, one
might think that one particular remedial line one had adopted
was giving undoubted benefit and then in the next two or
three hundred cases treated in exactly the same way the same
procedure would prove entirely disappointing. From experience,
extending over thousands of cases, the general conclusion was
that the nursing was of infinitely greater importance than the
drugs administered. This applies not only to drugs but also to
the use of antiseptics either by inhalation or by injection, and
also to attempts at specific antibacterial treatment by means of
vaccines or by sera. All kinds of antiseptics given by inhala-
tion were tried and they were given continuously, or inter-
mittently, for longer or shorter periods, and all without obvious
benefit. In the belief that injections of antiseptics into the
blood stream might do good, such things as flavin and eusol
were given* intravenously but without any apparent benefit.
Patients were venesected without good. Infusion with normal
saline either subcutaneously or intravenously or both, also gave
no benefit. Some patients were both venesected and infused,
but unavailingy; that is to say, the mortality was much the same
amongst cases treated thus and cases in which practically no
' treatment other than nursing was adopted.

For the cyanotic cases venesection does no good, for the
cyanosis is not due to cardiac failure but to anoxhæmia resulting
from the albuminous exudate into the alveoli, interalveolar
tissues, and tubes, in addition to whatever broncho-pneumonia
or haemorrhage has also developed. In theory, the correct
treatment for this anoxhæmia would seem to be the continuous
inhalation of oxygen, and repeated attempts to give this oxygen
continuously by means of Professor Haldane's special apparatus
were made; but in very few cases, even amongst those who were
willing to persist with the treatment, did the giving of oxygen in this way seem to really benefit the patient, whilst, on the other hand, in the great majority of cases it was only by urging and insisting that one could get the patient to submit to keeping the oxygen mask on; the wearing of it seemed more often than not actually to increase the patient's distress and he was generally only too thankful to have it taken off. At the same time one does feel that if oxygen, could be given continuously to the cyanotic type of case, without at the same time creating a sense of distaste to the treatment on the patient's part, it ought to be beneficial and the right thing to use. One wonders whether in future cases, should they occur, it would not be a better plan to give the oxygen through soft rubber catheters inserted through the nostril so that a constant stream of oxygen might thereby pass into the patient's respiratory passages without producing that sense of oppression and suffocation that the patients complain of when wearing a face mask. I saw no cases treated in this way, but I think it would be a procedure worth a trial should any similar occasion arise, using the technique described by Stokes and Ryle (Guy's Hospital Gazette, 9th August 1919) in connection with cases of acute gas poisoning in France.

It is difficult to give any opinion as to whether the patients should be nursed in warm rooms or in cold, or even out of doors; at the beginning of the attack warmth, and the production of perspiration, certainly does seem to tend towards a favourable course in the disease, so that in the early stages at any rate it would seem advisable to have the patient in warm, though well ventilated, rooms or wards. On the other hand, when one had the opportunity of seeing the severe "pneumonic" cases verging upon cyanosis, or with the heliotrope colour actually developed, sometimes nursed in relatively warm wards and sometimes put right out of doors with waterproof canopies to keep off the rain, and warm clothing to keep their bodies warm, though their faces were exposed to the autumn or winter air, there could be little doubt that of these two alternative procedures, the outdoor and apparently more drastic line of treatment gave the better results. In some hospitals where there was immense strain upon the available beds, it became necessary to put the worst, and apparently hopeless cases, elsewhere, in order to make a maximum amount of room for less bad cases that seemed recoverable, and one saw scores of extremely bad cases transferred from the wards to the quadrangles out of doors, under waterproof canopies, and whereas at first one felt that this—though a necessary procedure—would at least not help any of these dire cases to get better, one found to one's surprise that a larger number of those very worst cases put out of doors did, as a matter of fact, recover than would have been the case, one felt sure, had they remained indoors. One is therefore in this difficulty: one feels that at the beginning of the attack the
patient should be nursed in warmth; that if "pneumonic" cyanosis has developed he is better out of doors; but that one does not know just at what stage the change of conditions of nursing should be made for the best advantage of a bad case.

Although one formed no favourable impression of any of the vaccines or sera used by oneself during the epidemic, one equally feels that further researches by those skilled in connection with them should lead to the discovery of either a vaccine or a serum, or some similar product which would help to modify the attack and curtail its severity. One is very far from saying that no vaccine and no serum is likely to do good; one can only say that one was not at all impressed by any of those that one had to one's hand to use—whether autogenous or stock.

That serum treatment may possibly be of great value in future cases seems indicated by Dr. Huff-Hewitt's experiences. He reported four cases in the British Medical Journal for May 10th, 1919, page 575. His treatment needs trial in a very much larger number before one can give a final opinion upon its value, but it strikes one as being fraught with great possibilities. Briefly, it consisted in obtaining blood by the syringeful from a patient recently convalescent from a moderately severe attack of the disease, allowing it to clot and then injecting the resultant serum subcutaneously into the patient who is severely ill. In his case it was a convalescent mother who thus gave her serum to her child, and the latter though extremely ill at the time, forthwith improved and made a good recovery. The same result ensued in three other very serious cases treated in the same way, and one admires the resourcefulness of the practitioner who carried out this treatment without any laboratory to help him. It was not until the epidemic was over that one saw his publication, and one did not test the treatment oneself, but the possible value of making use of serum from convalescent individuals in the treatment of other patients still acutely ill seems clear, and one would advocate further use of the method in any future epidemic in the hope that thereby some more or less specific remedy will have been discovered for that which throughout the epidemic of 1918-19 baffled all other forms of treatment when once the "pneumonic" type of the disease had obtained a strong hold upon the patient.

Morbid Anatomy and Histology.

Three of the most striking points brought home to one by post-mortem examination in fatal cases are:

1. The fact that the lesion in the lungs in "pneumonic" cases is practically never a true croupous lobar pneumonia in the ordinary sense, but a complex and
variable mixture of inflammatory lung lesions, of which even broncho-pneumonia forms only a part.

(2) The fact that though the "pneumonic" lesions are very striking, changes elsewhere are also prominent, especially in the kidneys; suggesting that, although the condition is referred to generally as "pneumonic," the effect of the acute microbic toxaemia is much wider spread than this term would suggest. A general microbic toxaemia or even septicaemia rather than a purely pulmonary infection.

(3) The fact that even when the greater part of the changes produced seem to be in the respiratory tract, they are not confined to the lungs, but extend from the highest to the lowest parts of the track—from the sphenoidal air cells and nasopharynx above to the pleura below.

Having emphasized these three points, it is expedient to describe the chief changes found in the organs, seriatim.

The condition of the sphenoidal air cells, the ethmoidal and frontal sinuses, the significance of inflammation in the nasopharynx, extensions thence to the Eustachian tubes and middle ear; and similar extensions by Stenson's ducts from the mouth to the parotid glands has been discussed above.

The lymphatic glands in the neck and thorax, and often those in the abdomen also, have generally been swollen and crimson from acute inflammatory hyperæmia. Those most swollen and inflamed have generally been those situated below the bifurcation of the trachea, these sometimes being found many times their normal size, occasionally looking as if about to suppitate in their central parts, although but few cases of real abscess in these glands was met with. Presumably the great swelling and hyperæmia of the bronchial glands was the result of microbic absorption into these glands from the inflammatory foci in the lungs. The glands in the hilum of each lung were generally hyperæmic and swollen from the same cause, and from the thorax up in to the neck the deeper glands, especially those in the sulcus between the oesophagus and trachea on either side, were crimson and inflamed as a rule, though their enlargement was less marked than was the case with the bronchial glands. The abdominal glands, notably the mesenteric and retroperitoneal, were less constantly inflamed and enlarged than were those in the thorax and neck, but not infrequently they attracted notice by their swollen size, very often by their dull red or crimson colour.

The larynx did not as a rule show oedema or naked-eye evidence of laryngitis, and no case of laryngeal exudation was noted, but the mucosa of the trachea from nearly the level of the larynx down to the first subdivision of the bronchi was nearly always dark red or crimson from acute inflammatory injection, the degree of which became more and more pronounced
as the tubes were traced from above downwards. It was exceptional to find much purulent phlegm within the larger tubes, and no great degree of superficial exudate was present, but it was common to find a glazed look of the surface as though the dark red and inflamed mucosa were covered by a fine pellicle of coagulable exudate, in addition to which multiple pin-point depressions or pits were generally obvious on close inspection, due either to exaggerated openings of the sub-mucous glands or else to minute shallow abrasions from superficial ulceration.

The finer bronchioles, as seen in the cut surface of the lungs, showed extreme reddening of their mucosa; variable amounts of muco-pus or pure pus was expressible from them, but only exceptionally to anything like the degree that characterised the "purulent bronchitis" cases of 1916 and 1917.

The lungs were invariably heavier than normal, often greatly so, the increased weight being a feature of the lower lobes rather than the upper. Acute pleurisy, was not always present, but it was common, generally of the "lack-lustre" type, or with a pellicle of granular lymph that could be peeled off, but seldom presenting anything like the "buttery" exudate often seen with croupous pneumonia. Large pleuritic effusions were exceptional, many of the acute pleurisies were dry, but with some there was effusion of a few ounces, or perhaps a pint or more, of slightly turbid colourless or pale yellow or blood-tinged fluid containing fine flakes or threads or shreds of detached fibrin. This fluid practically always contained microorganisms and polymorphonuclear leucocytes in abundance, but it was not pus in the ordinary sense.

The acute pleurisy was commoner over the lower lobes than the upper, but it might be universal; beneath the inflamed visceral pleura there were generally angry-looking dark red petechial and larger haemorrhages. Acute pleurisy might be present over lung that was not consolidated, but more often pleurisy and obvious lung inflammation coincided.

The lung lesions, complex or variable, struck one as being quite different in character to anything one had met with at all commonly in the thousands of autopsies one has performed during the last 20 years. Broncho-pneumonia was very frequently a part of the picture-complex, but it was not like the common broncho-pneumonia of ordinary years; lobar pneumonia was not represented in any of its ordinary phases—simple congestion, red hepatization, grey hepatization, purulent infiltration. It would be inadequate to describe the lesions as those of either broncho-pneumonia on the one hand or croupous lobar pneumonia on the other. To obtain a picture of what was met with one would need to mingle together in different
proportions in different cases that had presented similar clinical signs, symptoms, and course, any or all of the following:—

Acute congestion, giving a more or less dark red colour to the whole lung.

Diffuse haemorrhage, producing still darker red, often almost black-red areas in the already deep red lung, these haemorrhagic areas being of all sizes and shapes from miliary to massive and scattered at random, throughout the lungs.

Broncho-pneumonia, sometimes recognisable only on careful search, sometimes widely disseminated and occasionally confluent; most marked as a rule in cases that had survived more than a day or two and generally giving one the impression of being rather a super-added development, almost inevitable in lungs so affected, than the primary and essential lesion.

Haemorrhagic infarcts, similar in colour to diffuse intrapulmonary haemorrhages but differing from the latter in their pyramidal shape, with the broad base of the pyram beneath the pleura.

Miliary abscesses, often aggregated together in little focalised groups of from three or four to a score or more, showing up as paler foci in the dark red background, often situated in the midst of a deep crimson hemorrhage, or an infarct, and not infrequently visible as a group of little abscesses beneath the pleura before the lung was cut. These grouped abscesses were similar to those seen in the midst of septic infarcts due to infected emboli such as result, for instance, from lateral sinus thrombosis.

Collapse, sometimes superficial only, sometimes associated with multiple areas of broncho-pneumonia, sometimes massive and independent of broncho-pneumonia, but adding an important factor to the lobar distribution of the signs of consolidation.

Croupous pneumonia, met with very rarely indeed in macroscopically recognisable degree, but occasionally forming part of general mixture of lesions.

Compression of one or other lower lobe by pleuritic exudate.

Purulent bronchiolitis, with thick pus expressible from the bronchioles seen in the cut lung.

Passive oedema of the bases.

Active oedema with extensive albuminous exudate into all parts of the lungs, not definable by the naked eye, but shown to be extreme in many histological sections; a peculiar and apparently highly important feature of these cases (see below).

Interstitial emphysema, often widespread throughout the lung tissue, but less easy to detect here with the naked eye.
than when the typical gas bubbles formed the familiar streaks beneath the pleura.

It is difficult to make a word picture which adequately describes what was the average admixture of the above diverse ingredients. Many writers have referred to them conjointly as "pneumonia," giving the impression that "croupous pneumonia" prevailed; this was emphatically not the case; others have spoken of the condition as in the main "broncho-pneumonia"; but this again gives a wrong impression because broncho-pneumonia was only a part of the whole; congestion, haemorrhage, infraction, inflammatory oedema, pleurisy and collapse might all be extensive in a case showing so little broncho-pneumonia that the latter had to be sought for carefully to be found at all. One feels tempted to coin a new word altogether to express so complete a type of lung-inflammation, and to term it "pneumonitis"; for no part of the lung tissue seemed to escape. One could then speak of the lesions as being of such varying types as the following:—

"Pneumonitis," with preponderance of congestion and oedema, but with little or no consolidation;
"Pneumonitis," with much congestion, bronchitis and some broncho-pneumonia in the lower lobes;
"Pneumonitis," with preponderance of congestion, interstitial haemorrhage, oedema, and collapse, but little broncho-pneumonia;
"Pneumonitis," with congestion, haemorrhages, collapse, and extensive broncho-pneumonia;
and so on.

It is worthy of particular note that here and there one came across a case with the same clinical picture as the rest, and yet with lungs so little altered to the naked eye that one might easily have passed them as almost normal—no consolidation in any part of any lobe; microscopically there would be bronchiolitis, peribronchiolitis, and diffused inflammatory albuminous exudate, both interstitial and intra-alveolar; yet without any discernible broncho-pneumonia, and no obvious consolidation anywhere; no part of any lobe, larger than a minute fragment, would sink in water, and yet the clinical picture of the case was indistinguishable from that in which extensive broncho-pneumonia would be found at autopsy. In short, though broncho-pneumonia was a common thing to find post-mortem in little or greater degree it was only part of a much more complex mixture of lesions and, though usual, it was not absolutely an essential part of the mixture.

Microscopically the lung lesions were found to be just as protean as the macroscopic appearances would suggest. Sections taken from different parts of the same lung would often look so totally different that at first one could hardly believe that they were from the same case. Some showed
typical broncho-pneumonia, the alveoli being crowded with desquamated swollen epithelial cells, leucocytes and red corpuscles, and the interalveolar wall thinned by compression so as to be hardly more than linear; others, extensive hemorrhage with myriads of red cells bulging the interalveolar walls and leaking into the interior of the alveoli; or again some alveoli might be full of extravasated blood bulging into nucleated cells of broncho-pneumonic origin, and others with fibrillated fibrin like that of croupous pneumonia; in another place the alveoli and the interalveolar walls might look normal, but a bronchiole would be found to be full of exuded small round cells and epithelial cells derived from partial disintegration of the mucosa with diffuse small-round-celled infiltration of the peribronchial connective tissue; in another section it might be difficult to recognise lung tissue at all, the whole being densely infiltrated with inflammatory cells, and the interalveolar walls apparently necrotic and destroyed; but none of the above seemed so remarkable as the "Gruyere cheese" changes which were so common and which were so entirely unlike what is met with in any ordinary forms of pneumonia that they seemed to be of essential importance, the other changes—haemorrhages, broncho-pneumonia and so on—being super additions. The condition has been illustrated in Special Report Series No. 36 of the Medical Research Committee, and it is not very dissimilar to the initial results of the action of acute irritant gasses on the lungs. The first impression a typical section gives one is that the paraffin wax may have been but partially dissolved out when the block was being prepared. All through the section—filling the alveoli in some places, distending the interalveolar walls in others or the peri-bronchial connective tissue, or blocking the bronchioles, or infiltrating all parts of the section simultaneously—there is a hyaline or homogeneous material, staining faintly pink with eosin, but containing few cells; resulting, one supposes, from the rapid out-pouring of an albuminous, non-cellular, coagulable exudate which, in the process of fixation of the tissues, becomes converted into what looks like hyaline material.

Amid this one discovers outlines of normal alveoli in some places, alveoli whose walls are disintegrating in other places, and in yet other places, spaces which are more or less obviously not alveolar spaces at all—round or ovoid holes of varying sizes without any defined walls, but reminiscent of the air holes which characterise a Gruyere cheese. Some of these may be the result of breaking down of interalveolar Walls so that two, three, four, five or more original alveoli have been thrown together into one larger one, looking like a microscopic cavity in the hyaline matrix; some, on the other hand, appear to be gas-bubbles—microscopic interstitial emphysema in the albuminous intra-pulmonary exudate. ...
Precisely similar non-cellular exudate into gas-holes is seen after acute gas poisoning. It seems likely that it is this acute inflammatory oedema of the lung tissue which, preventing inspired air from gaining access to the intra-capillary blood, accounts for the anoxhæmia and heliotrope cyanosis of the worst cases.

That broncho-pneumonia, hæmorrhage and other secondary changes should supervene on this lesion is what one would expect; they did so after acute gassing in France if the patient was able to survive long enough; but just as the lesion caused by gassing is not primarily broncho-pneumonia, but primarily and acute albuminous inflammatory oedema followed by broncho-pneumonia, so in these influenzo-"pneumonic" cases the primary effect was, in a large number of cases at any rate, an acute inflammatory albuminous exudate; broncho-pneumonia developing subsequently if the patient survived long enough.

The kidneys in almost all the fatal cases showed acute non-suppurative nephritis, milder in degree perhaps than that met with in scarlatina, but similar in type. The kidneys were uniformly enlarged, often weighing from 10 to 12 ounces the pair. The capsules peeled easily.

In colour they were sometimes irregularly blotched and mottled with alternate pallor and dark red; when cut the cortex looked swollen, with varying degree of lack of sharp definition between cortex and medulla; but most striking of all was the way in which, when the organ cut open was laid on the table with the cut surface upwards, the small vessels everywhere oozed out dark red blood without squeezing until in a brief space there was a film of blood obscuring everything; the condition was not quite the acute red blood-dripping kidney of scarlatina, but was approaching this condition and might be described as one of large red blood-oozing kidney.

Microscopically there were varying degrees of congestion of capillaries, swelling of glomerular tufts and epithelial cells, interstitial blood-extravasations and cloudy swelling of tubular epithelial cells, with desquamation of broken down cells into the lumen of the tubules; definite small-round-celled infiltration was less common.

The liver was generally of fairly normal colour and consistence; occasionally rather on the pale side, the pallor when present being fairly uniform. The whole liver looked swollen and enlarged in most cases, but otherwise not materially affected to the naked eye. On histological examination, however, nearly all the liver showed acute degenerative changes of many of the parenchymatous cells, particularly in the central parts of the lobules; cloudy swelling and lack of nuclear staining-power being more pronounced than actual fatty change. There was little or no tendency to small-round-celled infiltration of the portal canals.
The heart showed neither pericarditis nor endocarditis in any case I saw, nor any special tendency to supracardial ecchymoses. Many observers have reported dilatation of the heart in these cases, but what struck me most of all was the remarkable constancy with which the heart showed hardly any departure from the normal at all. When post-mortems were performed early, the heart muscle was firm and of good colour, rigor mortis in it was good, and there was little tendency to dilatation of any of the cavities; the right auricle and ventricle might be moderately distended with dark blood clot and, in comparison wish the empty and contracted left side, looked a little dilated; but the degree of distension was seldom great, and acute dilatation of the heart was quite unusual.

The thyroid gland was uniformly enlarged in nearly every case, a phenomenon which attracted attention though its causation was not obvious. The gland was sometimes quite three times the average size, and the isthmus was swelled as well as the lateral lobes, much in the same way that it is in Graves' disease. This change occurred as much in those who had never been overseas as in those who had been abroad, so that it could not be attributed to previous illness such as trench fever which may cause similar enlargement, notably in cases associated with D. A.H. The swollen gland was firm, and uniform in consistence, generally of its ordinary dull-red colour, and microscopically it did not show evidence of being acutely infected. The condition seemed to be one of simple uniform swelling of the gland secondary to the acute toxemia of the general disease.

The supra-renal capsules were in the main of normal size and external appearance; but their medullary portions were generally dark red and pulpy from breaking down.

Under the conditions that existed at the time it was not easy to make special observations upon them and one would not care to say how much of the change in them was due to rapid post-mortem disintegration and how much to disorder caused by the infective disease itself.

The alimentary canal seldom presented macroscopic evidence of infection; but there was a group of cases observed and recorded by Lowe in which the colon was in a state of extensive and acute ulcerative colitis, with destruction of the mucosa, similar in type and degree to that which results from acute dysentery. The caecum and ascending colon were the parts most affected, though no portion of the colon appeared exempt. Whatever the special factor may have been to cause this bowel ulceration in Lowe's cases one does not know; apparently it was not the use of calomel or similar drastic purge. Pneumococcal or streptococcal colitis are familiar enough in other circumstances, and one is not surprised that cases of this kind did occur during the epidemic; rather is one surprised that they were observed so seldom.

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