I found during the years in which cases of true influenza were rather fewer than a great many cases of catarrh and fever yielded very rapidly to salicin, although it was not necessary to give the big doses quite so often. I have noted carefully the clinical symptoms and appearances of all the cases that I have seen since 1889. There has, in each really bad epidemic, been a distinct difference in the effects produced on those who contracted the disease. The pain which is so characteristic of the complaint has varied in the different epidemics, sometimes being entirely referred to the back of the eyes, and in other cases being felt more in the limbs or in the head, but there has been a line of distinct resemblance running through all these outbreaks. There is evidently an acute infection, which in some of the earlier outbreaks was more inclined to affect the brain and the nerve centres, and in the latter ones the respiratory system.

It appears to me, from clinical observation only, that there is probably some minute filter-passing organism which is in the main responsible for these attacks, and that sometimes, as in the 1918 and 1919 epidemics, its virulence is augmented by the action of those other microbes which were separated and found in these cases.

Salicin seems to have a selective effect on what I may call the influenza infection pure and simple, and is not so rapidly effectual in those cases in which there is a treble or quadruple infection as well.

I have given my experience during thirty-eight years in the treatment of this disease, and it appears to me that it leaves only two alternatives. The first is that with this means of treatment, boldly and wisely pushed, it will be possible in any future epidemic of influenza to eliminate complications, save hundreds of lives, and restore patients to complete health free from fear of sequelae. The second is that from 1889 onwards, in a practice which is not local but embraces the whole of the West End of London, I have never seen a case of true influenza, and that the results which I have put before you were obtained in cases of simple catarrh with fever, which would have recovered under any form of treatment in the same way. This second alternative cannot be accepted.

A Lecture

ON

PULMONARY FIBROSIS IN CHILDREN.*

BY

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THE study of chronic pulmonary disease in childhood resolves itself into a consideration of fibrosis of the lungs in its various forms. Two of these forms are specific infections—namely, tuberculosis and syphilis—and I do not propose to consider these to-day, except in the discussion of the diagnosis of non-specific forms and with regard to the fact that the former, tuberculosis, may be engrafted upon simple fibrosis at any stage of the disease. The fibrotic form of tuberculosis may be looked upon as rare in childhood, while syphilitic fibrosis is probably very rare indeed and can never be diagnosed with any certainty.

This leaves simple fibrosis, which has in the course of years accumulated a wonderful variety of names. It is known as cirrhosis of the lung, a name which merely expresses the consistency to which the lung tissue is reduced; as chronic interstitial pneumonia, a name suggesting that the inflammatory process is progressive, which is not often the case; and as fibroid phthisis, a name which to many minds connects it too definitely with tuberculosis, and is therefore unsatisfactory.

It is non-specific in that we cannot point to any one organism that may be responsible, and indeed simple fibrosis must be looked upon rather as a healing than a disease, and might be disregarded as mere scar tissue if it

were not for secondary results which are responsible for symptoms.

By far the commonest cause of fibrotic lung in childhood is an unresolved bronchopneumonia, in many cases complicating whooping-cough or measles. The mortality rate of bronchopneumonia is so high that we have only too many opportunities for studying its morbid histology, and, in a certain type of case especially, the mechanism of early fibrous tissue formation may be demonstrated. This is the type which may be called the "wandering pulmonary infection," in which, during a period lasting from three to six weeks or even more, areas of the lungs are affected in succession, one part resolving or partly resolving while the process spreads to another. Some of the patients recover; others succumb from toxæmia and exhaustion.

Histological examination of active bronchopneumonic tissue raises the question why organization and fibrosis do not more often occur. For here, in marked contrast to the changes in lobar pneumonia, there is extensive damage of lung tissue. The walls of bronchioles and alveoli are infiltrated with both fluid and cells, while the peribronchial and perivascular tissues suffer from a similar exudation. The cells are partly red corpuscles, partly polymorphonuclear leucocytes. Penetrating into all affected parts are swarms of bacteria.

When resolution takes place the whole of this inflammatory exudate has to be cleared away. Tryptic ferments digest the mass, and the liquefied product is removed by absorption. But this is necessarily a much slower process than in lobar pneumonia, and occasionally restoration fails. When this happens, that friendly cell the fibroblast steps in, the inflammatory products are "organized"—that is, infiltrated and pierced by numerous fibrous tissue cells, which, spreading in all directions, form a firm network to weld the spongy mass together, kill the bacteria by cutting off their food supply, and finally, following the example of scar tissue all over the body, contract and become inactive. Meanwhile the alveoli of the affected area, some of which are isolated by occlusion of the terminal bronchiole and others pressed upon by the growth of fibrous tissue, are partly or wholly collapsed.

All this would matter very little if it simply meant putting a part of one lung out of commission. The body can well adapt itself to the possession of one lung, and get from it sufficient aeration of the blood.

Unfortunately, in nearly all cases bronchiectasis supervenes to a greater or less degree. The smaller bronchioles are usually obliterated, but the bronchi, weakened by the primary disease and frequently filled with muco-pus, are easily distended by the action of contracting fibrous tissue. Large cavities are unusual; the cavities are small or medium-sized, and grouped centrally towards the root of the lung. Eventually the pleura is involved, evidently a late event, as pleurisy is rare in bronchopneumonia, and the affected part of the lung becomes closely adherent to the thoracic wall. This anchorage is an additional factor in the stretching of bronchial walls and the formation of bronchiectatic cavities.

Bacteriology.

The bacteriology of bronchopneumonia embraces a large variety of organisms, and presumably any infection may lead eventually to fibrosis, but there is evidence to show that the pure pneumococcal forms are rarely followed by this complication, and as measles, whooping-cough, and influenza commonly precede the condition it must be assumed that a streptococcus causes the original infection in the majority of cases, though Friedländer's bacillus, Pfeiffer's bacillus, and others may be the cause at times. When bronchiectasis is established numerous organisms may cause secondary infection, and some of these are putrefactive.

Pleurisy.

Pleurisy may be a primary cause of pulmonary fibrosis in children. The pleurisies of children are the acute form accompanying lobar pneumonia, tuberculous pleurisy with effusion, and rheumatic pleurisy. In the first condition, if an empyema supervenes, the collapsed lung may become fibrous, especially if the abscess is left undrained too long.

* Notes of a lecture given for the Fellowship of Medicine in January, 1927, at the Royal Free Hospital.

In the second and third it is quite possible that the lung may be invaded by fibrous tissue from thickened and adherent pleura, but it is almost impossible to prove this sequence, and in any case it is but a matter of academic importance.

Lobar Pneumonia.

It is a matter for dispute whether true lobar pneumonia could be followed by fibrosis. The study of the morbid histology of pneumonia makes it seem exceedingly unlikely, as there is little or no damage to the actual lung tissue during the process of exudation, consolidation, and resolution. Exceptionally, however, damage may occur, and from time to time cases are reported in which a cirrhotic lung develops after a pneumonia of a definitely lobar type. It has been suggested that even in these cases the cirrhosis may have been a latent condition preceding the pneumonia, and caused by a previous bronchopneumonia. In connexion with this it is suggestive that out of 41 consecutive cases of lobar pneumonia in my beds at the East London Hospital for Children during the last two years, as many as 18 gave a history of previous bronchopneumonia or whooping-cough.

Symptoms and Signs.

Pneumonic resolution may be so long delayed in debilitated subjects that it is difficult to say exactly when fibrosis begins. A few crepitations persisting at the bases for several weeks or even months after bronchopneumonia must not be diagnosed as due to fibrosis. Usually the patient suffers very little once the condition is fully established, though there has probably been ill health, anaemia, and lassitude during the convalescence from the acute illness. Cough is rarely absent for long, however, and there is some dyspnoea on exertion. He may live a moderately active life with comfort, but is inclined to get attacks of bronchitis from time to time. If and when bronchiectasis has supervened, the symptoms are of a more distressing character. The cough is not frequent because the mucous membrane loses its sensitivity, but is apt to occur on change of position, as when rising or going to bed, is paroxysmal in character, and results in the production of large quantities of muco-pus. Haemoptysis is common, and frequently leads to a diagnosis of tubercle.

The child has a rather characteristic appearance, is generally well nourished, with somewhat prominent eyes, a coarse skin, and bright red cheeks. The brilliant colour gives a false air of robustness, which is dissipated when after exertion the bright red gives place to cyanosis. The finger-tips and toes are clubbed, with smooth shiny skin; this sign is always present, though perhaps not so marked as in congenital heart disease, but it must be remembered that clubbing of the fingers may appear after as short a period as three weeks of bronchopneumonia, and does not necessarily indicate that chronic interstitial changes are taking place.

The physical signs in a well marked case are not difficult to elicit. The changes are generally unilateral, and more often at the left base than elsewhere. On inspection, flattening of the thorax may be seen on the affected side, or even a marked concavity. When a measurement is made a definite shrinking is found to the extent of an inch or more between the two sides. If the case is a long-standing one, even the muscles are wasted, including the muscles of the shoulder girdle, and there is a falling in of the ribs. On palpation it is found that movement is diminished. Vocal fremitus may not be altered, but is usually decreased. The percussion note over the affected area varies considerably: flatness is common, but it is rather a flat tympany than a wooden note, and there may at times be resonance, either due to empty bronchiectatic cavities or to compensatory emphysema of unaffected lung. In any case the note is most unreliable. Air entry is poor, so that the breath sounds are usually considerably diminished, though still retaining their vesicular character; only rarely is bronchial or cavernous breathing heard. Adventitious sounds may be entirely absent if the lung is solid. More often dry crackling crepitations are heard, indicative of partial expansion. Added to these may be bubbling and crackling râles of various kinds when cavities are present; they change from day to day and disappear when the cavities dry up. It is not unusual to hear a few

crepitations at the opposite base; that is probably due to similar changes in a mild degree, for although the main damage is unilateral, it is quite possible that a certain amount of fibrosis affects the opposite lung, since bronchopneumonia is generally bilateral. The heart is drawn over towards the affected side. The area of cardiac dullness may not always be easy to define, as it blends with the impairment due to fibrosis, but if the position of the apex beat can be located the diagnosis of shrinkage of lung as against fluid in the pleural cavity is established.

The course of the disease is long, and the expectation of life depends chiefly on the amount of bronchiectasis and whether this is progressive. Care and favourable surroundings make a vast difference. The patient who has good food and adequate clothing, who can spend the winter in a warm climate, and take as much exercise as is good for him, may look forward to many years of comfort. Without these luxuries he is frequently the victim of recurring attacks of bronchitis, which leave him with bigger and freshly infected bronchiectatic cavities. He may be carried off by an acute exacerbation of pulmonary infection, by cerebral abscess, or by lardaceous disease. The connexion of tuberculosis with simple pulmonary fibrosis is an interesting one. The finding of tubercle bacilli in the sputum naturally suggests a tuberculous origin, but it is well known that tuberculosis may be engrafted upon a simple cirrhosis, and indeed finds there a favourable medium for growth.

The tubercle bacillus may be introduced at any time in the course of the disease, and eventually the lung may be riddled with other cavities which are not bronchial in origin. Patients suffering from chronic fibrosis should have a sputum examination done from time to time as a routine, as it is obviously better to know if infection has taken place, even though it may not make much difference in the treatment of the individual.

Diagnosis.

In the early stages fibrosis of the lung is difficult to diagnose. Bronchopneumonia must be given two or three months to clear completely in debilitated subjects, and the diagnosis of permanent damage should not be made on a few basal crepitations alone. The impaired percussion note and weak air entry may suggest fluid, but the note is of a different quality, and the position of the heart gives the clue. Once the chest wall has fallen in the diagnosis is no longer in doubt. One's main difficulty in the early cases is to decide whether tubercle is present or not. Tuberculous infiltration of the lung is generally basal in children, but tends to early caseation rather than fibrotic change. The child wastes, sweats, and has some degree of pyrexia. Sputum may not be coughed out if the child is young, but an examination of the faeces reveals the presence of the tubercle bacillus in a large proportion of positive cases.

The general appearance of the child aids in the diagnosis, but not until either condition is well advanced. The engrafted tubercle, of course, adds considerably to the difficulties in diagnosis. The main differences may be tabulated as follows:

<i>Simple Fibrosis.</i>	<i>Tuberculosis.</i>
Well nourished child.	Wasting.
Bright, slightly cyanosed complexion, with coarse skin.	Delicate, flushed soft skin.
No sweating.	Sweating.
Afebrile as a rule.	Evening fever.
Previous history of bronchopneumonia.	Gradual onset.
Sputum variable, but may be copious and offensive.	Not a large amount of sputum.
Sputum contains many and various organisms.	Sputum contains tubercle bacilli.

X Rays.—In the early stages the fibrous tissue infiltration may be seen over the damaged area, the changes in the periphery being as well marked as elsewhere. In tuberculosis, on the other hand, infiltration can be generally seen extending primarily from the roots. When bronchiectasis is established the cavities can be demonstrated, especially after an injection of lipiodol.

Treatment.

This is mainly directed towards keeping the bronchial tree as healthy and free from catarrh as possible. A warm climate is to be preferred, where the patient can be kept

as much as possible in the open air. Where conditions are bad, the disease can best be held in check by preventing accumulation of secretions. The child should be made to hang over the bedside every morning and evening so that the cavities may drain to the greatest advantage. The position alone is enough to start the cough. Since putrefaction of the sputum is one of the most distressing features, vigorous attempts at disinfection should be made. The methods are:

1. By enclosing the patient in a chamber saturated with the vapour of creosote or some similar disinfectant, for increasing periods of time.
2. Inhalation of disinfectants by the use of the Burney Yeo mask.
3. Intratracheal injections of disinfectants—for example, menthol and guaiacol in olive oil.
4. Administration of pulmonary disinfectants—for example, creosote and guaiacol—by the mouth.

Artificial pneumothorax has been suggested and occasionally tried for bronchiectasis, but it is rarely likely to be successful because, even if the attempt is made before fibrous pleural adhesions prevent collapse of the lung, it is obvious that just those parts which are meant to collapse—namely, the dilated bronchi—are least likely to do so; as they are supported by dense, inelastic layers of fibrous tissue.

"POLYCYTHAEMIA HYPERTONICA":

Is POLYCYTHAEMIA A SOMEWHAT FAVOURABLE SIGN IN HIGH BLOOD PRESSURE CASES?

BY

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It has often been noted in typical cases of splenomegalic polycythaemia (Vaquez-Osler disease) that in spite of the extremely high so-called "blood viscosity" (due to the great excess of erythrocytes in the blood) the blood pressure is only slightly if at all above the normal, and the heart is scarcely if at all hypertrophied. Thus, in the first really complete *post-mortem* examination on a typical case¹ (a man, aged 58 years), though the erythrocytes numbered 9 to 11 millions to the cubic millimetre of blood, the heart weighed only 298 grams (10½ oz.). The blood pressure had been moderately above the normal. In many other cases the brachial systolic blood pressure has been only about 150 mm. of mercury. That there is a dilatation of the blood capillaries in such cases has been proved by the observations of Brown and Giffin,² and also by the measurements of Hisinger-Jägerskiöld.³ L. J. Del Baere⁴ points out that in polycythaemia it takes less blood to supply sufficient oxygen to the tissues than in anaemia. Therefore, though, owing to high blood viscosity, the blood pressure may be somewhat increased, the heart has less work to do than in anaemia, and consequently cardiac hypertrophy is not rarely absent in polycythaemia, even where the blood pressure is above the normal.

The so-called "polycythaemia hypertonica" without splenomegaly (or "hypertonia polycythaemica") to which F. Gaisböck drew special attention in 1904 and 1905,⁵ seems to me to be probably a secondary or symptomatic polycythaemia, connected in some way with the persistent high blood pressure of early stages of granular kidney, or with primary arterial hyperpiesia (using Sir Clifford Allbutt's term) without definite signs of renal disease. In other words, the condition is one of renal or primary arterial high blood pressure with secondary polycythaemia rubra. It is not a very rare syndrome, occurring chiefly in middle-aged persons, and probably more frequently in males; perhaps Hebrews are somewhat more often affected than others. From true splenomegalic polycythaemia or erythraemia (the Vaquez-Osler disease) it is distinguished by the absence of splenomegaly, by the higher blood pressure, and by the absence of such marked facial cyanosis as is seen in old-standing splenomegalic polycythaemia. Moreover, the syndrome may, I believe, represent merely a phase or episode in the course of a case of high blood pressure, the polycythaemia rubra disappearing after a time, though the high blood pressure persists or increases.

Thus in a nervously active Hebrew man, aged 59 years, who was first seen by me in 1920 (when he was 52 years of age),⁶ the brachial systolic blood pressure has been generally 180 to 210 mm. Hg. The blood count in June, 1920, gave 8,000,000 erythrocytes and 12,000 white cells to the cubic millimetre of blood, haemoglobin 130 per cent. Since then the blood count has varied considerably, the case tending to become one of arterial hyperpiesia without polycythaemia. He is thin rather than fat, and has a red face, with a little generalized rosacea, without cyanosis. Sometimes there is slight conjunctivitis, probably of rosaceous origin.* His complaints have been chiefly pains, probably of fibrositic nature, in one part or another. At present he has a little sanguineous mucous expectoration; there are no x-ray signs of any pulmonary tuberculosis.

His brachial blood pressure is now (June, 1927): systolic 205 mm. Hg, and diastolic 150 mm. Hg. A blood count gives: haemoglobin 90 per cent.; erythrocytes 5,100,000 per c.mm. of blood, white cells 6,200 (polymorphonuclear neutrophils 65 per cent., lymphocytes 27 per cent., monocytes 4 per cent., basophils 1 per cent., eosinophils 3 per cent.). The urine (which was formerly free from albumin) is now of specific gravity 1020, and rather high-coloured; it contains a little albumin but no tube casts; it is free from sugar and does not contain excess of urobilinogen. The heart is somewhat enlarged towards the left.

In spite of considerable business and other worries this patient's general condition has on the whole remained remarkably good, and he looks rather young for his age; but he now almost certainly has an early stage of granular kidney. This brings me to the question of polycythaemia being a somewhat favourable sign in cases of persistent high blood pressure and early granular kidney. I think it must be taken on the whole as of relatively favourable prognostic significance. The polycythaemia, as Del Baere (see above) points out, to some extent relieves the heart from overwork. In fact, I now regard cases of "polycythaemia hypertonica" or "hypertonia polycythaemica" as cases of primary arterial hyperpiesia or of persistent high blood pressure of early stages of granular kidney, in which, owing to a favourable (sometimes familial) constitutional "polycythaemic disposition," the patient's organism has succeeded in developing a secondary blood reaction—namely, the polycythaemia—which to some extent helps to mitigate or delay the harmful effects of persistent high blood pressure.

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DEATH BY ELECTROCUTION.

BY

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WE feel justified in submitting for publication the following details, as cases of electrocution are comparatively rare, and there is a paucity of information concerning them, in the books usually possessed by the general practitioner. This was stated at the time to be the first known recorded case of electrocution associated with broadcast reception.

The unfortunate lady was aged 61; she had been treated by one of us (E. E. J.) for some time for a chronic mitral lesion of the heart, but had been able to lead an active business life. She had gone to bed early, about 9.15 p.m., when she was last seen alive by her daughter. At 10 p.m. her daughter returned to wish her good-night, by way of

* With acne rosacea there is sometimes a kind of "rosaceous conjunctivitis," as some eye specialists have noticed. In regard to rosacea-like dermatoses affecting polycythaemic subjects, compare E. Pick and P. Kaznelson, *Derm. Woch.*, 1925, lxxx, p. 159; W. Richter, *ibid.*, 1925, lxxx, p. 1075; J. H. Sequeira, *Proc. Roy. Soc. Med.*, Section of Dermatology, 1917, x, p. 14.