5-1-1933

Etiology of pulmonary abscess

Lucien Sears
University of Nebraska Medical Center

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Recommended Citation
Sears, Lucien, "Etiology of pulmonary abscess" (1933). MD Theses. 291.
https://digitalcommons.unmc.edu/mdtheses/291

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.
THE ETIOLOGY OF PULMONARY ABSCESS

By

Lucien Sears

University of Nebraska Medical College

Senior Thesis

1933
PREFACE

The purpose of this paper is to present so far as possible a modern and rational explanation of the etiology of pulmonary abscess.

No attempt has been made, in writing this paper, to include the symptomatology, pathology, diagnosis or therapy of pulmonary abscess.

The references were chosen with the idea that they are useful references, that is to say references which I have found useful, although for the most part quite recent, they will be found to contain a number of older articles.
INTRODUCTION.

The term lung abscess has come to be rather loosely applied to a variety of conditions. Strictly speaking it consists of a focus of suppuration within the parenchyma of the lung; but in current literature has come to include suppuration within the bronchial tree and even between apposing pleural surfaces. It may perhaps be best to consider the condition in this wider sense; but with the realization that different conditions may vary in their mode of production, in their pathology and their amenability to treatment.

Abscess of the lung is much more common than was thought to be the case before the introduction of the bronchoscope. Since then an increased amount of attention has been paid to suppurative conditions in the lungs. It is being realized that many cases, which for years have been regarded as tuberculosis, are in reality not tuberculous at all, but are suppurative in character. Large sanatoria are full of such cases, and the differentiation of these from true pulmonary tuberculosis has always been a difficult problem.

Abscess of the lung appears to be a complication of pulmonary infection rather than a disease per se,
as such, it has long been known to have a rather low incidence. But with the enormous development of operative surgery which the last fifty years has witnessed it has taken on a new significance as a post-operative accident.
THE ETIOLOGY OF PULMONARY ABSCESS.

Clinically, pulmonary abscess includes a variety of conditions which present sometimes the features of abscess, sometimes those of gangrene, and sometimes those of bronchiectasis. When the lesion is single, discrete, and encapsulated, the condition is spoken of as pulmonary abscess; when otherwise and often in doubt as to the nature of the lesion, a definite and inclusive term chronic, non-tuberculous pulmonary suppuration is used. (28).

The fact that the development and anatomy of the lungs has such a definite bearing on the etiological factors in pulmonary abscess, demands a short discussion or review of the developmental anatomy, gross anatomy, histology and physiology.

The lungs are developed from the two diverticula of the caudal end of the median longitudinal groove and the mesodermal tissue into which these grow. Originally single, this caudal end soon becomes bilobed and pouches out on each side into two lateral diverticula, which represent the primitive bronchi and lungs. From the first the right pulmonary diverticulum or vesicle is slightly the larger of the two. Both diverticula
elongate, and almost immediately undergo a subdivision to the right into three vesicles, and the left into two vesicles - thus early indicating the three lobes of the right lung and the two lobes of the left lung. As the primitive respiratory tube lies in the median plane in the dorsal attachment of the septum transversum, the pulmonary diverticula grow laterally and dorsally into the dorsal parietal recesses, that is into the future pleural cavities, carrying before them a covering of mesoblast. From this mesoblast are derived the blood-vessels and other tissues which build up the lung, while the entodermal cells which form the lining membrane of the primitive respiratory tube eventually develop into the epithelial lining of the air-passages, and are embedded within the surrounding mesoderm. The main entodermal subdivisions continue to branch and re-branch, pushing their way into the pulmonary mesoblast, until the complete bronchial tree is formed. When the ramification of the entodermal tubes into the lung-mesoderm is complete, the small terminal flask-shaped extremities of the various branches represent the atria of the lung. (14).

When healthy and sound each lung lies free within the corresponding pleural cavity, and is attached
only by its root and the ligamentum pulmonale. It is uncommon, however, in the dissecting-room, to meet with a perfectly healthy lung. Adhesions between the pulmonary and parietal layers of pleura, due to pleurisy, are generally present.(14).

Like the cavities in which they are placed, the two lungs are not precisely alike. The right lung is slightly larger than the left, in the proportion of about eleven to ten. The right lung is also shorter and wider than the left lung. This difference is due partly to the great bulk of the right lobe of the liver, which forces the right cupola of the diaphragm to a higher level than the left cupola, and partly to the heart and pericardium projecting more to the left than to the right, thus diminishing the width of the left lung.

The lung is light, soft, and spongy in texture; when pressed between the finger and thumb it crepitates, and when placed in water it floats. The elasticity of the pulmonary tissue is very remarkable. A striking demonstration of this is afforded when the thoracic cavity is opened, and the atmospheric pressure acting upon the interior and exterior of the lung is equalized. Under these conditions the organ immedi-
ately collapses to about one-third of its original bulk, and it becomes impossible in such a specimen to study its proper form and dimensions. The surface of the adult lung presents a mottled appearance. The ground colour is a light slate-blue, but scattered over this there are numerous dark patches of various sizes, and also fine dark intersecting lines. The coloration of the lung differs considerably at different periods of life. In early childhood the lung is rosy-pink, and the darker colour and the mottling of the surface, which appear later, are due to the pulmonary substance, and particularly its interstitial areolar tissue, becoming impregnated, more or less completely, with atmospheric dust and minute particles of soot.(14).

The foetal lung differs from the lung of an individual who has breathed. After respiration is fully established, they soon come to occupy almost the whole space allotted to it in the pleural cavity; in the foetus, on the other hand, the lung is packed away at the dorsal aspect, and occupies a relatively much smaller amount of space in the thoracic cavity. Furthermore, it is firm to the touch,
and sinks in water. It is only when air and an increased supply of blood are introduced into the lung that it assumes the soft spongy and buoyant qualities which are characteristic of the adult lung. (14).

The lungs are accurately adapted to the walls of the pleural chambers in which they are placed, and in the natural state they bear on the surface impressions and elevations which are an exact counter-part of the irregularities on the walls of the cavity in which they lie. When care has been taken to harden it in situ, each lung presents for examination an apex, diaphragmatic, mediastinal, and costal surfaces, and ventral and inferior borders. (14).

The root of the lung contains a number of structures which enter and leave the lung at the hilum on its mediastinal surface. They are held together by an investment of pleura, and constitute a pedicle which attaches the lung to the mediastinal wall of the pleural cavity. (14).

In their mode of development and architecture, the lungs resemble compound alveolar, or saccular, glands, the repeatedly subdividing air tubes, or bronchioles representing the duct-system of a gland and the ultimate compartments of the respiratory
tissue, the alveoli, corresponding to the glandular alveoli. Before birth, the resemblance to glandular tissue is very striking, the alveoli being lined with cuboidal epithelium, but after birth the alveoli are distended with air, and the lining epithelium is extended into extremely thin cells. (45).

The blood-vessels of the lung, as those of the liver include two sets, the larger venous and the smaller arterial. The former are the branches of the pulmonary artery; the latter are the bronchial vessels. The bronchial arteries arise from the aorta, not directly from the heart. The branches of the pulmonary artery follow closely the ramifications of the air-tubes, entering the lobules near their apices, along with the intralobular bronchioles, and finally breaking up into the close capillary net-works in the walls of the alveoli. From these networks arise the radicles of the pulmonary veins which carry away the oxygenated blood. These vessels, however, do not immediately join the arteries, but running first on the outside of the lung-units, unite with others and then emerge at the periphery of the lobules and run in the interlobular connective tissue, later meeting the interlobular parts of the arteries and bronchi which
they thence accompany to the hilum of the lung. At the surface, where the pulmonary tissue is in contact with the overlying serous membrane, twigs from the pulmonary artery communicate with the pleural capillaries. The bronchial arteries supply the walls of the air-tubes as far as the terminal bronchioles, as well as the walls of the branches of the pulmonary artery and veins, the bronchial lymph-nodes and the visceral pleura. Within the walls of the bronchial tubes they form a deeper capillary network for the muscle and glands and a superficial one for the tunica propria. The bronchial veins are tributary for the most part to the pulmonary veins; to a small extent, however, blood passes from them into the azygos system. Both the bronchial arteries and veins communicate with the pulmonary vessels at many points.

The lymphatics include a superficial network, well developed and beneath the pleura, and a deep interlobular plexus surrounding the bronchi. The deep ones probably begin as tissue-spaces distal to the terminal bronchioles, around which tubules definite lymphatics first appear. The superficial vessels are connected with small, uncertain, subserous lymph-
nodes, subsequently joining the interlobular trunks, which ultimately are efferent to the larger nodes situated in the hilum and roots of the lungs. The pulmonary lymph-nodes are deeply pigmented owing to the accumulation of inspired dust particles. Where cartilage exists, the plates are enclosed by double networks of lymphatics, the inner one lying within the submucosa. (45).

The frequency of operations on the mouth and nose has added greatly to the number of septic infections of the lungs. Bronchoscopic examination has revealed the important role which foreign bodies of a certain type may play in the production of these disturbances. And last, though not least, the introduction of lipiodol as a means of investigating the condition of the bronchial tree has provided a tremendous stimulus to our interest in these matters. (8).

Of those conditions in which suppuration and destruction of tissue are the principal features, the most important are abscess, gangrene of the lung, and bronchiectasis. Abscess and gangrene are so closely related that they could easily be considered together. Bronchiectasis should logically be taken with diseases
of the bronchi, but for some reasons it is considered along with suppurative conditions of the lung.(8).

There are so many points of similarity in gangrene and pulmonary abscess that it is believed conducive to clearness if they are considered together. The conditions predisposing to them are practically identical, and hence the determination of the local peculiarities depends upon the nature of the microorganisms responsible for their production.

The modern conception of disease consequent upon bacteriological investigations has cleared up much that was before obscure concerning the causation of abscess and gangrene of the lung. There are still some questions remaining unanswered, however, which will be touched upon.

The presence of micro-organisms is indispensable to the production of suppuration within a circumscribed area in the lungs. In some instances, perhaps, an intense nutritive disturbance prepares the soil for the action of germs; in others the germs themselves exert so malign an action that this leads to death of the part. The same remarks hold true of gangrene, but it is likely that a serious interference with local nutrition precedes in the majority of in-
stances.

As might be expected, the organisms responsible for abscess are most commonly pus-cocci, and yet, it is likely that pathogenic bacteria may, when unusually virulent, cause necrosis and suppuration. Of these latter, the pneumococcus and the influenza bacillus are the most frequent. In numerous instances which were studied with special reference to the germs of an abscess cavity or sputum they were found to consist of mixed forms. (2).

The predisposing causes of pulmonary abscess are, lowered resistance to infection, impairment or inhibition of the local defense mechanism, and the presence of a sufficient number of pathogenic organisms capable of producing suppuration. (27).

Lowered general resistance usually occurs in prolonged severe illness from any cause. Impaired local resistance follows devitalizing trauma and any condition that impairs the blood supply. Conditions which inhibit the activity of the cough reflex and cilia of tracheobronchial mucosa facilitate the invasion of infection through the bronchi. Neither lowered resistance nor impaired defense mechanism, or their combination, will lead to abscess except in the presence
of pathogenic bacteria, but a sufficient amount and virulence of infection may lead to abscess formation following their accidental introduction, even in the absence of general or local lowered resistance. (27).

Normally there is a powerful germicidal activity in the lung but if the bronchus is blocked the normal secretions below the obstruction become purulent. (52).

The most common causes of abscess of the lung as given by John B. Murphy are; (1) acute circumscribed inflammation, as pneumonia, followed by necrosis and softening of the lung. (2) Peribronchitis; (3) septic embolism of the pulmonary artery, or a single branch of the bronchial artery (pyemic embolism); (4) rapid tubercular caseation and necrosis, with secondary infection; (5) perforation of the lung by infection from malignant disease of the esophagus and mediastinum; (6) Subphrenic perforations into the lung with retention; (7) foreign bodies in the bronchi; (8) infections following injuries; (9) suppurative inflammation around calcareous deposits, the latter either a sequence or a cause of the suppuration. From the above it will be seen that the pathology of abscess of the lung
is as varied as its etiologic factors. Its clinical course, its treatment and its termination will vary with its etiology. (42).

Abscess of the lung may be either single or multiple. The former variety is met with in pneumonia, and as a result of injuries such as fracture of the ribs, or wounds penetrating the lung, and in some other conditions. Multiple abscesses are generally pyaemic in origin and are due to the softening of infarction or to septic bronchio-pneumonia. The abscesses of this nature are usually of small size, and beyond the occasional presence of limited patches of pleural friction and possibly a small amount of foetid expectoration, give rise to no symptoms which can be distinguished from those of the general condition and in some cases even these symptoms are absent. (21).

A lung abscess usually develops in one of the following ways: (1) By a pneumonic or broncho-pneumonic area that may break down with the formation of an abscess. A primary carcinoma of the lung may behave in a similar manner. (2) Septic emboli carried to the lung by the blood stream may give rise to multiple abscesses. Here the lesion originates in the
the connective tissue framework of the lung rather than in the bronchi or the alveolar spaces. (3) By aspiration of septic material. This is by far the most important type. The passage of a foreign body into the bronchial tree may be included in that. Bronchiectasis may also give rise to an abscess of similar type. All of these may be said to be intra-bronchial.

In reading Babcock I find that, acute fibrinous pneumonia is complicated by abscess and gangrene of the lung less frequently than is generally supposed. Such is the rarity of abscess of the lung in the course of acute fibrinous pneumonia that some authors are inclined to doubt its occurrence in this form of the disease. Thus, Sonnenberg, judging from his own experience, was inclined to believe that when an abscess develops it is in influenza and not pneumococcus pneumonia. Such a view is too radical, however, since there are many trustworthy observations by competent clinicians which prove conclusively a direct etiological connection between acute fibrinous pneumonia and the form of lung destruction now considered.

Of greater importance is the query concerning the
conditions which cause a given case of pneumonia to terminate in suppuration. Probably the intensity of the local inflammation is of great moment, but inasmuch as these complications are observed more often in the so-called asthenic than in the sthenic type of pneumonia, it is the degree of tissue resistance or the admixture of other germs more than the primary infection that is responsible for the mishap. It is certain that abscesses are seen more frequently in the alcoholic and enfeebled than in the robust as previously stated, the pneumococcus alone is capable of exciting a destructive inflammation, and if we assume a low state of local as well as general nutrition it becomes easy to understand the relatively frequent development of abscess or gangrene in drunkards and other debilitated subjects.

It is stated by some writers that abscess is particularly apt to befall the upper lobe, a circumstance attributed to the likelihood of a previous tuberculous invasion of this portion of the lung and consequent malnutrition of the part. When you consider the relative immunity of the upper lobes to pneumonia as compared with the lower, it appears peculiar indeed for abscess to find its favorite seat at the apex but
it is doubtful in such cases whether or not these cases were acute fibrinous pneumonia. Acute tuberculous broncho-pneumonia of the apex is so common and is so difficult of clinical differentiation from croupous pneumonia as to make it not unreasonable to infer that when abscess in this situation was observed by early clinicians it was many times of tuberculous and not of pneumococcus origin. Some authorities seem to think that it is in reality an aspiration pneumonia, or in other words, an infection of the already inflamed lung in consequence of the aspiration of secretions or food particles from the mouth and throat which carry with them putrefactive organisms. Whether or not such is in reality the case, it is not at all impossible and renders very pertinent the suggestion to take particular pains in the hygiene of the mouth in every case of acute pneumonia, especially when the patient is delirious or greatly debilitated.(2).

In a case of acute broncho-pneumonia that type of inflammation may also terminate in an abscess or gangrene, especially when it is of influenzal origin. Yet, as will be seen later on most instances of lobular pneumonia thus terminating are, in reality, cases of so called aspiration or deglutition pneumonia. In
such, the pulmonary organs suffer traumatic damage while at the same time putrefactive and pyogenic organisms are carried along with the inhaled substance. In other cases the circumscribed inflammation is a sequence of pulmonary embolism which, as will now be seen, is a powerful etiological factor in pulmonary abscess and gangrene. (2).

A later conception of abscess formation in pneumonia is given by Boyd in which he says, "the pneumonic group is not so well defined, but the development of abscess or gangrene from lobar pneumonia is a very rare occurrence. In certain forms of broncho-pneumonia, however, abscess formation may be an important feature, as was frequently seen during the influenza epidemic. In those cases the streptococcus appeared to be the organism responsible for the suppuration, but it probably played the part of a secondary invader." (8).

If, in lobar pneumonia of pneumococcus origin, abscess formation occurs at all, it is due to secondary invasion with a pyogenic organism. The pneumonia runs a short course, the temperature comes quickly to normal and there is complete restitution to normal. Therefore, empyema following pneumococcus lobar pneu-
monia is probably due rather to the pleura over the inflamed lung becoming involved in the process, the bacteria passing through the inflamed area into the pleural cavity, than to the rupture of a sub-pleural abscess. Similar events occur in the peritoneum and the pericardium, where there is often no gross rupture of an underlying lesion to account for the inflammation. (8).

In streptococcus and staphylococcus pneumonia abscess formation is not uncommon, and rupture of a sub-pleural abscess may lead to the development of an empyema.

Not only may intrapulmonary suppuration be produced by aspiration of infected material and by infected emboli, but recent experiments reported by Holman have shown that sterile emboli liberated in the blood stream may become secondarily infected form a coexisting or subsequent bacteriemia, and that the previously sterile embolic area breaks down with the formation of a typical abscesses. (29).

Whatever may be the origin of the infecting organism, whether by way of the blood stream or by the bronchi, the resulting destruction of pulmonary tissue leaves a dead space - the abscess cavity - which must
be obliterated before a complete cure is accomplished. (29).

In the embolic group the abscesses are nearly always multiple. Septic emboli from the female pelvic organs, from the thrombosed lateral sinus of the middle ear disease, and from the vegetations of bacterial endocarditis are among the principle causes. Endocarditis may involve the valves on the right side of the heart, but occasionally the vegetations have been on the left side, in which case minute vegetations must have traversed the capillary system and so reached the right side of the heart and thence the lungs. An infarct may first be produced which breaks down to form an abscess, or an abscess may develop without any infarct formation. In the embolic group no protective barrier is formed between the lesion and the surrounding tissue, and there is rapid extensive breaking down of the lung accompanied by sudden extreme prostration and profoundly toxic symptoms.(8).

Infected emboli are relatively frequent and well recognized causes of either abscess or gangrene according to the characters of the bacteria concerned. Thus of eighty-three cases of pulmonary gangrene analyzed by Heschl embolism and thrombosis were given as the cause
in twenty, while of twenty-nine instances in which the causation was determinable, Coupland found four that could be referred to the same condition. The diseases responsible for pulmonary infarcts are numerous, but puerperal sepsis is one of the most common. In septic metritis or endometritis, as well as in other suppurative processes, venous thrombosis is set up and the coagulum not being firm, fragments are broken off which are carried either to the lungs directly or to other organs. In the latter event abscesses are formed which may, in turn give rise to septic infarcts in the lungs. There may be but a single focus of suppuration in the pulmonary parenchyma, but often there are multiple abscesses of variable size.

Other diseases sometimes responsible for pulmonary infarcts of a septic nature are ulcerative endocarditis, chronic otitis media, erysipelas and suppurative inflammation within the intestinal tract, as rectal abscess or ulcer. In the case of septic endocarditis it is not necessary for the process to be located in the right heart, for emboli may be given off from the mitral or aortic valves and, being carried in the bloodstream, may pass through the arteri-
oles and capillaries to reach, at length, the capillaries of the lungs.

In the case of middle-ear disease, thrombosis may occur in the sinuses of the petrous portion of the temporal bone or in the internal jugular vein, whence infective particles finally reach the lungs. Leyden is of the opinion that when pulmonary abscess or gangrene results from chronic otitis, the infective agent reaches the lung by means of the blood-stream, even when thrombosis cannot be demonstrated since this is far more likely than is the discharge of pus from the eustachean tube, as has been suggested.

In a male patient who died at Cook County Hospital with clinical and post-mortem evidences of multiple areas of gangrene scattered throughout the right lung, there was found a septic thrombosis of the right internal jugular vein secondary to an old middle-ear disease. The clinical findings were those of multiple faci of broncho-pneumonia without demonstrable cavity formation but with the characteristic factor of sputa breath.

If rectal abscess or pyelophlebitis be the ultimate cause of the pulmonary infarcts, infective emboli may pass directly from the veins of the portal system
to the capillaries of the lungs, or they may first give rise to multiple foci of suppuration in the liver and there in turn may cause embolic necrosis and suppuration within the pulmonary parenchyma.

Patients who develop an abscess after an operation expectorate foul pus in large quantities, have a fever, marked lassitude and weakness, rapid pulse and clubbed fingers. The patient usually feels well for several days after the operation. When a general anesthesia is used in the performance of the operation, the onset of symptoms occurs within about six days. Local anesthesia has an elapse of about the same length of time before the onset of symptoms.(30).

It is suggested that the protective barrier to the formation of abscesses in the aspiration cases is an intact blood supply. In septic infarction due to infected emboli, the blood supply is interrupted, and in the presence of infection the illnourished, necrotic pulmonary tissue undergoes liquefaction and cavitation. A similar process of thrombosis and septic necrosis of pulmonary tissue may be the important factors in the mechanism of the production of the pulmonary abscesses that follow broncho-pneumonia.(30).

It is possible that most postoperative pulmonary
suppurations are due to a factor common in all operations, that is a transportation of emboli by way of the blood stream to the pulmonary capillary bed. Postoperative pulmonary suppuration has apparently had its origin less often in the aspiration of blood and mucus than in the setting free of septic emboli into the blood stream.(30).

Not only may intrapulmonary suppuration be produced by the aspiration of infected material and by infected emboli, but recent experiments have shown that sterile emboli liberated in the blood stream may become secondarily infected from a coexisting or subsequent bacteremia, and that the previously sterile embolic area breaks down with the formation of typical abscesses.

Under the heading foreign bodies must be considered those instances of aspiration or deglutition pneumonia which results in suppuration or putrefaction according to the kind or organisms introduced with the substances. A great variety of conditions may be responsible for these pneumonias. Thus pus or fragments of tissue may be aspirated into the lungs during operations on the throat or larynx; or innumerable bodies of various sorts may be inhaled, as particles of food
breathed in by the apoplectic, the insane or feeble bedridden patients. In all such cases, pyogenic or putrefactive bacteria gain access to the lungs along with the foreign materials; or the retained bronchial secretions, themselves infected, excite an intense inflammation which results in necrosis and suppuration. Foci of this kind may be single and of considerable dimensions, but are sometimes multiple.

The inhalation group is the largest and most important. At least fifty per cent of the lung abscesses are due to aspiration. In many papers it is reported that as high as one out of every three was due to operations on the upper respiratory tract. Of such operations tonsillectomy is by far the most common. As a rule in most of these cases a general anesthetic has been used, but by no means always. The removal of adenoids, operations on the nose and the extraction of teeth help to swell the list. In all of these cases it is difficult to exclude the possibility of septic thrombosis and embolism. Many pathologists are still of the opinion that most of these cases of lung abscesses are embolic in origin, arising in a peritonsillar region which they have shown to be studded with thrombi, often septic, and readily dislodged by the ac-
tivity of the muscular pharynx. The similar incidence of abscess following operations about the mouth and of abscess following the aspiration of foreign bodies is an argument in favor of the bronchogenic origin of the former; in both instances the lower lobe is involved in about sixty per cent of the cases, the right lung much more frequently than the left. This argument loses force when it is recalled that emboli are commoner in the right lung than in the left. The lodgment in the bronchi of foreign bodies, either metallic or vegetable, may lead to abscess formation; of these bodies the most common on the North American Continent is the peanut. Food may pass into the bronchial tree especially during unconsciousness. Material from new and other pathological conditions of the tongue, jaw, and the like, may be responsible. In obscure cases the condition of the nasal sinuses should be investigated. Indeed, it may be said that in suppurative diseases of the lungs it is well to look first above the clavicle for the cause of the condition.(8).

There is abundant clinical, and some experimental evidence in support of the statement that certain types of disease of the paranasal sinuses tend to produce peri-bronchial infections, with resultant chronic bronchitis,
asthma, bronchiectasis, and pulmonary abscess.

Experimental investigations have been principally directed, as one would suppose, to identifying the lymph channels that drain the sinuses. By tracing carbon deposits carried by the lymphatics from the sinuses it was found that the lymphatic absorption from the nasal sinuses was by way of the submaxillary and internal jugular nodes, the lymph ducts, the great veins, the right side of the heart and the lungs. Moreover, the bronchial and mediastinal glands were involved also. In the case of the injection of infectious material, the resultant infection followed the same route. It is interesting to note that in none of the experiments were the faucial tonsils affected.

If treatment of infected sinuses is neglected or is insufficient, the chronic purulent stage tends to produce great thickening and a pyogenic condition of the mucus membrane; the infection becomes alternated, and the chronic hyperplastic form of sinus disease may often seem to be a distinct entity because there may have been no antecedent symptoms more definite than the occurrence of frequent colds. The process is particularly likely to begin in childhood when acute infectious diseases and malnutrition predispose to the
development of chronic involvement of the sinuses.

Mullin says, "the hyperplastic is by far the most important form of paranasal infection as a factor in the causation of chronic chest infections. There is almost no drainage through the natural opening, the thickened mucosa is deeply infected and there is protracted drainage and absorption of bacteria and of bacterial products through the lymph channels. Eventually this continuous drainage through the lymph channels leading to the upper mediastinum produces a chronic peribronchial glandular enlargement and later involvement of the adjacent lung tissue."

The four types of lung infections that may be caused by the paranasal infection are, (1) acute bronchitis, (2) bronchial asthma, (3) chronic bronchitis, and bronchiectasis and (4) lung abscesses.

Protracted flooding of the lymph channels with infectious material from the hyperplastic maxillary sinus tends to produce a chronic peribronchitis. This is often clearly shown on the roentgenograms of children. Frequent chest colds may be the first evidence of this extension, but a definite chronic bronchitis is eventually established and in a neglected case the final result is a bilateral bronchiectasis or lung abscess.
which, when well established, are fairly difficult and offer great problems in treatment.

The association of sinus disease with a chest complication is far too common to be accidental. The most typical cases start in childhood. Early recognition of sinus involvement and treatment will prevent the chest complications.(41).

A great number of pulmonary abscesses following tonsillectomy go unnoticed as such and are often diagnosed as pneumonia or simply as a "cold." Obtrusive symptoms of the abscess do not usually develop until at least a week after the operation, thus often the operation is not thought of as a direct cause. In the light of the number of tonsillectomies performed, the pulmonary condition is rare.

While the etiology of pulmonary disease is complex, it would seem that post-tonsillectomic pulmonary abscess is the result of infection, probably from the mouth. Normally the organisms are destroyed before the air cells are invaded through the bronchioles. There are three possible routes of infection: the bronchi, the lymph channels and the blood vessels. As many as two or even all routes may be traveled in a case.(33).

During recent years the chief interest in the
subject of etiology has centered in the question as to whether these postoperative pulmonary abscesses have developed predominatingly from blood stream infection or from aspiration of infectious material. Cutler and his associates, found it impossible to produce abscess in dogs by instilling infected material into the bronchial tract, but were able to produce abscesses almost at will by introducing infection into the jugular or femoral vein. They also found thrombi, often infected, in veins in the tonsillar fossae in patients following tonsillectomy. They pointed to the action of the pharyngeal muscles as favoring the dislodgement of such infected thrombi into the venous circulation. The disproportionately large incidence of post-tonsillectomy abscess they considered more apparent than real, since tonsillectomy comprises a large proportion of the total number of operations performed in many hospitals. On the basis of such considerations embolism has come to be considered by many as the most frequent cause of postoperative abscess.

The evidence pointing to aspiration of infection as the most important factor is both experimental and clinical. A number of investigators have been able to
produce abscesses in experimental animals including the dog under conditions that closely simulate clinical conditions.

Smith, Allen, Joannides and others produced abscesses by instilling pus from patients with pulmonary abscesses within the bronchial tract. Joannides, working with Hedblom was able to produce abscesses in over seventy per cent of the dogs by mixing blood with infection from tonsils which precisely copies actual conditions in the human. Pilot has produced abscesses by mixing the infection with lipiodol. Apparently the blood and lipiodol simply prevent the cough expulsion of the infection. Crowe and Scarff produced abscesses in dogs by introducing into a bronchus, pledges of cotton soaked with scrapings from pyorrhea alveolaris and by producing a frontal sinus infection with secondary purulent rhinitis.(26).

The clinical evidence of aspiration infection seems very convincing. Some of the most important features of this clinical evidence are as follows: Bronchitis is the most common postoperative pulmonary complication, representing from one-third to one half the total number of them. Infection in the bronchus may pass through the damaged mucosa into the pulmonary
tissue by way of the lymphatics. Most postoperative pulmonary abscesses follow operations under general anesthesia during which the protective pharyngeal reflex is obliterated by the local anesthesia. By far the largest proportion of abscesses follow tonsillectomy, teeth extractions, and other operations about the mouth. Wound infection not infrequently occurs following thyroidectomy in which there must result infected thrombi in large veins, yet postthyroidectomy abscess must be very rare. Pathogenic organisms, including spirochetes and fusiform bacilli, have been demonstrated from tonsillar crypts and about the teeth by Pilot and Davis in fifty per cent of patients of the free dispensary class, and identical organisms have been found at autopsy in a large proportion of postoperative pulmonary abscess. Myerson has shown that blood had found its way into the trachea in over seventy per cent of two hundred patients following tonsillectomy performed in the horizontal position. Lemon has shown that dogs will regularly aspirate wet bismuth powder into the bronchial tract during the course of one hour general ether anesthesia. The ease with which nonirritating material may find its way into the bronchial tract is well shown in the routine diagnostic instillation of
lipiodol through the mouth without any preliminary local anesthesia. Recovery of teeth, dentures, and other material from the abscess following dental operations under general anesthesia and the frequent incidence of other foreign body abscesses is indisputable direct evidence of the occurrence of aspiration infection. The occurrence of abscess in epileptics and following alcoholic intoxication and in stupor or unconsciousness from any cause can scarcely be explained on any other basis. The largest nose and throat clinics of the country record a total of several hundred thousand operations about the mouth, with a minimal incidence of pulmonary abscess. If any large proportion of such complications were due to embolic infection, the condition for the formation of which, from the embolic standpoint are constant and largely unavoidable, it would seem reasonable to expect everywhere uniform incidence of this complication. (26)

Patients should be carefully watched for at least a month after tonsillectomy and the slightest cough or other sign of bronchial irritation or pulmonary trouble should place the patient in the pulmonary abscess class unless proved uninfected. (33).
Dr. Charles W. Richardson states the following regarding the symptomatology and diagnosis of the condition. The invasion may occur immediately or be delayed three or four days after the operation has been done. If the invasion is apparently delayed, the patient does not do well in the interval between the operation and the first signs of invasion. The actual invasion is usually manifest by a chill or chilly sensation, followed by a rapid rise in the temperature, which immediately assumes a septic character. Pain in some area of the lung is early present. Cough paroxysmal in character is a very early symptom. Odor of the breath first noted by the patient, later by attendants, is a fairly characteristic symptom. Profuse, heavy-pus expectoration, which may become rusty as the case progresses. Hemorrhage may occur. Profuse sweating at night. Temperature, pulse, and respiration run in usual ratio. The earliest physical signs are those of an infiltrated area, frequently unrecognizable in the early stages, becoming more clearly defined on successive days. The radiograph gives an absolute picture which cannot be misinterpreted. Diagnostic bronchoscopy should be employed in all cases which have symptoms, physical signs or Roentgen-ray
findings indicating pulmonary or bronchial complications following any operation about the upper air passages.(33).

Clendening in 1920 conceived the idea that the motor driven anesthesia apparatus caused the post-tonsillectomy pulmonary abscess by creating a positive pressure in the pharynx. He said, "at any rate, the danger is sufficiently great to justify the discontinuance of their employment until comparative data can be secured." At some time later Clendening retracted this statement but before doing so the above statement created considerable comment.(12).

The most outstanding comment in regard to the above statement was given by Dr. Ralph M. Waters in which he said, "some condemn the motor driven ether and suction apparatus saying that it is a frequent cause of lung abscess at the time or following tonsillectomies, but this apparatus is of great value to the operator in doing a very thorough and complete operation. Now, if ether blower and suction apparatus is responsible for the complication of lung abscess after these operations is it not more reasonable to say that the suction portion of the apparatus is responsible in that it has made thorough tonsillectomy possible
in the dorsal position and has so delayed men in changing to some position in which the throat draining is by gravity forward into the mouth, such as the lateral position of the patient's body with mouth turned down or capable of being turned down when necessary. Prevent the dorsal position when the patient is taken to and put in bed." (54).

Tonsillectomy was responsible for largest single group of lung abscesses reported by Dr. Frederick T. Lord. The impression gained from such a review of cases is that ingress into the deeper parts of the respiratory tract of infected material from above is to be regarded as the chief cause of pulmonary abscess. It is one of the hazards of surgery in the course of which, in certain instances it appears to be an inevitable misfortune, while in others the history suggests that it might have been prevented by greater care in the avoidance of the aspiration of blood tissue or infected material. Search for spirochetes in a number of the more recent cases has failed to demonstrate them in such a number as to suggest that they are to be regarded as important in the etiology. (37).

Dr. Frederick T. Lord says, "lobar pneumonia appears to be of little moment in etiology and an
origin in broncho-pneumonia is uncertain. "(37).

Among the sure causes of pulmonary abscess must be mentioned suppuration of contiguous organs as hepatic, esophageal, vertebral and subphrenic abscesses which break through into the bronchi. Empyema and suppurating bronchial or mediastinal glands must also be included in this list. Unquestionably these and other collections of pus may rupture into the bronchi and be discharged without causing pulmonary abscess or gangrene; but now and then the infective material is aspirated into neighboring lobules with disastrous consequences. They come properly, therefore, under the head of cases of aspiration pneumonia.

Tumors found at varied locations may be the cause of lung abscess. They may be situated within the thorax where they probably exert their effect through pressure, or they may have their seat in the tongue, as seen in ten of Heschl's, and four of Coupland's cases. In cancer of the tongue the gangrene results from inhalation of oral secretions or other substances. Among the intra thoracic tumors may be enumerated aortic aneurysm, cancer of the oesophagus, mediastinum or lung. The exact mode of production of pulmonary gangrene in these cases is still not quite clear. By some writers it is
attributed to a pressure pneumonia or to occlusion and thrombosis of a pulmonary artery. (2).

Carcinomata of the esophagus not infrequently extends into the trachea, bronchi, or mediastinum, and may give rise to pulmonary abscess. Malignant tumors of the bronchi similarly may extend into lung, become infected, and give rise to an abscess. Primary carcinomata of the lung commonly are complicated by abscess. In a series of thirty intra-thoracic tumors observed and reported in Nelson's loose leaf surgery, lung abscess was a complication in five.

In a series of one hundred eight cases of pulmonary suppuration, referred to by Graham nineteen cases or ten and seven tenths per cent were associated with malignant tumors of the lung.

Fowler is of the opinion that the gangrene may be due to retention and decomposition of bronchial secretions, particularly if bronchiectasis has resulted from pressure by the tumor. In support of his contention he cites the instance of a mediastinal lymphosarcoma which terminated in gangrene and in which the left lung was found in a state of sacculated bronchiectasis with septic pneumonia.

Bronchiectasis not infrequently leads to gangrene
of the lung as is shown by five instances in Heschl's table and two in Coupland's. The explanation is to be found in the retention of the bronchial secretions and their contamination by germs of decomposition. But how these latter gain access to the bronchiectatic cavities is not quite clear unless by aspiration from the mouth in the act of coughing, which may be so violent and prolonged as to necessitate vigorous inspiration at its close if not during its continuance. (2).

One frequently hears of cases of acute phthisis which display the clinical features of an abscess of the lung, that is, there is a sudden expectoration of a large quantity of purulent material with subsequent signs of excavation. Strictly speaking, these are instances of acute caseation, but whether induced by the tubercle bacillus alone or by pyogenic organisms either alone or in conjunction with the bacillus is not always quite certain.

The occurrence of gangrene in the course of pulmonary tuberculosis is very exceptional. As with medical statistics in general the figures on this head are somewhat conflicting. Thus, Boudet is said to have found gangrene but once out of one hundred and sixty cases of phthisis, whereas Fox discovered six instances
among ninety-nine cases of pulmonary tuberculosis that came to necropsy. It is interesting to note, however, that of ten instances of gangrene of the lung, tuberculosis in other parts of the organ existed in five.

The cause of the putrefaction in such cases is obscure. The complication occurs most often in cases of vomica or bronchiectasis and hence is probably owing to retention of the contents of the cavity and its infection by saprophytic bacteria derived in some manner from the oral mucosa. An odor of decomposition may sometimes be perceived in the breath of tuberculous individuals, but the conclusion is not necessary that gangrene is present. For some reason gangrene is most likely to occur in cases of fibroid phthisis, possibly because of the liability to bronchiectasis in this class of cases.

Blunt trauma from a blow, fall or a crushing injury may result in actual rupture of the lung or more commonly in an interstitial ecchymosis and oedema followed by a pneumonitis or gangrene and liquefaction of the injured portion of the lung. In eight of twenty-three cases the onset of symptoms was from one to five weeks after injury. Abscess following penetrating injury to the lung, such as produced by a gunshot wound
or stab, is due in large measure to the devitalizing trauma to the tissues, but the infection may result in abscess long after the injury has healed. Abscess formation following retained gunshot missiles or other foreign material has been observed months or years after the original injury. Penetrating injuries may produce an acute fulminating suppurative pneumonitis. An example of such a course was that of a man who was stabbed in the chest five times with a slender ice pick about twenty centimeters long. The patient died the third day with findings of a virulent pneumonic process. (27).

Babcock reports a case of a soldier who coughed out a piece of his coat along with a quantity of pus several months after his wound was received. (27).

In many chest injuries pneumonia first becomes apparent, then later it may speedily undergo suppuration and abscess formation.

Putrid lung abscess is a definite clinical and pathologic entity. The infection is probably initiated and is certainly maintained by pathogenic anaerobes. It is of bronchogenic origin, and is due to aspiration of infected material. Only very few cases have been definitely determined as embolic in nature.
or origin. These rare exceptions present clinical manifestations and pathologic changes quite different from those described now. Experimentally embolic lung abscesses can be readily produced but lung abscesses via the bronchi, only with difficulty. This observation among others has led many to assume that human lung abscesses are often of embolic origin. The fact is that experimental embolic lung abscesses in no way resemble putrid lung abscesses in human beings. The mechanism and the characteristics of embolic abscesses in the human body have been amply demonstrated. Embolic abscesses are derived from a septic focus, usually in a vein, are ordinarily multiple, and are scattered in various parts of the pulmonary parenchyma. They only rarely and then only accidentally communicate with the bronchi, and are thus a silent feature of sepsis in most instances except where there is pleural invasion, and are usually due to the staphylococcus or streptococcus and are thus non-fetid. These and other features are in striking contrast to those of putrid lung abscess. The question is of clinical as well as of scientific interest, for a belief in the embolic origin and pathology of putrid lung abscess would lead to a different plan of treatment from that to be out-
It is believed that putrid lung abscess results from the aspiration of infective particulate material bearing pathogenic anaerobic bacteria. The concept of the disease solely as a sequel to operations on the nose, mouth or throat, or to operations under inhalation anesthesia, or to known aspiration of foreign material, is erroneous. About one third of the cases given in a series of cases reported for a period of two years occurred in previously healthy persons who presented no proved predisposing factor. The concept of putrid lung abscess as a complication of pneumonia is also incorrect. The evidence is clear that the disease is not a complication of pneumonia, but that the so called pneumonia was a putrid abscess from the onset this is a matter of no little clinical significance, for the diagnosis can be made in the stage of so called pneumonia in many instances. Long before conclusive bacteriologic studies were made it was thought that putrid lung abscesses were an anaerobic infection. The clinical characteristics were those of an anaerobic infection and observation of the results of surgical treatment based on that view tended to corroborate the same belief. It is now stated that
appropriate bacteriologic study of pus obtained at the time of operation has confirmed this clinical impression. Certain pathogenic anaerobic bacteria are to be found in every stage of the infection and disappear when the infection subsides. There is no absolute evidence that the disease is initiated by those anaerobes. They are, however, the only pathogenic organisms found in putrid lung abscess which produce gangrenous lesions of the lungs experimentally when introduced into the bronchial tree. (43).

Three striking instances of what appear to be primary pulmonary amebiasis caused by infiltration of the lung by endameba histolytica, independent of liver abscess, have recently been seen by Monson-Bahr. All three cases responded in dramatic and striking manner to emetine therapy. Pulmonary amebiasis may develop independently of hepatic abscess. It may simulate a bronchopneumonia or miliary tuberculosis. The diagnosis is based on the previous evidence of amebiasis, the leukocytosis, and the remarkable and lasting response to emetine and ipecacuanha. Rupture of a hepatic abscess into the lung may take place without previous warning or the coexistence of hepatic symptoms. The resulting pulmonary infection may closely resemble
idiopathic disease of the lung, such as tuberculosis or bronchiectasis, both in local signs and general effects. Secondary infection of the respiratory tract with yeast fungi may occur, so called bronchomoniliasis. This appears to be of little diagnostic and possibly of no etiologic importance, because only one case is cited.

There remains a considerable group of cases in which the cause of the abscess can not be ascertained. Many of the patients in this group will say, that the onset was with a productive cough following a "cold". In most instances the symptoms, physical findings and course present nothing that distinguish this group. It seems probable that many of them originate from unrecognized aspiration of infected material.

There is a case of lung abscess reported by Knowlton associated with meningocerebritis of a slow subacute type. It was apparently the cause of the more acute condition in the lung as was shown in the autopsy.
CONCLUSION.

In reviewing the literature on the etiology of pulmonary abscess I find that it is fairly definitely and uniformly agreed that the vast majority of cases are of inspiratory origin, because of the time of development, and involvement of the lower lobes of the lung in sixty per cent of the cases (right lower, forty per cent; left lower, nineteen per cent), being almost the same relative incidence as in cases of inspired foreign bodies; blood stream transmission of infected material, causing pulmonary abscess, occurs in a relatively small number of cases; lymphatic extension is a relatively rare mode of infection. (40).

We are justified in believing, that the wear and tear of life falls more on the right than the left. It has three lobes as against two and is about ten per cent larger by volume than the left, if one may judge from Krause's old figures, so that on the doctrine of chances it would suffer more from localized disease, and it receives more emboli through the blood stream. The right pulmonary bronchus is slightly larger than the left and is more directly in line with the trachea,
so that in the natural process of respiration it gives admission to more floating material, bacteria or the like, both in conscious and unconscious states. Undoubtedly the right lower lobe suffers most of this wear and tear, receives more gross foreign bodies and is more exposed to the irritation of ether. The semi recumbent and upright positions are not as free from these complications as has been heretofore supposed. (31).

Clinically lung abscess is a disease of symptoms rather than signs, therefore, the roentgen examination is of great aid in indicating the site and extent of the process in its early stages.

The lesions are usually single, although they may be multiple. They may occur in either lung field, but show a decided tendency to appear at the bases, particularly the right. They assume the form of irregular areas of increased density in the roentgenogram and are most marked at the center, fading out toward the periphery. Abscesses may be confused with tuberculosis, bronchopneumonia and bronchiectasis, but are rarely mistaken for metastatic malignancy.
BIBLIOGRAPHY.


(12) Clendening, Logan: The Cause of Abscess of


(37) Lord, Frederick T. : Certain Aspects of Pulmonary...


(44) Nelson : Loose Leaf Living Surg. - Infections of the Lung. 4: 549R.


