Basic concepts underlying chronic pulmonary diseases have remained unchanged for more than 80 years despite the radical changes in medical and surgical treatments of these diseases within the past two decades. The need for changes in basic concepts is underlined by the fact that the main tenet which is the keystone in several of the interconnected concepts implies that phagocytic cells are endowed with intelligence! This is the conclusion which must be drawn from the existing concept of alveolar hygiene.

According to the long-established, and still prevalent, theory which seeks to explain the mechanism by which the alveolar membrane is kept immaculate, phagocytic cells situated upon the alveolar surface and having engulfed bacteria or dust particles, will purposefully migrate either to the mucous blanket covering the tracheobronchial ciliated epithelium or through the alveolar membrane. Having arrived in the lung interstitium by penetrating the alveolar membrane, the phagocytic cells are pictured seeking out lymphatic vessels singlemindedly, entering them, and beginning their transport to satellite lymph nodes.

This is but one example of concepts no longer in tune with the present state of our knowledge. It is the purpose of this paper to discuss briefly some newer concepts of basic pathologic and physiologic processes affecting the lungs, and to list the published supporting experimental and clinical evidence.

Pulmonary Clearance Mechanisms

Inhaling approximately one-half liter of air 12 to 20 times per minute, or about ten cubic meters per day, a considerable amount of finely divided material (animate as well as inanimate) suspended in the air may reach and settle upon the alveolar membrane during the lifetime of a person. The amount depends, of course, upon the environment. In a large measure, because of the extraordinary efficiency of the pulmonary clearance mechanism, clinical pulmonary disease is the exception rather than the rule. It has been estimated that French miners, during 30 years of occupational exposure, have inhaled about 6 kg. of dust. However, post-mortem examination of the lungs of these miners have yielded only 60 to 80 gm. of dust, approximately 1 per cent of the inhaled total.

The mechanism of this wonderfully efficient cleansing mechanism has two components, the upper respiratory and the alveolar mechanisms, and both must work together to make the whole effective. The upper respiratory mechanism must be capable of removing adventitious material, cells, and debris at least as rapidly as this material is brought to the mucous blanket by the alveolar mechanism.

The upper respiratory mechanism consists of a moving 5 micron-thick blanket, extending from the terminal bronchioles to the pharynx. It is propelled proximally by the whipping action of the cilia (220 beats per minute) at a rate of 15 to 18 mm. per minute. The mucous blanket consists of a viscid superficial portion secreted by the goblet cells of the tracheobronchial mucosa and a deeper, less viscid, more fluid portion derived from certain apocrine cells of the respiratory bronchioles, as well as from the film of fluid covering the alveolar membrane.

The alveolar mechanism consists of a film of fluid about 0.2 micron thick which moves continuously over the alveolar surface toward the terminal bronchiole, finally joining with the secretions of the respiratory bronchiole and carrying with it...
all materials adhering to the surface of the film. The source of the film of fluid is probably a combination of a transudate from alveolar capillaries and a specific secretion from the alveolar surface epithelium. There are two theories which attempt to explain the proximal motion of the alveolar film of fluid. One theory postulates that the ventilatory excursions of the alveolar wall in conjunction with a gradient of viscosity in the alveolar fluid film create an ebb-and-flow motion on the surface of the film and a resultant proximal increment with every respiratory cycle. The other theory assumes that the ciliary activity which moves the mucous blanket creates a sort of suction upon the fluid distal to the terminal bronchiole. The theory which postulates dependence of alveolar clearance upon adequate alveolar excursions is supported by the every-day observation that inadequate alveolar clearance is to be found about vessels, bronchi, tubercles, and other rigid structures where respiratory excursions of alveolar walls are inhibited. This theory is also supported by experimental observations.

The importance of the pulmonary clearance mechanism to the health of the individual is seen in those cases in which this mechanism is known to be defective. For example, children afflicted with mucoviscidosis are recognized to develop pneumonia easily because the excessive viscosity of the mucous blanket in these patients impairs its transport. Likewise, the greater susceptibility of patients with chronic bronchitis and bronchiectasis to develop pneumonia may also be related to an impairment in the clearance mechanism.

PNEUMOCONIOSIS

The development of pneumoconiosis is caused by an inadequacy of the pulmonary clearance mechanism relative to the amount of dust inhaled. Most dusts, when inhaled and deposited upon alveolar walls, are removed without difficulty everywhere except in the regions of vessels and other rigid structures. Because of deficient alveolar clearance, multifocal dust deposits develop in these regions. These multifocal deposits are characterized by alveolar cell and stromal proliferation, exemplified by anthracotic foci and silicotic nodules. The alveolar clearance mechanism may fail to cleanse the respiratory surface not only in certain foci, but such failure may be generalized and diffuse. In the latter event, a diffuse pulmonary fibrosis will result. There is evidence which indicates that the inhalation of ultrafine dust (about 0.02 micron) as in bauxite fume pneumoconiosis or extremely high concentrations of dust (as in acute silicosis), or a filamentous dust (as in asbestosis) will predispose to a generalized, diffuse inadequacy of the alveolar clearance mechanism.

The continued expectoration of coal dust by miners many years after removal from further exposure, suggests that the dust sequestered within intrapulmonary dust foci is being mobilized. The mechanics of this mobilization has been found to consist of edema fluid which not only supplies the vehicle, the proper direction, and the force for the reverse dust transport, but also opens the interstices of the restraining stromal network.

TUBERCULOPNEUMOCONIOSIS

Progressive massive fibrosis of the lungs in coal miners characterized by intensely black lungs which contain large, extremely hard, fibrous masses has been called antrasilicosis in the United States, usually without determining whether or not there had been significant exposure to respirable quartz dust and without a determination of the free silica content of the lungs. In Britain, because tubercle bacilli have been demonstrated in the lung tissue of about 40 per cent of these cases, it has been assumed that all such cases are "infective" pneumoconiosis or tuberculoanthracosis. Support for the latter concept was obtained by the reproduction in E. J. King's laboratory of the human lesion in guinea pigs by injecting them intratracheally with coal-mine dust (low in silica) and tubercle bacilli of low virulence. It is a fact that
when a tuberculous infection is caused by
an organism of low virulence and this in-
festation is associated with anthracosis, the
mycobacterial component of the inflamma-
tion may defy recognition bacteriologically
and anatomically, grossly or microscopically.
On the other hand, a possible silicotic
component may be similarly submerged
so that it defies identification. Pulmonary
cavitation in progressive massive fibrosis is
not necessarily indicative of tuberculosis
because the necrosis may have an ischemic etiology. Thus, it may be
very difficult in some instances to decide
whether a given case is one of tuberculo-
anthracosis or anthrasilicosis.

It is prerequisite to the development of
progressive massive fibrosis of the lungs (or
tuberculopneumoconiosis) that extraordi-
narily large quantities of dust must be de-
posited in the lung tissue. The publication
of the findings in three kaolin workers with
massive pulmonary fibrosis indicates that
other so-called "inert" dusts may, in con-
junction with a tuberculous infection, pro-
duce a tuberculopneumoconiosis. However,
as with coal dust, the report indicates that
a large amount of dust must have been de-
posited in the lungs for the production of
the massive fibrosis. An experimental re-
port dealing with a variety of "inert" dusts corrobates the excessive dust exposure
as a prerequisite.

CHRONIC INTERSTITIAL PNEUMONITIS

The basic response of the alveolar wall
to a low grade chronic irritant consists of
a proliferation of alveolar cells and associ-
cated reticulin stromal fibers. This re-
sponse of the alveolar membrane has been
observed to such diverse agents as various
insoluble dusts, phosgene, ozone, Mycobac-
teria, fungi, and allergens. Although the
initial response is qualitatively monotonous-
ly uniform, the subsequent evolution of the
inflamations may result in vastly differ-
ent disease patterns. The reticulin stroma
may undergo lysis in days or weeks and
the proliferated cells, deprived of support,
will desquamate (as in a pulmonary tuber-
culin reaction); the reticulin stroma and
cells may persist without significant change
for the life of the individual (as in pneu-
moconiosis due to natural diatomite); the
reticulin stroma may become converted to
collagen and the cells largely disappear (as
in silicosis); the stroma and cells may fo-
cally undergo necrosis (as in Mycobacte-
rial or fungal infections); or mixtures of
these changes may eventuate.

The proliferation of alveolar cells and
associated reticulin stroma thickens the
alveolar wall and correspondingly narrows
the air space into which the alveolar cellular
accretion extends. This cellular accretion
may progress until the entire air space is
obliterated. The capillary of such an al-
veolar wall is usually not visible and is
probably obliterated. The intra-alveolar
tissue, composed of proliferated alveolar
cells and reticulin stroma, is usually avas-
cular and does not receive oxygen from the
inhaled air. Degenerative changes in this
intra-alveolar tissue are, therefore, not un-
common.

ORGANIZING PNEUMONIA AND THE
HAMMAN-RICH SYNDROME

It has recently been demonstrated that
when an acute inflammation is superim-
posed upon a chronic interstitial pneumo-
mitis, the intra-alveolar tissue with its labile
reticulin stroma readily degenerates and
becomes separated from its parent alveolar
wall. Such sequestered tissue appears as
an island of nondescript matrix which, in
addition to remnants of alveolar cells and
reticulin, may contain fibrin, leukocytes,
and red blood cells. Because the degene-
rated remnants of alveolar cells in the se-
questered intra-alveolar tissue have been
interpreted as newly developed fibroblasts,
it has been assumed that there was here
organization of intra-alveolar exudate and
the concept of organizing pneumonia there-
by became firmly established.

It is an interesting fact that although
fibrous thickening of alveolar walls in "or-
ganizing pneumonia" has been noted fre-
quently, this finding has not been recog-
nized as pathognomonic of chronic interstitial pneumonitis. That an acute pneumococccic pneumonia superimposed upon a subclinical chronic interstitial pneumonitis would present a clinical picture different than is presented by the same type of pneumonia in previously healthy lungs is readily understandable. The protracted course, the failure of clinical and radiologic resolution, and the higher mortality would seem logical and expected sequel in the presence of chronic interstitial pneumonitis.

In the Hamman-Rich syndrome, the presence of a fibrous interstitial pneumonitis has been recognized as the main feature, but because clinically and pathologically an acute component is also present, the syndrome has been designated as an "acute diffuse interstitial fibrosis of the lungs." A recent study of these cases, however, has demonstrated that, similar to "organizing pneumonia," the acute component is an acute bronchopneumonia superimposed upon a hitherto clinically silent chronic interstitial pneumonitis. Thus, "organizing pneumonia" and the Hamman-Rich syndrome have some essential points of similarity. The important differences are the following: in "organizing pneumonia," the history indicates a recent episode of acute pneumonia which, although it may have been pneumococcal, was atypical and did not resolve. Furthermore, this pneumonia, clinically and radiologically, is usually fixed in position. There are also many cases of this disease which escape clinical diagnosis because the inflammation is focal and involves a relatively small portion of the lungs. These are diagnosed at necropsy, often only by microscopic examination. In contrast, the patient with the Hamman-Rich syndrome gives a longer history of increasing or recurring dyspnea. There is a migratory pneumonia, clearing in one area and developing in another. The patient is more dyspneic and much more seriously ill than can be accounted for by the pneumonia and he dies either from pulmonary inadequacy or cor pulmonale. Pathologically there is a diffuse, fairly uniform and generalized chronic interstitial pneumonitis which is usually much more pronounced than is the case in "organizing pneumonia," whereas the superimposed acute pneumonia is usually less severe in the Hamman-Rich syndrome than in "organizing pneumonia." Exceptions are those sporadic cases of "organizing pneumonia" which are diagnosed only at necropsy and where the disease is an incidental finding involving a relatively small portion of a lung. To summarize, both the Hamman-Rich syndrome and "organizing pneumonia," are examples of chronic interstitial pneumonitis with superimposed acute pneumonia, but with a more severe form of the chronic inflammation and a less severe form of the acute component in the Hamman-Rich syndrome, whereas the reverse obtains in clinically diagnosed "organizing pneumonia."

**THE INTRAPULMONARY LYMPHATIC TRANSPORT**

Within minutes after the intratracheal injection of dust particles, the presence of these particles was detected in the cannulated pulmonary lymph. A similar observation was made regarding intratracheally injected non-motile bacteria which quickly invaded the blood and were cultured from the peripheral blood. The lapse of time from the initial injection was too short to allow for phagocytosis. These demonstrations forced the conclusion that animate and inanimate particles penetrate the alveolar membrane without the aid of cells, as naked particles.

However, there is experimental evidence which indicates that such direct penetration of the alveolar membrane accounts for only a very small portion of the dust which ultimately reaches the satellite lymph nodes. If the direct penetration of the alveolar membrane by dust particles played an important role in the lymphatic transport of dust, then a significant portion of the original lung dust burden should be transported to the satellite lymph nodes within one month of its imposition because...
it is during this early period that the dust particles are most likely to be free upon the alveolar surface. This has not been found in a number of well-controlled investigations. Significant lymph node deposition of quartz dust was found only three months or more after the imposition of a lung dust burden. When it is recalled that within three months after the intratracheal injection of quartz dust, the air spaces have largely been cleared of dust, and the dust that remains in the lungs is at this time sequestered within well-circumscribed nodules, it appears that the quartz dust which is being transported to lymph nodes must be derived from that sequestered within the silicotic nodules. The manner in which this is accomplished is a matter for conjecture. The following would appear to be reasonable assumptions:

1. The destruction of alveolar basement membranes and capillaries which seems to occur early in the development of a silicotic nodule, removes delimiting barriers so that the accreted dust-filled cells thereby become situated within the lung interstitium.

2. In time, individual dust cells will die, disintegrate, and the dust particles become extracellular.

3. Extracellular dust particles become transported by the flow of tissue fluid which is normally oriented toward lymphatic vessels.

It should not be necessary to emphasize that these new concepts are to be regarded as working hypotheses and not as immutable laws. They should, however, replace older concepts that have outlived their usefulness. These new concepts, hopefully, may serve to stimulate research in renewed attempts to establish the truth.

SUMMARY

Some of the newer concepts of chronic lung diseases are briefly described. The following new concepts are included:

1. The pulmonary clearance mechanism
2. Pneumoconiosis
3. Tuberculopneumoconiosis
4. Chronic interstitial pneumonitis
5. Organizing pneumonia and the Hamman-Rich syndrome
6. Intrapulmonary lymphatic transport.

RESUMEN

Se han descrito algunos de los más nuevos conceptos sobre las enfermedades pulmonares crónicas, incluyendo:

1. El mecanismo de la eliminación pulmonar
2. Neumoconiosis
3. Tuberculopneumoconiosis
4. Neumonitis crónica intersticial
5. Neumonía organizada y el síndrome de Hamman-Rich

RESUMÉ

L’auteur décrit brièvement certains des concepts les plus nouveaux des atteintes pulmonaires chroniques. Ils concernent les conceptions suivantes:

1. le mécanisme de la "clearance" pulmonaire
2. les pneumoconioses;
3. les tuberculo-pneumoconioses;
4. la pneumonie chronique interstitielle;
5. la pneumonie fibrosante et le syndrome de Hamman-Rich;
6. la circulation lymphatique intrapulmonaire.

ZUSAMMENFASSUNG

Einige der neueren Auffassungen über chronische Lungenkrankheiten werden kurz dargestellt. Folgende neue Auffassungen werden aufgeführt:

1. Der pulmonale Clearance-Mechanismus
2. Pneumonokoniose
3. Tuberkulopneumonokoniose
4. Chronische interstitielle Pneumonie
5. Organisierende Pneumonie und Hamman-Rich-Syndrom
6. Intrapulmonaler Lymphstrom.

REFERENCES


A case of ingestion of household ammonium followed by gastric necrosis, esophageal, gastric, duodenal and jejunal stricture is presented. The patient, a 39-year-old woman, survived and ultimately had a jejunal pouch and retrosternal descending colon cervical esophagostomy performed, thus bypassing esophagus, stomach and duodenum. The postoperative period was uncomplicated.

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BYPASS CIRCULATION IN COMBINATION WITH HYPOTHERMIA IN CONGENITAL HEART SURGERY

Observations on 270 open heart operations performed for congenital heart diseases are reported. The following methods were utilized: heart bypass in hypothermia at 20-30°C; artificial circulation at normal temperature associated with cardioplegia induced by potassium citrate or local cooling of the heart to 8-12°C; deep hypothermia with cooling up to 8-12°C and circulatory arrest for 40 minutes, and the bypass circulation in combination with hypothermia until the onset of the ventricular fibrillation. The method of the bypass circulation associated with hypothermia seems to be very advantageous and most atraumatic since during cooling, the body becomes more resistant to the operative trauma and oxygen deficiency, the blood damage in the apparatus is lessened and a surgeon is able to manipulate on fibrillating heart, the blood flow in its vessels being maintained.

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