

abdomen had re-filled and 154 ounces were removed. The pulmonary condition did not clear and he died from heart failure a few days later.

The condition of the subdiaphragmatic viscera is shown in Fig. 4. Here the surface of the outer half of the right lobe shows anteriorly granulations which are larger than is generally seen in these cases. The inner portion of the right lobe is almost smooth on the surface, while the whole of the left lobe shows the fine surface granulations characteristic of malarial cirrhosis. The capsule of the spleen is especially thickened at its upper pole, where there is a broad attachment to the lower surface of the left lobe of the liver. The stomach is greatly deformed, the small curvature being hardly apparent and the pylorus approximated to the cardiac opening. The remains of the omentum are rolled up into a long cord which, running up to the right of the spleen, is attached by a firm, band-like adhesion to the posterior part of the under surface of the left lobe. There was a marked

FIG. 4.



Case 2. Malarial cirrhosis and splenomegaly, showing deformity with dilatation of the stomach and A, a long omental band adherent to lower surface of left lobe. D, Diaphragm. J, Jejunum. L, Left lobe turned up, adherent to upper part of spleen where the capsule is thickened.

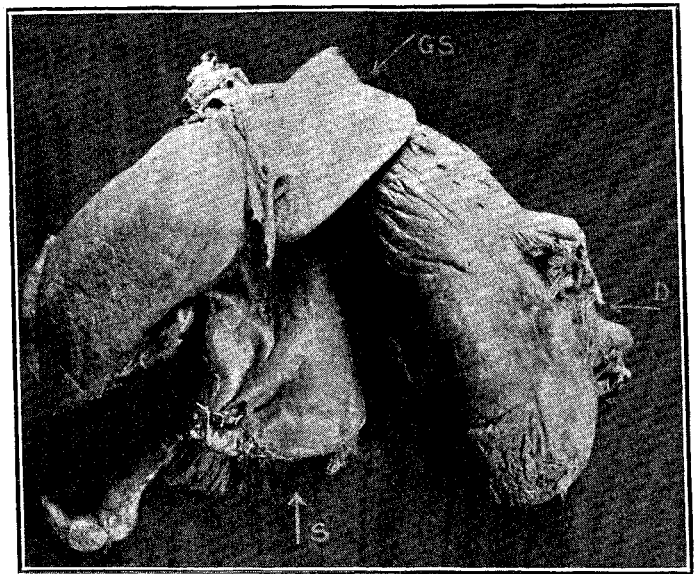
oesophageal varix above the cardiac opening. The liver was excessively adherent to the diaphragm. It was also enlarged and fatty and bile-stained on section. The renal cortex showed congestion and marked fatty degeneration. Sections of the liver showed the same irregular deposit of fibrosis as in the preceding case, with pigment in and among the hepatic cells.

CASE 3.—This patient was a Hindoo boy, aged about 17 years, who was admitted into the Jamsetjee Jeejeebhoy Hospital under the care of Dr. Surveyor on August 13th and who died on Nov. 10th. The enlargement of the spleen had been noticed for more than two years: accumulation of fluid had been noticed about a month before admission. Fever with rigors preceded the enlargement of the spleen. On admission the latter could be felt to be enormously enlarged and the liver was retracted beneath the ribs. On August 25th 350 ounces were withdrawn, and again on Oct. 3rd 424 ounces were removed. The spleen was punctured shortly after the first tapping but revealed nothing.

Necropsy.—At the post-mortem examination about twelve pints of fluid were found in the abdomen. Death had resulted from a general pulmonary oedema. Fig. 5 shows the condition of the subdiaphragmatic organs. The surface of the liver was finely granular. Practically the whole of

the great curvature of the stomach was directly adherent to the spleen. From the lower portion of the latter there were many long thread-like adhesions running to the diaphragm, while the upper part of the organ was directly adherent both

FIG. 5.



From Case 3. Malarial cirrhosis with very great enlargement of the spleen. GS, Granular surface of the liver well marked. D, Diaphragmatic adhesions to the spleen. S, Stomach. The pylorus is approximated to the cardiac end.

to the diaphragm and the lower surface of the left lobe of the liver. The spleen capsule showed the usual irregular patches of ivory-white thickening.

To sum up, therefore, we have in malarial cirrhosis a complex condition of which the hepatic disease is the terminal event. Ascites is late and may be only slight. In the early stage the liver is a little enlarged and in the last stages the organ is not very small. The finely granular surface is quite distinct from hob-nailed liver. The cirrhosis results from repeated attacks of malarial hepatitis and capsulitis, associated with perisplenitis and a plastic peritonitis. The adhesions resulting from the latter, along with the dragging of the enlarged spleen, produce extreme deformities of the stomach. Plastic peritonitis varies in amount but is always a feature of the case. I have recently seen two instances of this affection in Professor Childe's wards, both children; in one there was a marked peritoneal friction rub over the spleen and in both the liver was enlarged. There are accompanying degenerative changes in the kidneys involving both the interstitial tissues and the renal epithelium, and this nephritis may accelerate the fatal issue.

Bombay.

## THE ACUTE SUFFOCATIVE CATARRH OF LAENNEC AND OTHER CONDITIONS FROM WHICH IT SHOULD BE DISTINGUISHED.<sup>1</sup>

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SUFFOCATIVE catarrh is a term often used in a vague way to describe cases which have the two features in common—pulmonary catarrh and suffocative dyspnoea. The name was invented by Laennec to denote an affection rare but characteristic enough. It is as little recognised, it would seem, in the present day as it was when Laennec first described it. The following is Laennec's original description, which can hardly be improved upon:—

The disease is an acute catarrh affecting the whole or a very large portion of the mucous membrane of the lungs. The duration is from 24 to 48 hours, or at the most some days. At the end of that time either the patient dies or expectoration commences and puts an end to the suffocation, and the disorder then follows the course of a simple

<sup>1</sup> A paper read before the Medical Section of the Royal Society of Medicine on Feb. 25th, 1903.

acute catarrh. While the suffocation lasts there is but little cough and the expectoration, if any, is altogether pituitous or fluid. It retains this character for some days at least and then becomes more abundant; recovery sometimes takes place without its ever becoming properly mucous, in which case the disease is only a variety of the acute bronchial phlegmorrhage or pituitous catarrh. When, on the other hand, the expectoration becomes mucous the disease is simply an ordinary acute catarrh in which the suffocative character of the invasion is caused by the extent of tumefaction of the bronchial membrane and by the large quantity of fluid excreted at once.

Laennec further states that it is very rare in adults and for this reason had escaped the attention of physicians. In fatal cases the necropsy shows little morbid change. The lungs are somewhat congested and the tubes contain more or less (often only a very small amount) of frothy fluid. Laennec's opinion seems to be the correct one, that the urgent symptoms are due to the rapid tumefaction of the mucous membrane of the medium- and small-sized bronchi.

It is interesting to remember that similar symptoms may arise as the result of the inhalation of violently irritant vapours, such as bromine and iodine.

The following is a typical instance of the affection and the bacteriological find suggests that in some instances, at any rate, the exciting cause of the irritation may be a bacillus.

**CASE 1. Acute suffocative catarrh associated with peculiar bacilli of indefinite nature (diphtheroid).—**On Oct. 24th a young man, aged 23 years, was admitted into hospital deeply cyanosed and suffering from great dyspnoea. It was found that he was a strong man and had been in perfect health until about 36 hours before, when he felt some tightness and constriction round his chest and his breathing became short. He struggled on with his work for one day and during the next night became so much worse that he was brought to the hospital in the early morning and admitted at once as an urgent case. On examination in the wards, in spite of the extreme dyspnoea and cyanosis, which were so severe that the patient seemed in imminent risk of suffocation, no physical signs could be found in the chest except rhonchus and sibilus. The respirations were 32, the temperature was 101.2° F., and the pulse was 104, falling next day to 98° and 80 respectively.

The clinical condition was altogether unlike that of pneumonia, nor was it or the respiration like that of asthma. There was no laryngeal or tracheal obstruction. The case suggested most that form of acute miliary tuberculosis in which the lung is stuffed with tiny tubercles and where the physical signs bear no relation to the general distress and dyspnoea. Such diagnosis, however, was not consistent with the history obtained of the attack in the present case.

The usual remedies were applied and the patient improved rapidly, so that by the next day he was out of danger though still suffering from shortness of breath. The temperature had fallen to 97.8° and the pulse to 80. The cyanosis was still well marked. Crepitation appeared in the chest and a little mucoid sputum was coughed up. On the next day (Oct. 26th) the sputum was more abundant. I had a bacteriological examination of it made and the report was: "A few pneumococci but very large numbers of diphtheroid bacilli of uncertain nature. Cultivation yielded nothing but a coliform bacillus."

From this time the chest began to improve. Moist sounds became more abundant; the expectoration increased but was never more than scanty; and the symptoms, dyspnoea and cyanosis, became rapidly less. In about 48 hours from the time of admission all very urgent symptoms had passed away and nothing remained but what might be called slight bronchitis. The patient did not, however, convalesce as rapidly as might have been expected. The physical signs did not clear up completely and an amount of prostration and feebleness continued which was altogether out of proportion to the apparent mildness of the bronchitis. The sputum was again examined bacteriologically on Nov. 22nd—i.e., just a month from the onset of the illness—and still contained the same bacilli. The report was: "Pneumococci present and bacilli coli communis both in small numbers. Gram-positive bacilli, diphtheroid in appearance, were still numerous." Ultimately the patient made a good recovery and was sent home.

The case recorded tallies in all points with Laennec's description. The only new fact is its association with the peculiar diphtheroid bacilli. As similar cases occur in association with the pneumococcus, the question may fairly be raised whether the acute symptoms do not depend upon the bacilli and their wide dissemination through the bronchial tubes.

The following conditions are such as are either likely to

be confused with the true suffocative catarrh of Laennec or else present interesting relations with it. I. Acute suffocative pulmonary oedema, or, as it has been termed, acute non-inflammatory congestion of the lungs. II. Certain acute inflammatory conditions: (1) capillary bronchitis; (2) secondary broncho-pneumonia; (3) acute congestive or, as I should call it, primary broncho-pneumonia; and (4) the early congestive stage of acute pneumonia. III. Lastly, collateral fluxion and physiological breakdown or respiratory failure are conditions which stand in an interesting relation with it.

#### I.—Acute Suffocative Pulmonary Oedema.

The acute congestion is in these cases due to sudden failure of the heart and the group falls naturally into two divisions, according as there has been antecedent morbus cordis or not.

1. *With antecedent morbus cordis.*—The simplest instance to take is a case of mitral disease. Here, owing to the obstruction at the mitral orifice the lungs are always pathologically congested—i.e., contain more blood than they normally should. When from any cause, and it may only be a very slight cause, the obstruction is in any way increased exudation will take place from the vessels into the lung and will give evidence of its appearance by wheezing and crepitation—i.e., by the signs of bronchitis. In most instances the extra obstruction is slight and the signs of bronchitis are not severe. But the gravity of the symptoms depends upon the degree of extra obstruction and the rapidity of its development, and when these are great the symptoms will be urgent even to the extent of suffocation. Of this the two following cases are good illustrations, the first occurring in the course of disease of the mitral valves and the second of the aortic valves.

**CASE 2. Acute oedema of the lungs in a case of chronic mitral disease; death in 17 hours; necropsy.**—A boy, aged 12 years, a strong and healthy child, went to bed well but woke up an hour later crying with pain in the pit of the stomach and bringing up some "congested blood" from the mouth. He was taken at once to the hospital, spitting up blood on the way and vomiting several times. When seen he was found to be much cyanosed, suffering from great dyspnoea, gasping for breath, constantly coughing up bright red frothy blood, and groaning with pain over the præcordium. The pulse was 120, the respiration very rapid, and the temperature 96° F. Loud coarse crepitation was heard all over the chest but there was no dulness to percussion or bronchial breathing. The apex of the heart was displaced two inches outwards, the cardiac dulness increased upwards and to the right, and a loud presystolic murmur was audible. During the night the symptoms grew steadily worse. The spitting of blood continued and the lad died from suffocation 17 hours from the onset of his illness. At the necropsy the heart showed well-marked mitral stenosis. The lungs were extremely congested and oedematous and the air-tubes contained much blood-stained fluid like that which had been expectorated. Microscopical examination showed the ordinary appearances of the heart-lung but in addition the alveoli and small bronchi were filled with a turbid coagulable fluid, containing numerous cells both epithelial and lymphatic as well as blood.<sup>2</sup>

**CASE 3. Acute oedema of the lungs due to sudden failure of the left ventricle in a case of double aortic disease; free venesection; recovery.**—A man, aged 40 years, was admitted into hospital with double aortic disease, &c. All at once extreme dyspnoea set in with rapidly increasing cyanosis. Wheezing and crepitation developed over the lungs and the patient began to spit up blood-stained sputum which soon became almost pure blood. The symptoms were so urgent and suffocation so threatening that venesection was performed and 30 ounces or more of blood rapidly removed from the arm. As the blood flowed the colour improved and in an hour or two all urgent symptoms had passed off, the patient recovered from the attack and lived for more than three months, dying at last from gradual heart failure. The amount of dyspnoea and its sudden onset might have been explained by infarct. But the rapidity with which the symptoms subsided after the bleeding proved the diagnosis of acute congestion due to sudden failure of the left ventricle to be the correct one. The venesection no doubt saved the patient's life for the time.

2. *Without antecedent morbus cordis.*—Acute oedema of

<sup>2</sup> For a similar case under the care of Dr. J. Lindsay Steven see THE LANCET, Jan. 11th, 1902, p. 72; also other writers in THE LANCET of 1907.

the lungs consequent upon sudden cardiac failure is the actual cause of death in many cases of hyperpyrexia and of grave septic fevers, especially with high temperatures, such as typhus or malignant scarlet fever.

CASE 4. *Acute pulmonary oedema in a case of hyperpyrexia; cold baths; temporary relief; death.*—A man, aged 36 years, was the subject of an ordinary attack of rheumatic fever. After a few days, when he was apparently convalescent, the temperature began to rise and rapidly it reached 108° F. After about three hours or so I saw the patient and found him quite unconscious, with noisy rattling all over the chest, and apparently moribund. He was at once placed in a cold bath. After a few minutes he came to himself so that he could answer questions. The heart became several beats slower and the noisy rattling over the chest disappeared. He was kept 20 minutes in the bath when he complained of feeling cold and was transferred to bed. The temperature rose again in an hour and the rattling returned. Again he was put into the cold bath and the rattling disappeared. He had several baths with the same result, but in the end died from exhaustion.

## II.—*Acute Inflammatory Congestion.*

It is among the inflammatory group of congestions that the other affections are found which are more commonly confounded with suffocative catarrh. *Capillary bronchitis* and the *secondary broncho-pneumonia*, though often attended by severe dyspnoea and cyanosis, are usually so evidently consecutive to a bronchitis as to give rise to little real confusion, though when such cases end with suffocation they have been called suffocative catarrh. But the distinction is clear, for the attack begins as an ordinary bronchitis which spreads gradually with more or less rapidity to the smaller tubes, the symptoms increasing in severity as the disease progresses and death ensues as much by heart failure as by suffocation. It is the other form of broncho-pneumonia, the acute primary, presumably pneumococcal, form, or as it has been called *acute congestive broncho pneumonia*, that is more closely allied to suffocative catarrh. There is also probably a form of *pneumococcal bronchitis* of great intensity and mortality and, as the case which forms the basis of this paper suggests, acute suffocative catarrh may really be an acute bacterial bronchitis of pneumococcal and some other bacterial origin. The onset in both these affections is very sudden, as in other pneumococcal inflammations, and if the affection be general or widespread the symptoms may be very severe and well deserve the name of suffocative catarrh. Indeed, it is to this group that I am inclined to refer many of the cases of Laennec's acute suffocative catarrh.

Finally, in this connexion a remarkable group of cases deserves mention in which pneumonia begins in a very peculiar way—*diffuse congestion-stage of acute pneumonia*. Dyspnoea is urgent from the onset and examination shows widespread congestion of one lung with possibly secondary congestion of the other. If death happened now the case might be not incorrectly described as suffocative catarrh. At first there is no expectoration but after an hour or two it may appear and be more or less blood-stained. Sometimes there is so much blood that the attack might be described as commencing with acute hæmoptysis. If life be preserved the local lesions develop and as one lobe becomes consolidated the congestion of the rest of the lung passes off and the hæmorrhage ceases. The condition in the lungs may be compared with what is sometimes observed in a furuncular inflammation of the arm, for instance. It may set in with an inflammatory oedema which rapidly involves a wide area, for example, the whole forearm from knuckle to elbow, though the boil which ultimately forms may not be larger than a shilling. And as the local inflammation develops the widespread inflammation and oedema subside. The following is a case of the kind.

CASE 5.—A woman, aged 35 years, who had mitral stenosis of many years' duration, was attacked one evening with high fever and great dyspnoea. The dyspnoea was so severe that her life seemed in danger. The only physical signs were those of acute congestion of the whole right lung. She immediately began to expectorate pure blood, bright and frothy, and brought up several ounces in three or four hours with considerable relief. Gradually the signs of general congestion subsided and became limited to the top of the upper lobe, where the ordinary signs of acute pneumonia subsequently developed. In spite of the acute and alarming onset the pneumonia ran an ordinary course and terminated on the fifth day. The patient then made a rapid recovery.

A curious case of a similar kind came recently under my observation.

CASE 6.—A girl, aged nine years, was taken suddenly ill on Jan. 31st. I saw her on Feb. 4th, when she had a temperature of 104·8° F., a respiration-rate of 48, and a pulse-rate of 150. She was deeply cyanosed and so urgently ill that strychnine was injected subcutaneously, ether and brandy given by the mouth, and oxygen administered by inhalation. This treatment relieved her, but the cyanosis continued till the temperature fell by crisis on Feb. 5th. During the two days I had her under observation there were no physical signs in the chest of any definite kind. It was diagnosed on general principles as a deep-seated pneumonia. It was not until the second day after the crisis that the signs of consolidation appeared in a small but distinct patch below the left scapula. The child ultimately recovered, but for three days after the crisis did hardly anything but sleep and lay in a condition of great prostration. The degree of cyanosis with the absence of physical signs to account for it was puzzling in the extreme, but the only explanation seems to be that of extreme pulmonary congestion of unusual duration. Except for the physical signs which ultimately developed and the high fever the case was very like suffocative catarrh.

## III.—*Collateral Fluxion, Respiratory Failure.*

In close connexion with acute suffocative congestive may be also considered those cases in which the respiratory symptoms are due to collateral fluxion or pulmonary failure. The conditions are interesting though not likely to be in any way confounded with suffocative catarrh, as they are so obviously secondary to some other affection. When, as in the case of pleuritic effusion, the fluid forms gradually, so that time is given to the heart and lungs to accommodate themselves to the altered conditions, the opposite lung may for long remain equal to the extra work demanded of it. But the margin is small and may easily be overstepped. The extra work may easily pass into overwork, and so soon as this occurs the signs of bronchitis—i.e., of pulmonary failure—appear. Thus the appearance of the signs of bronchitis on the sound side is an indication for immediate paracentesis. A similar condition may arise in connexion with abdominal distension, e.g., in acute peritonitis, acute tympanites, or ascites, the lower parts of both lungs being then collapsed from the pressure of the diaphragm upwards, and so again dyspnoea may arise and quickly become urgent.

If, as in a case of pneumothorax, the heart and lungs have no time to adjust themselves the signs of respiratory failure appear at once, so that in a few minutes, or at any rate in an hour or two, death may occur through suffocation. In this connexion may be mentioned also those cases of acute pneumonia in which during the active stage of the disease the signs of bronchitis develop in the opposite lung. These signs indicate physiological failure and are of very grave significance, for as so little can be done to relieve them the condition becomes rapidly worse and worse till death ends the struggle.

In a bad case of pneumonia the respiratory failure is not a problem of such simplicity as has just been represented, for the respiratory and cardiac nerve centres, as well as the heart and respiratory muscles, are themselves affected by the high temperature and more especially by the pneumococcal toxin. But these lesions all work together towards the same end.

## *Conclusions.*

Laennec's suffocative catarrh is a peculiar and characteristic affection, rare especially in the adult, and not generally recognised as a clinical entity. It has to be distinguished from capillary bronchitis and disseminated, post-bronchitic—i.e., secondary—broncho-pneumonia; more closely resembling it are primary broncho-pneumonia—i.e., disseminated pneumococcal pneumonia and possibly an acute pneumococcal or other bacterial bronchitis. In association with it may be placed (1) cases of acute pulmonary oedema which develop in the course of chronic heart obstruction or of acute heart failure; (2) cases of collateral fluxion or of pulmonary failure; and (3) cases of pneumonia which commence with widespread pulmonary congestion. A careful bacteriological examination of the sputum should be made in all cases of suffocative catarrh as being likely to throw light upon the true nature of the affection.

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## BRIEF NOTES OF INTERESTING CASES OF CRANIAL SURGERY.

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A PAPER read at the Medical Graduates' College and Polyclinic, London, on March 31st last with the above title included the records of six patients under my care, arranged in four groups. A few brief notes in abstract of the longer communication will, I trust, be of interest to readers of THE LANCET.

*Group 1.*—The first group comprised two cases of depressed fracture of the skull with extra- and intra-dural effusion, one of which was compound and complicated with two separate and extensive fractures on the same side of the vertex. In the first of these the lesion was in the left parietal region, causing aphasia and right-sided hemiplegia, clearing up after removal of the pressure, at least 48 hours after the injury.

The second case contrasted with the other in that whilst there were two extensive depressed fractures there was practically no shock and no paralysis or functional disturbance, though the injuries were severe enough to cause considerable effusion. This was probably the result of the pressure being applied chiefly to the frontal lobe. A brief reference was made to the use of lumbar puncture, the cerebro-spinal fluid in extradural hæmorrhage being clear, whereas it is blood-stained in extradural hæmorrhage from cerebral laceration or torn sinus.

*Group 2.*—A case of gumma of the pericranium and sub-jacent left parietal bone, occurring at the seat of repeated injury and causing tenderness and severe headache of several years' duration. The growth was of about the size and shape of an adult human patella. Complete disappearance of the headache was noted on the day after removal by trephining, &c.; iodide was administered, and there was no subsequent recurrence.

The facts illustrated by this case were: (1) that though the spirochæte of syphilis produces a generalised infection, it shows a well-marked tendency to settle in certain tissues or organs, and, amongst bones, notably the skull; and (2) that exposure of these tissues or organs to injury by creating an area of still lower vitality than usual greatly increases their liability to gummatous deposit. This is comparable with the fact that no effect may be produced on inoculating an animal with staphylococcus from a virulent lesion in man until an area is met with or created where the resistance has been lowered, such as the site of a fracture.

Another instance of this was afforded in my lecture on Liver Abscesses at the Polyclinic last year. Dysentery led to amœbic abscesses of the liver rupturing through the lung, there being also an associated appendix abscess. As the result of free drainage recovery was progressing when the most profuse discharge set in, the prognosis becoming almost hopeless. Potassium iodide, pushed to 35 grains four times a day for 29 days before iodism occurred, resulted in rapid and permanent recovery by absorbing the gummata which were complicating the liver abscess.

In making a diagnosis the patient's occupation should always be borne in mind. In the two instances here alluded to both men had been in the army, and specific trouble may be aptly, if a little unkindly, included amongst the "Diseases of Occupation." Allusion was made to the danger of leaving alone any sequestrum resulting from necrosis of the outer table, perforation of the skull leading to fatal meningitis or cerebral abscess being recorded (see Fig. 84, Vol. I., Treves's "System of Surgery").

*Groups 3 and 4.*—The last two groups of cases comprised (a) a case of suppurative mastoiditis and cerebral abscess in which the cardinal symptoms of abscess were completely masked by those due to thrombosis of the bulb of the jugular vein, and there was no optic neuritis; and (b) two cases of pneumococcic meningitis, one supervening upon pneumonia, the other resulting from infection from the tonsils, lateral sinus thrombosis preceding the meningitis, a noticeable feature being absence throughout of rigors.

A brief recital of the cardinal symptoms of the chief complications of middle-ear and mastoid disease followed and it was seen that absence of optic neuritis is of no value in

excluding cerebral or cerebellar abscess, that rigors are highly significant of thrombosis, which if not present in the lateral sinus itself will be found in the bulb of the jugular vein, though, again, the absence of rigors does not negative sinus thrombosis, as was illustrated in one of the present cases. High leucocytosis and high percentage of polymorphonuclears when present are of supreme importance as evidence of sepsis generally, if not of intracranial sepsis; in cerebral abscess, other signs pointing that way, 80 per cent. polymorphonuclears and over would be diagnostic (Potts). It is of equal value for the same reason in lateral sinus pyæmia, but was absent in the second of the two cases of pneumococcic meningitis and thrombosis, the blood count being only 10,000 per cubic millimetre (5,000 to 10,000 leucocytes being within the normal range). The ideal treatment of the mastoid-cerebral abscess case where the symptoms were masked was outlined.

In conclusion, more active treatment of meningitis, septic and tuberculous, was urged, the diagnostic and therapeutic value of lumbar puncture being reviewed, and recent suggestions for the treatment of these otherwise generally fatal cases were summarised. These were: 1. Early bilateral drainage of the skull, perhaps with counter-opening of the lumbar spinal canal, to permit of continuous irrigation (Ballance). 2. Repeated lumbar puncture (Barker). 3. (Author's suggestion.) The use of appropriate vaccines, combined with lumbar puncture where necessary, either for diagnosis or to obtain material for the vaccine, or for the relief of urgent pressure or other symptoms. When the indication otherwise is clear, if lumbar puncture is negative, trephining would be justifiable, with a view to confirming the diagnosis, relieving the urgent symptoms, such as headache and optic neuritis, and securing material for vaccine treatment, if not otherwise obtainable.

With more active attempts at treatment of this kind, the writer hopefully anticipates the steady reduction in the mortality from meningitis and also a vast improvement in the prognosis in other diseases, in which so often it has hitherto proved the fatal complication.

## A METHOD OF SUTURING THE LATERAL RECTI TO INSURE GREATER MOBILITY OF THE STUMP AFTER ENUCLEATION OF THE EYEBALL.

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AT the meeting of the British Medical Association in 1902 I read a paper on this subject illustrating the manner in which I recommended the operation to be performed. I have had considerable further experience of the great value of this method of removing the eye and have, moreover, improved on my original method.

As everyone knows, the method of enucleation mostly adopted and found described in almost every text-book is the simple and, it seems to me, careless plan of merely dividing the muscles and relying entirely on the attachment of these muscles to Tenon's capsule for the mobility of the stump. Fisher says: "After a well-performed excision the anterior extension of Tenon's capsule still remains intimately adherent to the ocular conjunctiva and so enables the recti muscles to exercise their action upon the conjunctival socket and thus to produce the movements of the artificial eye."<sup>1</sup> This, of course, is true, but the lateral movement of the stump resulting is nothing like so extensive as when these muscles are attached to the conjunctiva well over the middle line.

The method is as follows. The conjunctiva is opened all round the sclero-corneal margin and freely separated from the globe. The two lateral recti are then separately hooked up and thoroughly isolated from their attachments, clamped with a Prince's forceps, and finally divided close to the eyeball; the other muscles are divided close to the eyeball and the globe removed in the ordinary way, but especial care is taken to remove the eye as *cleanly* as possible. One of the lateral recti is then sutured with catgut to the lower lip of the conjunctival opening on the opposite side and the other