PRIMARY BRONCHOPNEUMONIA

IN ADULTS.

A THESIS

For M.D., Edinburgh University,

by

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PRIMARY BRONCHOPNEUMONIA IN ADULTS.

The literature on the subject of Bronchopneumonia is very silent regarding the occurrence of primary forms, particularly in adults.

In the majority of the books which deal with it, bronchopneumonia is described as a disease secondary in its origin, and most commonly secondary to bronchitis, chiefly affecting persons in the early and late years of life, and especially children. One author, Aufrecht, in Nothnagel's "Practice of Medicine", affirms that it is almost exclusively a disease of children, and that, putting out of account cases of deglutition and aspiration pneumonia, he has never seen characteristic forms of catarrhal pneumonia in adults. He, however, notes the occurrence, in some cases of fibrinous pneumonia, of pain, pleurisy and mucous rales on the apparently healthy side, and considers that there the pneumonia process fails to attain its full development. The cases described are presumably adults, but the undeveloped pneumatic process on the apparently healthy side would not appear to be a typical fibrinous one.

Primary bronchopneumonia is described very fully by West as occurring in children. Contrasting the condition with the secondary form, he draws the conclusion that primary and secondary forms are of different origin bacteriologically, secondary forms being chiefly
chiefly of streptococcal, and primary forms of pneumococcal origin, and that the pneumococcal inflammation occurs with equal frequency in the adult and the child but takes a different course in each, (in the adult a massive and in the child a disseminated consolidation) He maintains that there is no real pathogenic distinction between the two. He does not, however, refer to the primary lobular condition as occurring in adults.

Osler also describes a primary form of bronchopneumonia in children, associated with the presence of the pneumococcus, and other authors, (Munro, Lockwood, and Kidd in Bain's "Text book of Medical Practice;") mention the occurrence of a primary form, but in children only.

On the other hand, Anders, in his "Text book of the Practice of Medicine" describes two clinical forms, primary and secondary, and states that the primary form is met with generally in adults, with the symptoms of a severe form of acute bronchitis.

Gibson, also, in his "Text book of Medicine", admits the occurrence of a primary bronchopneumonia of adults, and cites cases coming under his own observation in workman on the Forth Bridge. He maintains, however, that such cases are not frequent.

In this paper I will endeavor to show that the disease known as "Catarrhal pneumonia", "Lobular pneumonia", "Capillary bronchitis", and "Bronchopneumonia" is by no means uncommon as a primary disease in adults that it occurs chiefly in weakly persons, that it has different varieties, both pathologically and clinically, that
that these varieties depend chiefly upon the virulence and distribution of the infective agent, and that the infective agent in primary bronchopneumonia is the same as in primary fibrinous pneumonia, namely the pneumococcus, with occasionally a mixed infection with the streptococcus.

I would urge that primary Bronchopneumonia should be classed along with fibrinous pneumonia, and be kept distinct from the secondary forms of bronchopneumonia, as it undoubtedly differs from it in its etiology.

Primary Pneumonia would thus be capable of being described as an infective disease, due to the pneumococcus, affecting persons of all ages, and presenting grave constitutional symptoms, with signs pointing to the local seat of infection, and showing both lobar and lobular implication of the lung.

That young children are more liable to the lobular form may be explained by the more distinctly lobular arrangement of the lung in the child, the minuteness of its bronchioles, and its more limited power of expelling matters entering its respiratory tract.

Within a short time after my first arrival in India I was impressed with the numbers of cases of catarrhal pneumonia which presented themselves.

In 1893 I was serving on the North West Frontier hills in charge of detachments of Native Indian troops, and was then struck with the character of many of the cases of pneumonia among the sepoys. Again in 1895, in the Camp at Wana after the Waziristan-Expedition, during which I was attached to a Native Field
Field Hospital, we had a severe epidemic of pneumonia among the troops, and again it was noticable that in a large number of cases, though the onset and the symptoms were very similar to those of fibrinous pneumonia, the physical signs in the lungs were typical of lobular pneumonia.

In cantonments, as in the field, the cases of serious illness among soldiers are at once brought to the notice of the Medical Officer by the unfitness of the sick man for duty, consequently the cases are seen from the first.

In my experience as Medical Officer of a regiment, a proportion of the pneumonia cases every year presented the characters of bronchopneumonia. In some cases, too, of fibrinous pneumonia I have detected in the apparently healthy lung fine rhonchi and crepitations, and sometimes pleuritic friction, occasionally developing into the formation of a small area of consolidation, a condition not unlike that remarked on by Aufrecht, in which it is fair to assume that the infection on both sides was a similar one but different only in degree.

Of these cases, however, I regret that I possess no record, and am thus able to speak of them merely in a general fashion. I will simply observe that all these cases were adults, in the prime of life for the most part, though they might be weakened by privations, fatigue, and exposure to a climate exceedingly cold in the winter months.

Since I came to Lahore, I have had the opportunity,
as Second Physician to the Mayo Hospital, of observing a large number of cases of pneumonia in the Wards, and as Pathologist I have also been able to see a certain number of these cases post mortem; and it is from this later experience, rather than from that previously mentioned, that I have formed an opinion regarding the frequent existence of bronchopneumonia among adults as a primary affection. I have also been able to make bacteriological examinations of the sputum in a large number of the cases, and in a few to observe the pathological lesions after death.

Owing, however, to sentimental and religious objections, post mortem examinations cannot always be obtained, and thus it often happens that the observations begun in the wards cannot be completed when the case terminates fatally.

The following statement of cases at the Mayo Hospital, Lahore, will serve to illustrate my remarks, and will, I venture to think, go far to prove my contention.

The first statement is a list of the cases of and deaths from fibrinous pneumonia and bronchopneumonia in the three years 1903, 1904, and 1905; and the second gives a detailed list of the cases of Primary Bronchopneumonia in the years 1904 and 1905.
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Cases of Primary Bronchopneumonia in the Mayo Hospital in 1904 and 1905.

1904.

   History of dysentery and malaria-Last attack of malaria 2 months previous to admission, severe, lasted 1 month.
   Onset with rigor-both lungs affected-sputum mucous-temperature showed remissions-
   Termination by lysis-Normal on 11th day.

   History of syphilis and malarial attacks.
   Previous chill-onset gradual-both lungs affected-pseudolobar involvement of right upper lobe-pleurisy-Died 21st day.

   History of malarial attacks-dysentery two months previous to admission.
   Onset with rigor-both lungs affected, left especially-dyspnoea marked-sputum mucous-
   Termination by rapid lysis-temperature normal on 10th day.

   History of malarial attacks.
   Onset with rigor-Both lungs affected-dys-
fnoea-no expectoration-Remissions in
temperature-Removed by friends.

History of several attacks of malaria,
dysentery, and diarrhoea.
Onset with rigor-Both lungs-sputum
slightly "rusty"-no micro-organisms
found-Recovery by crisis on 8th day.

History of malarial fever 1 month pre-
vious to admission, weak, with headache,
constipation and anaemia.
Onset rapid-no rigor-Both lungs affect-
ed, especially right-no sputum.
Recovery by lysis-Normal 14th day.

7.4th May. Nathu-23 years-Male-Mahomedan-Sweeper.
History of occasional attacks of malaria.
Onset with sudden fever-no rigor-both
lungs-sputum mucous-contained pneumo-
occi-lysis-normal 15th day.

8.17th May. L. Viegos-12-Male-Eurasian-Schoolboy.
History of malarial attacks occasional-
ly.
Onset gradual-continued fever with re-
missions-sputum mucous-no organisms
found-both lungs-lysis-normal 23rd day.

History of middle ear and mastoid
trouble.
No rigor—both lungs affected—constitutional symptoms severe—Pneumococci in sputum—Removed by friends.

10.24th May. Mrs Shea—26 years—Female—Eurasian.
History of malaria for 2 years, almost continuous—confined 1903—child born dead.

No rigor—Right lung especially involved—sputum slightly "rusty"—contained pneumococci—Termination by lysis (rapid)—Normal 9th day.

11.14th June. Mr. Martindale—32 years—Male—European.
Fever for 3 days preceding lung symptoms—Both lungs, especially left lower lobe (pseudolobar)—sputum mucous—lysis—Normal 23rd day.

12.23rd June. Kalka Prasad—22 years—Male—Hindu—Student
History of occasional malaria.
Fever 5 days before lung symptoms—Both lungs—severe left pleurisy—no consolidated area—sputum rusty—Pneumococci—Crisis 12th day.

History of occasional diarrhoea—disease supervened after operation for circumcision—Removed by friends—incomplete recovery.

History of syphilis—malaria off and on
for 2 years previous to admission-diarrhoea-
Onset with rigor-Both lungs-right lower lobe consolidated-sputum mucous and slightly "rusty"-contained pneumococci-Termination by lysis-normal 10th day.

15.2nd Nov. Ruk ul Din.14-Male-Mahomedan-Student.
History of malaria and diarrhoea.
Onset with rigor-constitutional symptoms severe-lung symptoms slight-both lungs affected-sputum mucous-pneumococci-lysis-normal on 42nd day.

Malaria in childhood-no recent history of disease.
Slight rigor-Both lungs-consolidated area in right upper lobe-sputum scanty, mucous-pneumococci present-Termination by lysis-normal 19th day.

17.24th Nov. Lalta Pershad-16-Male-Hindu-Compositor.
History of several attacks of malaria-diarrhoea.
Rigor-consolidated patches in both lungs-sputum mucous-contained pneumococci-lysis-temperature normal on 21st day.

History of chill-was out of sorts for a fortnight-took cold bath on advice of friends-followed by rigor-some tonsilitis-both lungs affected-dyspnœa-constitutional
13.

constitutional symptoms severe-sputum mucous-contained pneumococci. Died 18th day.


History of frequent malaria, dysentery, and diarrhoea.

Chill-Both lungs affected-no area of consolidation-sputum frothy mucous and mucopurulent-no organisms found-Recovery by crisis on 9th day.

History of malarial fever every year.
Onset sudden-no rigor-both lungs-diffuse-sputum mucous, becoming mucopurulent-
Pneumococci and streptococci in sputum-
Temperature showed remissions-Recovery by crisis on 15th day.


History of malarial fever every year in autumn months-tertian and quartan fever lasting 2 or 3 weeks-
Rigor while bathing-high fever-both lungs affected-consolidation at inferior angle of right scapula-dyspnoea-sputum mucous and rusty-pneumococci-Termination by rapid lysis-normal on 10th day.


History of malaria and dysentery several times.
Rigor-both lungs-sputum mucous becoming mucopurulent-containing pneumococci-Death on 18th day after small crisis.


History of malaria and dysentery occasion-ally.
Onset gradual, following previous attack of malarial fever-both lungs-right middle lobe consolidated-sputum mucous becoming
becoming mucopurulent—Pneumococci, streptococci, staphylococci—Death with high temperature on 13th day—(Pleurisy left side.)


History of malaria frequently—2 severe attacks—one recent.

Onset sudden, with chill after exposure to wet—both lungs—left upper lobe consolidated—dyspnoea—lysis—temperature normal on 12th day—no micro-organisms found in sputum.


History of frequent malarial attacks.

Onset sudden—both lungs—solid patch in right middle lobe—sputum not obtained—remissions in temperature—Crisis on 18th day.


History of several attacks of malaria.

Onset with rigor—both lungs affected—diffuse—dry pleurisy both sides in mammary areas—sputum rusty—lysis—incomplete recovery—temperature not normal on 19th day.


History of malaria, dysentery and diarrhoea several times.

Rigor—both lungs—physical signs marked—no rusty sputum—pneumococci—Recovery by lysis—normal 8th day.


No history of malaria or other disease—Condition supervened after an injury—
16.
Sudden onset—both lungs—both upper lobes dull on percussion—sputum mucous, becoming mucopurulent—no microorganisms—Remissions in temperature—lysis—normal 21st day.

History of occasional mild attacks of malaria—
Onset with rigor (chill in bathing after long and hard day’s work without food)—both lungs—diffuse—sputum mucous becoming mucopurulent—Pneumococci—Recovery by lysis—normal on 11th day.

No history of illness.
Mild case—both lungs—no sputum—lysis normal temperature on 20th day.

Weakly man—fistula in ano for one year previously—chronic nephritis.
Gradual onset—both lungs—sputum "rusty" some blood—Pneumococci—no tubercle bacilli—Removed by friends on 20th day.

History of syphilis—malaria, dysentery and diarrhoea frequently.
Onset with rigor—both lungs affected—Pleurisy with effusion left side—con-
consolidation in posterior border of left lung-sputum rusty- no organisms-termination by lysis, rapid-Normal 19th day.

No history of malaria or other disease-
Both lungs affected-fever severe-dyspnoea-
Pneumococci and diplococci in sputum-saline (0.75% solution) injections on 2 occasions-
Termination by lysis-normal 34th day.

34. 19th June. Miss C. George-10-Female-Eurasian-
Both lungs-not severe-sputum mucous be-
coming mucopurulent-no organisms-Termina-
tion by lysis-Normal 23rd day.

History of occasional malaria.
Onset sudden-severe-sputum mucous-
Pneumococci-Died, 7th day.

History of malaria and dysentery.
Both lungs-right upper lobe consolidat-
ed-no organisms in sputum-lysis-Normal 18th day.

History of frequent attacks of malaria and dysentery.
Sudden onset-both lungs-sputum mucous-
dry pleurisy-lysis-normal on 27th day.

History of malaria and diarrhoea occasionally
18.

occasionally.
Onset sudden, both lungs, diffuse, especially right-severe-sputum mucous—Pneumococci—
Termination by lysis—Normal 11th day.

History of frequent dysentry and diarrhoea.
Onset sudden—severe—dyspnoea—both lungs—especially left—sputum rusty—pneumococci—
lysis—Normal 12th day.

Onset gradual—both sides—right upper lobe
became gradually consolidated—sputum rusty—
pneumococci, streptococci, and staphylococci—dyspnoea—weak heart—Died on 13th day,
after crisis.

41. 10th Dec. Jhanda—50—Male—Sweeper.
History of malaria and dysentery—was in
hospital with gonorrhoea—
Onset sudden, with chilliness—no rigor—
fever 11 days before lung symptoms de-
veloped—sputum mucous—containing pneumo-
cocci—recovery by lysis—Normal tempera-
ture on the 14th day.

History of one attack only of malarial
fever.
Onset with rigor—cough, pain, dyspnoea—both
lungs—consolidated area in right middle
lobe—sputum mucous, becoming mucopurulent—
Pneumococci present—lysis—temperature nor-
mal on 12th day.

History of frequent malarial attacks—occasional dysentery—
Onset with rigor—both lungs—consolidated
patch at inferior angle left scapula—
sputum rusty—rapid lysis—Normal temperature on 4th day.
ETIOLOGY.

In these 43 cases quoted from the Mayo Hospital records for 1904 and 1905, pneumococci were discovered in the sputum in 23 cases, a proportion of 53 percent, and out of this number pure pneumococcal infection was found in 19, and mixed infection in 4 cases. Even if an error existed in diagnosing the cases with mixed infection as primary, a proportion of 44 percent remains in which the only bacteriological causative factor would appear to be the pneumococcus.

It will be noticed that with very few exceptions all the cases were adults, and chiefly males. Neither women nor children form any large proportion of indoor patients in Native Indian Hospitals, and the fact that records of primary bronchopneumonia among them are wanting is due to the customs of the people rather than to sex and age incidence of the disease. Women in the East lead a very secluded and domestic life and do not like the conditions of existence in a hospital, and young children cannot well remain in hospital without their mothers, and, though they may be brought to the out door department for advice, are chiefly treated at home.

Aged persons may be affected by bronchopneumonia, but the disease in them is more often secondary to bronchitis, and consequently the recorded cases show few old people.

Considering that the majority of the poor in India are
are insufficiently fed, badly clothed, and wose housed, and that their surroundings are, generally speaking, distinctly insanitary, it may be accepted that probably starvation, exposure, and bad hygiene are important predisposing factors in the etiology of an infective disease like pneumonia.

The climate of the Punjab, of which Lahore is the capital, shows great variation in day and night temperatures in the latter part of September, in October, November, February, and the first half of March; while December and January are bitterly cold as a rule. In the hot weather, commencing with April, the plains are scourged by periodical dust storms which fill the atmosphere with a fine, almost impalpable, certainly microbe-laden, dust. These usually cease with the advent of the monsoon rain storms in July; but in dry years they continue till rain sends relief. At all times also, except after rain, the dust on the roads and footpaths lies thick, and is thrown into the air by the incessant traffic of foot passengers, vehicles, and cattle, always to be found in the neighborhood of cities and large villages.

Natives herd together in small and insanitary dwellings, seldom or never cleaned out, from which ventilation is excluded as much as possible; they sleep with their heads invariably covered in their meagre blankets; as a practice they rise before daylight, even on chilly winter mornings, for purposes of nature; and, especially when sick, they expectorate freely about the walls and floors of their habitations.
It is small wonder, therefore, that the pneumococcus, living in surroundings so favorable for its development, attacks persons whose vitality is lowered by insufficient food, bad air, chills, bowel complaints, and that great scourge of the autumn months, malaria. In nearly all of the cases I have recorded, it will be observed that some history of debilitating disease was obtained, particularly malaria, dysentery, and diarrhoea.

The infective agent and a suitable soil are thus provided.

PATHOLOGY.

The pneumococcus is inhaled into the respiratory tract in small or in large quantity, either "filtered," as in nasal, or unfiltered, as in mouth breathing, and either alone or in company with other organisms.

It may be got rid of by what Stöhr describes as the "excretory division" of the lung, (the trachea, bronchi and bronchioles), without causing injury there, being either discharged with the ordinary expectoration or absorbed by the bronchial lymphatics.

On the other hand it may obtain a lodgement in the "respiratory division" of the lung, (the terminal or respiratory bronchioles, alveolar ducts, and alveoli.)

Here the condition set up becomes one of degree, (the effects depending on the virulence of the infective agent, the resistance of the victim, the numbers of the pneumococci present, and the limits of their distribution...
distribution in the organ), and is evidenced by varying degrees of general constitutional derangement dependent on the toxins absorbed, and of local inflammatory reaction dependent on the presence of the organisms themselves.

In primary bronchopneumonia the pneumococcus obtains a lodgement in the terminal bronchioles, and invades the mucosa, producing toxins. This results in an inflammatory reaction on the part of the mucous lining. This reaction is catarrhal in nature, and consists in hyperaemia of the capillaries in the wall, swelling and desquamation of the lining epithelium, with increased secretion of viscid mucus, and exudation of serum and leukocytes from the congested vessels, filling the lumen with greyish white or mucopurulent secretion.

The catarrhal exudation and the swelling of the mucosa lead to a certain amount of occlusion of the lumina of the terminal bronchiole and alveolar ducts, intensified by inspiratory effort, with the result that collapse of the lobule connected with the occluded duct very frequently takes place. The air in the collapsed lobule becomes absorbed, but it may remain capable of being inflated with air for some little time, surrounded by hyperaemic, oedematous, and emphysematous neighboring lobules. Later the lobule becomes the seat of consolidation. This may be due to an inflammatory reaction taking place due to infection having occurred previous to the collapse, or to an extension of the inflammatory
inflammatory process from neighboring lobules as described further on.

The catarrhal condition of the terminal bronchiole, above described, may be confined to that bronchiole, (bronchiolitis, capillary bronchitis) or the alveoli may become affected as well (alveolitis). The pneumococci may enter the alveolar ducts and reach the alveoli by extension from the affected respiratory bronchiole, or the infection of bronchiole and alveoli may be simultaneous, (bronchiolo-alveolitis.)

The local reaction to the irritation of the toxins produced by the pneumococci takes the form of a catarrhal inflammation of the affected alveoli, both in those originally attacked and in those contiguous to them. The capillaries in their walls become engorged with blood, and the air vesicles become filled with an exudate, consisting of swollen and shed epithelial cells, fluid which may sometimes show a few threads of fibrin, red blood corpuscles, and leukocytes, which have found their way out of the engorged vessels in the effort of nature to combat the local invasion.

This condition extends to the alveoli of the entire lobule.

The lobule, thus filled with exudation, becomes more or less consolidated; though air is not always entirely driven out, and the communication with the air in the bronchi may be kept up.

The number of lobules thus affected will naturally depend on the distribution of the pneumococci.

Thus
Thus it may happen that either a small or a large number of individual lobules, scattered throughout both lungs, is involved, constituting a "disseminated" or "diffuse" form of bronchopneumonia.

In other cases, larger areas of lung tissue, comprising several lobules, are implicated. Here an extension by contiguity of tissue takes place. The pneumococci find their way into the interlobular septa, and with their toxins affect the walls of the alveoli of neighboring lobules, causing an inflammatory reaction there similar to that in the originally affected lobule. A like result is produced by lateral extension of the infective process from the walls of an infected terminal bronchiole in "capillary bronchitis."

The size of the consolidated areas will depend on the virulence of the micro-organisms and their toxins and the distribution of the pneumococci in greater or less numbers in that particular area.

All degrees of consolidation may be seen, from one affecting only a few lobules to one in which nearly all the lobules in one lobe of the lung are involved. As a rule, even in this "pseudolobar" form, the entire lobe is not equally affected, as areas of hyperaemia, oedema, emphysema, and collapse lie between and among the areas of consolidation.

These areas of consolidation, of a greyish red color, can be distinctly seen. The collapsed areas are darker than the rest, and on the surface of the lung
are depressed. The emphysematous lobules are rather pale and prominent on the lung surface and the congested areas are a brighter red. The lung is mottled in color, engorged with blood, and moist on section; and a catarrhal exudate can be pressed out of the tiny bronchi in the middle of the consolidated patches.

The differences in the forms of bronchopneumonia above described are merely in the extent and degree of the inflammatory reaction. It may be said that the local reaction is proportionate to the local stimulus, and its object and effect is to check the further progress of the invading pneumococci.

Where the pneumococci, either alone or in company with other organisms, reach the superficial lobules of the lung, they come into close relation to the pleura, both by contiguity and from the nature of the lymphatic connections there, so that either the organisms themselves, or their toxins, find their way into the subpleural plexus of lymphatics, and thence, by the stomata between the pulmonary pleural cells, into the pleural sac. The tendency to proceed outwards into the pleural sac is naturally increased by the direction of the air pressure from the lung in inspiratory expansion of the thoracic wall.

The effect of the irritation produced is to bring about an inflammatory reaction on the pleural surface, an attempt on the part of nature to protect the pleural cavity by sealing up the stomata and localizing the disease. This reaction will differ in degree according to
to the severity of the stimulation. Thus we may have a mere hyperemia with some swelling of the endothelial cells lining the membrane, or we may get a serous or a "fibrinous" exudate poured out in small or large amount, or the process may result in pus formation and the development of empyema. Where pus formation occurs it is probable that a pyogenic organism such as the staphylococcus is present, but in other cases the pneumococcus is sufficient.

It is more than possible that many of the cases of primary pleurisy which are not followed by tuberculous disease are in reality due to an infection of the pleura by the pneumococcus in the manner described above, either with or without bronchopneumonia in the subjacent lobules of the lung.

Bronchopneumonia, as described in the foregoing pages differs widely from fibrinous pneumonia. The appearances in the latter are distinct, largely owing to the nature of the exudate, and the general, equal, and simultaneous involvement of a great part or the whole of one lobe. Here there is a portion of lung consolidated and "hepatized" from coagulation of the inflammatory exudate. On section, its surface is more or less granular, and much less moist than in bronchopneumonia even where a lobe is involved. The difference, however, as in the case of the diffuse and pseudo lobar forms of bronchopneumonia, is probably a matter of degree in the reaction produced by the infective agent. The pneumococci being highly virulent, the
the resulting reaction is correspondingly violent, and leads to the outpouring of a serous fluid, and the escape from the capillary walls of large numbers of red blood corpuscles and leukocytes. The leukocytes, dying under the influence of the toxins, set free nucleoproteid, which, with the lime salts in the fluid, produces fibrin ferment, precipitating the fibrinogen and forming a fibrinous coagulum in the air vesicles. This has the effect of sealing up the entire lobe and preventing the further invasion of the infective agent as far as the lung is concerned.

While these processes are taking place locally, a general infection is also proceeding, and the blood is saturated with the toxins produced by the pneumococci, even if some of the organisms themselves are not also present, having evaded the attempts of nature to keep them in situ in the affected lung. As a rule, however, the pneumococci, travelling from the lymphatics in the interalveolar and interlobular septa, are stopped in the bronchial glands at the roots of the lungs. Here by the irritation of their presence and of the toxins produced by them, an inflammatory reaction ensues, and the bronchial glands become enlarged and swollen. The toxins meanwhile find their way into the blood stream. Disturbances of temperature secretion and excretion result, constituting the general symptoms observable. The cells of the body, however begin to manufacture antitoxins, and in favorable cases the toxins are neutralized and the general and local conditions are brought to a termination.
When this occurs the disturbance of temperature ceases either suddenly or gradually, and resolution sets in in the lungs.

The catarrhal exudate is disposed of, some by being expelled into the bronchi and ultimately expectorated, and the remainder by being re-absorbed, the fluid by the blood vessels and lymphatics, and the cellular elements probably by the lymphatics alone. These lymphatics are interalveolar and their contents pass from them to the interlobular and peribronchial lymphatics, and on to the lymph glands at the roots of the lungs.

The inflammatory products are usually completely got rid of, and the lobules return to the normal; but in other cases a degree of interstitial thickening may remain in the alveolar walls and interlobular tissue. This is a result of the inflammatory reaction in the course of the lymphatic vessels along which the actively virulent pneumococci have travelled while the disease was in progress. Some interstitial cellular increase is commonly met with in bronchopneumonia, but it usually disappears with resolution. It may, however, persist in a chronic form, and lead to permanent fibrous changes.

Tuberculous conditions are by no means necessary sequels to bronchopneumonia, but it is possible to imagine that the way is often paved for the entry of the Tubercle bacillus by the devitalization of the lung tissue by an attack of bronchopneumonia in weakly and susceptible persons, more especially if the condition persists in a more or less chronic form.
CLINICAL HISTORY.

The character of the onset varies considerably. This can be readily understood from our knowledge of infections generally. It may be said that the effect produced is proportionate to the dose of the poison.

In the case of fibrinous pneumonia the invasion by the pneumococci is a severe one, in which the micro-organisms are virulent and in large numbers; and their toxins are suddenly thrown into the blood, with the result that severe general symptoms are suddenly produced.

Similarly in the case of primary bronchopneumonia, if the pneumococcus is virulent, and the attack, even though diffusely distributed through the lung, is simultaneous, the evidences of general infection are sudden and severe. As will be seen in the Mayo Hospital cases, rigor is a not uncommon feature; in a number of instances the invasion, though sudden and definite, is unaccompanied by shivering; and in other cases, again, the onset is more gradual, indicating a less acute infection.

In the severe cases the chief symptoms are usually fever, pain in the chest, and cough, with rapid respiration and pulse, and frequently dyspnoea. In the milder cases these symptoms are modified, and some, such as the pain and dyspnoea, may be absent altogether.

The fever may commence with or without rigor. In 17 out of the 43 Mayo Hospital cases recorded rigor
Rigor was definitely present, a proportion of 39.5 per cent. In 9 other cases, (20.9 per cent) the onset was sudden and definite, with fever or a feeling of chilliness, but no distinct shivering.

The fever may be high, attaining a temperature of 103° or 104° and not infrequently 105°F. It presents remissions as a rule, showing a difference of 2 or 3 degrees between the morning and evening temperatures, but this is not invariable, and occasionally it is practically continuously high, and resembles the temperature of lobar pneumonia.

The duration is variable, depending probably on the character of the infection and the vitality of the patient.

In the Mayo Hospital cases in 1904 and 1905 the longest duration was 42 days, and the shortest 4 days. Eleven cases had a duration of 21 days and over, including 2 cases removed by their friends, on the 19th and 20th days respectively, still showing symptoms of pyrexia. Three cases were removed by friends during the course of the disease and their subsequent history is unknown; but the remainder, (29 cases out of the 43 cases recorded), showed an average duration of 12.9 days.

Defervescence was by lysis in the majority of the cases, namely 22, and by crisis in 6, while 4 cases showed a rapid lysis or what might be termed a "gradual crisis", a steady almost uninterrupted fall of temperature covering 2 or perhaps 3 days.

The pain in the chest is sometimes a general oppression, but is frequently referable to one region.
In the latter case it is usually sharp and stabbing in character and indicates almost certainly involvement of the pleura.

The cough is generally short and dry at first. It may remain so all through, but more commonly becomes more pronounced and more free; occasionally it appears to come spasmodically in paroxysms. The sputum is glairy and mucous, becoming mucopurulent later on, but in a number of cases it shows the "rusty" character found in fibrinous pneumonia, as noted in eleven of the Mayo Hospital cases here recorded.

Dyspnoea is sometimes intense and accompanied by considerable cyanosis. This may be due partly to weakness of the cardiac muscle in debilitated subjects, and partly, possibly often entirely, to the extensive distribution of the lung affection, and the occlusion of a large number of lobules.

Where any considerable extent of respiratory surface is shut off, the rapidity of the respiration increases in compensation, and its ratio to the pulse rate may be markedly altered, as in lobar pneumonia. In the Mayo Hospital cases this disproportion was noted in the majority, though in 8 out of all the cases (43) it was not observed, and was only slightly noticeable in 8. The average ratio, where a disproportion between the respiration and the pulse existed, was 1 to 3.

In severe cases the breathing is markedly hurried, with no pause. The inspiration is short, with a short expiratory groan or grunt. The diaphragm, and
and the accessory respiratory muscles, and also the alae nasi, are brought much into action to assist the breathing. In milder cases the rapidity of the respiration is naturally considerably modified, as also its character.

PHYSICAL SIGNS.

Inspection of the chest frequently shows, besides the character of the breathing mentioned above, some indrawing of the lower intercostal spaces on inspiration. This naturally occurs only when there is any extensive lobular occlusion or collapse.

Palpation may reveal some rhonchoidal fremitus in one or other area of the lung, but it is rare to obtain any increased vocal fremitus.

Percussion sometimes shows no change. At other times, especially in the lower lobes, a somewhat tympanitic sound, probably due to relaxed lung tissue, is elicited. In other cases, especially about the inferior angle of the scapula and in the mid axilla, an area of dullness can be made out, though generally only by comparison with the opposite side. Occasionally, however, a marked dull note is evident over the greater part of one lobe.

Auscultation gives varied and sometimes conflicting results. At times the breath sound is much diminished, and the air entering the lung at the area examined is evidently very small in amount. More generally, the breathing is of the harsh vesicular variety, and the
the expiration rather prolonged; while in other cases bronchial breathing can be heard even though the percussion note is resonant. The area over which bronchial breathing can be heard naturally varies with the extent of the consolidation present.

The accompaniments vary from an extreme amount of high pitched rhonchi, with medium crepitations (often copious), in all areas of both lungs, to an occasional sibilant rhonchus, audible in only one or two places, and possibly accompanied by a few crepitations. Sometimes, again, the only sign discoverable is a few medium crepitations. Pleuritic friction can sometimes be heard; but not infrequently, even when pleurisy exists, it is absent owing to the patient restraining his breathing to avoid the rubbing of the inflamed pleural surfaces, or is elicited only on making him take a deep breath.

The vocal resonance, in many cases of bronchopneumonia, is unaltered; in others it is markedly increased either over the whole or part of one lobe, (when other evidences of consolidation are usually present.) Sometimes, however, it is only by listening carefully over most of one lung (for instance, the axillary and infrascapular regions, with the patient lying on one side with his arm raised above his head) that a variation in the quality of the vocal resonance in different spots can be appreciated.

These indications help us to assign more or less typical cases of bronchopneumonia to one or other of the
the three classes defined by Osler, namely, (a) one in which there is little or no implication of the lobules, and the symptoms are indicative of an extreme capillary bronchitis, (b) one in which there is a diffuse lobular affection of one or both (nearly always both) lungs, with generally some accompanying change in the terminal bronchioles, and (c) a pseudolobar form, in which there is a marked involvement of one lobe, with extensive consolidation, although there is usually also evidence of diffuse lobular affection elsewhere.

There are cases, generally falling under the second category, in which neither the invasion nor the symptoms are very definite. Pain in the chest, cough, dyspnoea, and hurried respiration may be absent, and the only symptom at first may be fever, the nature of which is determined only after the lapse of two, three, or more days, when physical signs have manifested themselves.

The case of Jhanda (No: 41 in the list of Mayo Hospital cases) is an extreme case in point. Here the only symptom was fever. The patient had the usual pharyngeal cough of the inveterate hookah smoker, but his lungs were quite clear. Various fruitless blood examinations were made, but it was not until the eleventh day of the fever that physical signs were found in the lungs, and some sputum was obtained which yielded the pneumococcus on examination.

The physical signs may be very slight and quite out of proportion to the gravity of the disease.
Quite recently, in January 1905, I found, post mortem, extensive bronchopneumonia involving chiefly the right lung, and almost all of its lower lobe (smears from which yielded numerous pneumococci along with streptococci and staphylococci) in a case whose symptoms during life had been exceedingly severe; fever, with no cough, and only a few crepitations in the right base, without dullness on percussion, as physical signs.

These perhaps are extreme cases, but they give an idea of some of the variations to be met with in the disease, and some of the difficulties to be encountered in making a diagnosis.

Recovery from bronchopneumonia is gradual, even though the fever may subside by crisis.

The pain, when present, usually goes with the fever, if not before it. The pulse and respiration settle down to their normal ratio after a few days, certainly within a week.

The cough, however, remains, and a mucopurulent sputum continues until nearly all the physical signs have disappeared. The bronchial breathing which may have existed over consolidated areas is the first of the physical signs to become modified, and the increased vocal resonance ceases to be heard soon after this. The alteration of the percussion note, and the crepitations and rhonchi are very persistent, and even after the entire lung has "cleared up" an occasional rhonchus may be elicited on making the patient
patient cough.

In most instances recovery is complete, but it occasionally happens that cases do not fully convalesce, and continue to show some febrile symptoms and persistent local signs. My experience of such cases is very limited, and chiefly to those secondary to bronchitis, and while recognizing that a chronic interstitial form may follow upon an acute bronchopneumonia I have always suspected that a tubercular infection has supervened, even though the tubercle bacillus has not been demonstrable in the sputum. Such cases rarely remain in hospital long enough to observe the later developments. They are satisfied to have recovered from the acute attack, and then go to their homes for change of air.

In the preceding pages I have not ventured on a consideration of the statistics shown in Statement A of the Mayo Hospital cases as indicating the seasons when primary bronchopneumonia is most prevalent, or the question of the greater virulence of the pneumococcus at one particular time, because the figures do not cover a sufficiently long period to warrant any definite pronouncement. In my experience, however, we generally look for what may be termed an epidemic wave of pneumonia at the commencement and at the end of the cold season, though lesser "waves" may appear during the winter. In the epidemics the earlier and the later cases are usually bronchopneumonic in type. Possibly in the earlier cases the
the infection is mild, and virulence is gradually acquired by the passage of the organism through successive individuals, while in the later cases the pneumococcus is diminishing in vigor. It is noticeable that both previous to and during the months when the virulence of the pneumococcus is greatest, as shown by the higher proportion of deaths to cases in fibrinous pneumonia, the cases of bronchopneumonia are numerous. This may, however, be due to the amount of infection to which different individuals are exposed, a severe infection causing fibrinous pneumonia, while bronchopneumonia results from a less infection in individuals who might otherwise have escaped if they had not been weakly and in ill health.

In the absence, however, of larger data, no conclusions are possible.

For the rest, I trust that I have shown sufficiently clearly the existence of primary bronchopneumonia in adults as a definite clinical entity, and demonstrated the relations of its clinical forms to one another and to fibrinous pneumonia as being one of degree rather than of pathogenesis.
APPENDIX.

Charts illustrating cases of bronchopneumonia in adults, showing the temperature, remissions, mode of termination, and the relation of the pulse and respiration.

I. Two cases of termination by crisis.

II. Two similar cases, one with marked remissions of temperature.

III. Two cases terminating by rapid lysis.

IV. Two cases showing a less rapid defervescence.

V & VI. Two cases ending by a very gradual lysis.

VII. Two cases terminating in death, in A, by hyperpyrexia; and in B, after crisis.

In the majority, the ratio of respiration to pulse is increased to 1 in 3 or less, but in several cases this disproportion is not marked, namely in I B., III A. IV B., and VII A and B (where the ratio of respiration is increased only at the end.)
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**Note:**
- B.O.: Blood Sugar
- Sugar
- Albumen
- Reaction
- Pulse

**Graph:**
- X-axis: Dates
- Y-axis: Values
- Graph shows fluctuations in B.O., Sugar, Albumen, Reaction, and Pulse over time.

**Results:**
- Analysis of the graph indicates variations in the patient's health parameters over the specified dates.
APPENDIX NO: 2.

Chart of a peculiar case at present in the wards of the Mayo Hospital, Lahore, showing the characters of a bronchopneumonia, with those of fibrinous pneumonia supervening.

Sheno-25 years-Mahomedan-Laborer.

Admitted on 24th February 1906 complaining of pain on the left side of the chest, fever, and cough. Duration 6 days.

History of malarial fever every year for nearly 10 years, last attack lasting 2 to 3 weeks-No history of diarrhoea nor dysentery.

Onset gradual-fever not noticed with first appearance of pain.

Patient emaciated and anaemic.

Both lungs affected, showing a diffuse bronchopneumonia-cough dry-occasionally paroxysmal-no expectoration.

Temperature markedly remittent-sudden rise, without rigor, on afternoon of 20th day-followed by complete consolidation of left lower lobe-Temperature remained high till 27th day of disease, when crisis occurred followed by recovery.