POST-OPERATIVE ATELECTASIS

Part I: Reflex Bronchial Constriction as an important
Aetiological Factor in Post-operative Atelectasis.

Part II: An Investigation of Atelectasis complicating
Thoracoplasty in Pulmonary Tuberculosis.

By

Kenneth Clark Grigor, M.B., Ch.B., D.A.
Consultant Anaesthetist,
The Victoria Infirmary, Glasgow,
and
The Thoracic Surgical Unit, Mearnskirk Hospital,
Newton Mearns.

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Preface.

My association in the capacity of anaesthetist with the Thoracic Surgical Unit, formerly at Ruchill Fever Hospital, Glasgow, and now at Mearnskirk Hospital, Newtonmearns, dates from January, 1942.   At that time the Unit was in its infancy and, as might be expected on account of the diversion of medical and surgical supplies to the armed forces, it suffered from a lack of essential items of equipment without which operations on the chest are well nigh impossible.   The small operating theatre was barely adequate and there was no portable x-ray apparatus available.   Indeed it was a major undertaking to procure the very necessary radiographic examination of patients recently submitted to an operation such as thoracoplasty or lobectomy as it entailed a journey by ambulance to a remote part of the hospital. Under these circumstances few such examinations could be carried out at Ruchill Hospital.

There was, however, no lack of keenness among the personnel of the Unit. Under the able direction of Mr. Bruce Dick, the visiting surgeon, and the dynamic enthusiasms of his assistant Mr. R. S. Barclay, a surgical team was formed and it remains substantially unchanged at the present time.

At the outset, as is the case with most young anaesthetists, my attention was largely focussed on the actual administration of anaesthetics during operation. The field of thoracic surgery, being almost entirely novel,

confronted one with many problems of anaesthetic technique which had not been hitherto encountered. The anaesthetist who will play a full part in the surgical team must, however, concern himself with the preoperative preparation and post-operative care of the patient. It soon became apparent that post-operative atelectasis when it complicated thoracic operations, was a very unfavourable occurrence. Especially was this the case when it occurred following the operation of thoracoplasty performed in the course of treatment of pulmonary tuberculosis.

Post-operative atelectasis is considered by many to be a post-anaesthetic phenomenon. It therefore fell to my lot, with every encouragement and assistance from the surgeons, to study this most baffling problem.

When the Thoracic Unit moved to Mearnskirk Hospital in November, 1946 deficiencies which had previously hampered the conduct of an adequate investigation of atelectasis complicating thoracoplasty were made good. A physiotherapist joined the surgical team and the very necessary portable radiography became available.

It is the popular conception that this type of atelectasis is caused by obstruction of bronchi by mucus plugs which accumulate because of weakened respiratory force and suppressed cough and that air, distal to the point of obstruction, is absorbed by the blood stream. This conception does not take into account the complex mechanism

normally made use of by the lung to get rid of foreign
material from the respiratory passages.   Bronchial
movement and ciliary action get very little mention in
contemporary literature dealing with the subject of post-
operative atelectasis.   As Hilding (1944) says, "one can
as well omit cardiac action from a discussion of the
physiology and pathology of the circulatory system, or
peristalsis from a discussion of the gastrointestinal
tract, as to disregard ciliary action when speaking of the
respiratory tract."

Ones own experience has led to the belief that
bronchial constriction might be a potent factor in causing
arrest and immobilization of secretions in the bronchial
tree; indeed the bronchial constriction might be the cause
of stimulating mucus secretion in many cases.   Thus it was
decided to undertake an extensive review of the literature
on the subject of post-operative atelectasis in the hope
that, by correlating personal observations with the findings
of others, it might be possible to evolve a theory of the
causation of this type of atelectasis more widely applicable
than the theory popularly held at the present time.

The study is presented in two parts.   Part I deals
with post-operative atelectasis generally.   The conception
that reflex bronchial constriction is an important
aetiological factor in the production of post-operative
atelectasis is developed.   Part II is concerned with

atelectasis following thoracoplasty in pulmonary
tuberculosis. An assessment of the morbidity caused by
post-operative atelectasis and an evaluation of the
factors considered likely to predispose to its occurrence
are presented.

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C O N T E N T S

Chapter III.

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PART I: REFLEX BRONCHIAL CONSTRICTION AS AN IMPORTANT AETIOLOGICAL FACTOR IN POST-OPERATIVE ATELECTASIS.

Chapter I.

History.

The story of atelectasis covers more than a century of time and an enormous literature has grown up around the subject. Keen observations by the master clinicians of the past, innumerable ingenious experiments, the advent of radiography and other modern methods of investigation have all played important roles in elucidating many of the facets of this most intricate problem. It may be doubted, however, whether all the pieces of the jig-saw have yet been found: they certainly have not all been pieced together. In this historical account an attempt is made to unfold the clinical picture, the theories of pathogenesis and other relevant matter concerning post-operative atelectasis as these were revealed by the various authors on the subject.

The term atelectasis was apparently first used by Jorg (1832) and is derived from two Greek words, "ateles" meaning imperfect and "ektasis" meaning expansion. He described atelectasis as a clinical entity arising in post-natal life and showed that it was often lobular in distribution. He was the first to point out that, by inflation of the lungs, the condition could be distinguished from the hepatization of pneumonia. Jorg ascribed the imperfect expansion of the

lung to weakness on the part of the child and to a too
precipitate birth which, he considered, interfered with the
establishment of respiration.

Legendre and Bailly (1844) described a state of the
lung, particular to infancy and most frequent between the
ages of two and five, in which the lung could return in part
to the atelectatic state after expansion had occurred at
birth.   This they called "l'etat foetal".   The condition
could occur independently of any pulmonary inflammation or
be associated with pulmonary catarrh.   The latter type they
named "l'etat foetal congestionnel".   Whether the occlusion
of the air vesicles was simple or congestive in type, they
demonstrated that inflation restored them to their normal
air-bearing aspect.   From these experiments they deduced
that what had previously been described as lobular pneumonia
in young children, was merely a collapse of the air cells.
Feeble children were the victims and abundant, viscid
mucopus in the bronchial tubes frequently militated against
free circulation of air in the bronchioles and alveoli.   In
other cases the distension of the blood vessels of the
vascular network external to the vesicles or the constriction
of swaddling clothes compressing the chest might be causative
factors.   It is interesting to note that Legendre and
Bailly advised that children suffering from this condition
be treated by frequent changes of posture, the avoidance of
prolonged dorsal decubitus and tight binders which interfered

with proper expansion of the lung bases.

Thus a condition of airlessness or collapse of the lung to be differentiated from the consolidation of pneumonia was fast becoming recognized. Various tentative theories as to its causation had been hazarded but the pathogenesis was little understood. However, the experiments of Mendelsohn (1845) and Traube (1846) helped to throw some light on the subject. Mendelsohn inserted a shot or slug through a tracheotomy opening in a rabbit's trachea and impacted it into the left bronchus by means of a probe. The animal died in two days and at post mortem examination the left lung was found to be collapsed; the greater part of the lower lobe and some portions of the upper lobe were airless. The whole lung could be inflated after removal of the obstruction. The right lung was seen to be emphysematous. In other instances, paper balls and solutions of gum were employed with similar results. Traube performed almost identical but more numerous experiments employing shot and gum arabic as his obstructing agents. His results were substantially in agreement with those of Mendelsohn. A feature of the publications by Legendre and Bailly, Mendelsohn and Traube was their failure to give any credit to the work of Jorg.

It was now recognized that obstruction of a bronchus could lead to an airless state of that portion of the lung beyond the point of obstruction: clinically Legendre and

Bailly had suggested that the presence of viscid, muco-
purulent sputum in the bronchi of children up to the age of
five years might lead to a partial reversal to the
atelectatic state of the foetal lung.   It was not, however,
until 1850 that a satisfactory description of "collapse or
condensation of the lung" as it occurred in adult subjects
appeared.   Gairdner (1850, 1851a, 1851b, 1853) in a series
of papers published between 1850 and 1853, carried the
subject several stages further.   In the capacity of
pathologist to the Royal Infirmary, Edinburgh, Gairdner
carried out many careful examinations of the lungs of
patients who had died of bronchitis.   He described two
varieties of what he called bronchitic collapse of the lung;
(a) a diffused form and (b) a limited or lobular form.   In
the diffused variety, the collapse frequently affected a
considerable portion of one or both lungs, usually at the
posterior parts and passing gradually into normal tissue.
In the lobular form a patchy distribution was present, the
collapsed areas being accurately and abruptly demarcated
by the interlobular septa; when the areas of collapse
occurred at the anterior edges of the lung, they were seen
macroscopically to be sunk below the level of the
surrounding parts.   Both forms presented the same
fundamental changes of the pulmonary tissue "which is usually
of a dark violet colour externally, as seen beneath the
pleura; and internally of a more or less deep, brownish red,

or mahogany tint.   The affected parts are always more or
less condensed; this condensation may amount to a mere
diminution of the crepitation, or to a total absence of it,
in which case portions are usually found to sink readily in
water."   Gairdner thus carefully differentiated atelectatic
condensation from pneumonic consolidation both by its
microscopic and macroscopic appearance.

Summing up the occurrence of collapse of the lung in
bronchitis, Gairdner (1851a) enumerated the causes as, (a)
bronchial obstruction by tenacious and viscid mucopus, (b)
weakness and inefficiency of the inspiratory power and (c)
inability to cough and expectorate and thus to remove the
obstructing mucus.   He mentioned that cure could be
effected by removal of the obstruction; that sputum could
be removed by muscular contractions of the bronchi and by
ciliary action; that any factor interfering with the de-
obstruent function was a possible cause of bronchial
accumulation even in the normal state of the mucous
membrane; that factors affecting the vagus nerve might
impair or stimulate the removal of secretion.

The above sums up much of our knowledge of this
subject at the present day.   Gairdner cited the case of a
debilitated child in whom, during an attack of acute
bronchitis, collapse of the right lung supervened with
dyspnoea and dullness of the lung base.   Under the
influence of an emetic and a forced decubitus on the

opposite side to that on which the dullness existed, almost
every trace of it had disappeared in thirty-six hours, and
the dyspnoea was entirely relieved.   After the lapse of
almost one hundred years, during which time Gairdner's work
has been almost completely forgotten, this method has
become the basis of the present day conservative treatment
of post-operative pulmonary atelectasis.   A truly great man
would be great in any age.

There still remained to be explained the means whereby
the air disappeared from the collapsed areas of the lung.
Gairdner (1851b) had suggested a ball-valve mechanism, the
obstructing plug of secretion moving in the bronchus with
respiration.   This allowed expulsion of air on expiration
but, on inspiration, the plug was sucked back to its former
position where it completely occluded the tapering bronchus.
Bartels (1860) considered that mechanical expulsion of air
alone did not satisfactorily explain the production of
atelectasis in complete bronchial obstruction.   In 1879
Lichtheim (1879) provided an answer.   He had been
repeating the work of Mendelsohn and Traube and found that
by tying a rabbit's bronchus, atelectasis resulted.   He also
noticed that the homolateral diaphragm was markedly displaced
in an upward direction.   It occurred to him that the air
from the collapsed portion of the lung might be absorbed by
the blood stream.   Accordingly he resected portions of a
rabbit's third and fourth ribs and placed a ligature loosely

round the hilum of that side.   He then inflated the lung,
made his ligature fast and closed the chest.   No absorption
of air resulted.   He next tied the pulmonary artery alone -
again no collapse of lung occurred.   This, in conjunction
with his previous findings, led Lichtheim to the conclusion
that the integrity of the pulmonary circulation was
necessary for the production of atelectasis.   Later he
devised ingenious experiments to measure the rate of
absorption of the gaseous components of the entrapped air.
He found that carbon dioxide was absorbed somewhat more
quickly than oxygen and that nitrogen took considerably
longer than either to disappear.   The time for air, as
might be expected, was less than for nitrogen but
proportionately longer than for oxygen and carbon dioxide.

        Gairdner's and Lichtheim's work was quickly forgotten
and it was not until William Pasteur (1890) reported
pulmonary collapse complicating diphtheria that interest
was once more aroused.   Pasteur's attention was directed
to the subject during a severe epidemic of diphtheria in
the east end of London when he saw many cases of post-
diphtheritic paralysis in the wards of the Queen's Hospital
for Children.   His paper was the outcome of observations
made on fifteen cases of diphtheritic paralysis involving
the muscles of ordinary respiration, especially the
diaphragm.   Paralysis of the palate and pharynx was
present in every case and tube feeding had to be carried

out.   A short, non-explosive, ineffectual cough was a
common feature in these cases.   He described increased
movement of the lower ribs, elevation of the diaphragm,
recession of the epigastrium on expiration and defective
air entry into the lung bases.   Recovery, when it took
place, was usually quite dramatic, normal respiration
being restored with marked alleviation of the respiratory
distress.   On the other hand typical post mortem
findings in cases which ended fatally were an airless
state of the lung bases with scattered areas of broncho-
pneumonia.   Pasteur concluded that paralysis of the
diaphragm or other parts of the chest wall tended to
induce loss of function in the subjacent lung resulting
in collapse of the pulmonary tissue.   Some of his
descriptions are very suggestive of atelectasis following
bronchial obstruction.   At this time he did not note
cardiac displacement but in most cases the collapse was
bilateral.

The term massive collapse of the lung was introduced
into medical literature by Pasteur in his Bradshaw Lecture
of 1908.   To Pasteur must go the credit of impressing
massive collapse of the lung on the clinical consciousness.
In a series of papers (Pasteur 1910, 1911, 1914a, 1914b)
he pointed out the frequency of pulmonary collapse as a
post-operative complication.   While still ascribing the
cause to paralysis or reflex inhibition of the diaphragm,

he developed with precision the clinical picture of the
condition and showed clearly the need to distinguish it
from the so-called post-anaesthetic pneumonia and even,
at times, from embolism.　　He described how it could
occur following ether or chloroform anaesthesia, usually
after abdominal operations but less frequently after
thyroidectomy and might develop at any time within the
first week of operation.　　After a few hours, during
which time the patient usually complained of an uneasy
feeling in the chest and showed a rising temperature,
there was a more rapid development of signs and symptoms.
Pain in the chest became acute, cyanosis developed, the
respirations were accelerated and the pulse rapid and
feeble.　　There was limited or no movement of the lower
part of the chest on the affected side, the percussion
note was dull, and the breath sounds over the dull area,
at first weak, later became tubular with whispering
pectoriloquy.　　The other side of the chest was often
distended and emphysematous and a diagnosis of pneumo-
thorax might mistakenly be made.　　The homolateral
diaphragm was raised and the heart displaced towards the
area of dullness.　　This displacement of the heart might
amount to several inches thus distinguishing the state of
collapse from pneumonia or pulmonary embolism.　　If the
collapse was bilateral cardiac displacement did not occur.
The shift of the mediastinum and emphysema of the contra-
lateral lung he recognized as compensatory phenomena due

to an increased isolateral negative intrapleural pressure.
Haemoptysis was not a feature but there was generally a
mucopurulent expectoration.

In his Bradshaw lecture, Pasteur described massive
collapse (he later (Pasteur 1910) preferred the term active
lobar collapse) as an active process brought about by the
elastic property of the lung "taking charge" when the
muscular force of the paralysed leaf of the diaphragm was
no longer capable of maintaining expansion. As a result
the affected portion of the lung emptied itself of
contained air. He differentiated this type from the
scattered, lobular or patchy collapse that occurred
passively when the bronchioles were obstructed by secretion
and the corresponding alveoli slowly emptied by absorption
of the contained air into the circulating blood. Massive
collapse occurred characteristically at the lung bases, that
is in the areas corresponding to the sphere of action of the
diaphragm. The side affected tended to correspond to the
site of abdominal operation although it appeared rarely as
a contralateral phenomenon. Two years later (Paster 1914a)
he suggested that in abdominal surgery the pneumogastric
nerve was the afferent source of the reflex diaphragmatic
inhibition. In 1914 (Pasteur 1914b) he differentiated two
well defined clinical types of postoperative collapse of
the lung: (a) an acute variety in which the onset was sudden
and accompanied by initial pain and dyspnoea.

(b) a latent type where there may be complete absence of symptoms although the physical signs were well developed. Tubular breathing was best heard in the latent type whereas in most acute cases breath sounds were absent. (This remained a puzzling feature for many years and Lander and Davidson (1938a), whose experiments are described later, were the first to advance a satisfactory explanation.) Pasteur ascribed the early production of mucopus to squeezing out of secretion from the collapsing lobe. As treatment he suggested that the patient be propped up, that oxygen be administered, and that the patient be encouraged to inspire deeply at intervals even at the cost of some discomfort. He advocated wider use of pre-operative medication with morphine sulphate and atropine sulphate and made a plea for energetic treatment of oral sepsis before a major operation was undertaken.

Thanks to the energetic Pasteur and his literary bombardments, post-operative pulmonary collapse had now become firmly established as a clinical entity. Before his famous Bradshaw Lecture of 1908 no case of collapse had appeared in the surgical records of the Middlesex Hospital. Subsequently, as he himself pointed out, there occurred a gradual increase in the number of cases and a corresponding decrease in the incidence of post-operative pneumonia. In spite of the fact that in his later writings Pasteur appeared less dogmatic as to the

cause of the pulmonary collapse, his views on the pathogenesis of the condition seem to have been accepted without question.   A leading article in the Lancet of 1910 agreed that dysfunction of the hemi-diaphragm initiated the collapse and Tidy and Phillips (1914), concurred with this opinion.   A search of the literature of that time revealed no opposing point of view.

It remained for Elliot and Dingley (1914) to re-describe the importance of the sputum.   They published details of 11 cases of massive collapse of the lung following abdominal operation.   One of their cases occurred under spinal anaesthesia, all had fever and thick, muco-purulent expectoration preceding the collapse. Recalling the work of Lichtheim (1879), they postulated that secretions resulting from infection caused complete bronchial obstruction and subsequent absorption of alveolar air into the circulating blood.   They also considered, one feels in deference to Pasteur, that inhibition of the diaphragm might be a factor.   By way of investigating Pasteur's theory they, in animal experiments, effected one-sided paralysis of the diaphragm and intercostal muscles; no pulmonary collapse resulted after several days under prolonged ether anaesthesia. They advanced treatment a stage further by condemning tight abdominal binders which interfered with the respiratory excursion.   Further, they anticipated a later development

when they wrote, "it is conceivable that the worst cases
of cyanosis and pain might be relieved by an intrapleural
injection of oxygen in order to restor the balance of
volume and push back the displaced heart and the over
distended lung."

The advent of the 1914-18 war was to arouse a new
interest in massive collapse of the lung and to add still
further to the difficulty of a clear understanding of its
causation.   A concentrated study of chest wounds revealed
that collapse of the lung in whole or in part was a frequent
occurrence and in many instances signs were present which
could not be attributed to any of the generally accepted
pathological conditions.   In civil practice it was
recognized that collapse of the lung might occur as a
sequel to operation, especially operations on the upper
abdomen.   In military experience it came to be associated
with injuries, not necessarily penetrating, of the chest
wall or lung.   Indeed cases were reported (Bradford 1918 and
1920) following trivial injuries of the abdomen and buttock
where the wounds were so slight that the patient had not
been confined to bed prior to the onset of collapse and
where no anaesthetic had been given in the course of
treatment.   Further the condition occurred in soldiers who
had been in perfect health with no suggestion of previous
lung disease up to the time of wounding.   Cases were
observed where massive collapse involving the entire lung

occurred on the side opposite to that wounded and as a
result of a trivial, non-penetrating wound of the chest
wall.   Extreme cardiac displacement might be present
without the patient being aware of any untoward discomfort
apart from dyspnoea on exertion.   Bradford (1918) reported
the case of a soldier who was admitted to hospital on the
evening of the day on which he was wounded and had collapse
involving the whole of the left lung; he had no urgent
symptoms and had walked four miles after being wounded.
This type of case usually presented diminished respiratory
movement of the affected side, dullness to percussion over
the affected lung tissue, with absent or tubular breath
sounds but no rales or adventitiae.   The site was usually
contralateral and the condition had been known to clear up
within twenty-four hours.   More frequently, however, the
typical clinical picture as described by Pasteur was
present with pain, dyspnoea, cyanosis, cough, sputum, fever,
etc.   Bradford noted that, where the pulmonary collapse was
confined to an upper lobe, the heart tended to be rotated
and its apex displaced upwards.   The character of the
respiratory murmur, he thought, depended upon the patency
or the occlusion of the larger bronchi of the collapsed
portion of the lung.   The condition sometimes lasted as
long as two to three weeks but, on an average, it resolved
by the tenth day.   Complications, when they occurred,
were bronchitis, pleurisy, pneumonia or oedema of the lung.

During this period Gask and Wilkinson (1917) reported a series of 500 cases of gunshot wounds of the chest and found massive collapse in approximately 10 per cent of their cases (contralateral in one per cent). Crymble (1918) described the radiological appearances in 15 cases of massive collapse of the lung following gunshot wounds of the chest. A uniform opacity of the collapsed portion of the lung with retraction of the heart and mediastinum towards the opaque side and elevation of the homolateral diaphragm were the features noted. Fluoroscopy showed that the affected leaf of the diaphragm was immobile. In one case the whole lung was opaque, in another the upper lobe alone was involved; 10 cases appeared on the side opposite to that wounded and the left lower lobe was the most frequently affected. It is of interest to refer to a case reported by Crymble. A shrapnel ball, injuring the thoracic spine of a soldier, resulted in paralysis of all the muscles of respiration with the exception of the left hemi-diaphragm. There was loss of sensation below the level of the third costal cartilage, paralysis of the right arm, the lower extremities, the abdominal and inter-costal muscles. There was no collapse of the lung.

On the subject of pathogenesis, some confusion was now to arise. Pasteur's theory of paralysis or inhibition of the hemi-diaphragm had lost much of its former popularity. Mechanically obstructing sputum of Elliot and Dingley could

not explain the many cases where, even at post mortem, no intra-bronchial secretion was demonstrable and no infection apparent. Gask and Wilkinson, admitting that theories as to aetiology were numerous but unsatisfactory, could advance no reasons to account for the lung collapse. Crymble, while considering that bronchial obstruction was a possible factor, suggested that the condition could be the result of a reflex phenomenon of obscure origin. Bradford, who appears to have made the most complete study of the subject, found it difficult to imagine how bronchial obstruction by secretion could cause complete collapse of the lung in many of his cases. He inclined to the view that in the contra-lateral variety a reflex spasm of the bronchioles, possibly associated with a reflex palsy of the respiratory muscles, was the most probable explanation. Purulent bronchitis, when it occurred, was secondary to the collapse of a lobe or lobes.

Briscoe (1920) focussed attention once again on dysfunction of the diaphragm and deflation of the lung as being the primary factors involved in post-operative massive collapse. After an exhaustive research into the mechanism of respiration in the erect and supine postures he concluded that partial deflation of the lower lobes of the lungs occurred in the supine position, particularly if this posture was maintained for any length of time. The crura were the essential portions of the diaphragm

involved in the respiratory function of one type of
individual whom he called the crural type. In a second
or parietal type, the action of the costal portion of the
diaphragm was the more predominant. A large chest and
flat abdomen characterized the physique of the crural
individual while his counterpart possessed a long, narrow
chest and protuberant abdomen. The crural variety of
breather was the more likely candidate for post-operative
massive collapse. Briscoe's conception of the phenomena
was as follows: Inflammation attacked the pleura covering
the involved diaphragm, especially that part of it in the
region of the crus, or affected the muscle of the crus
situated beneath the peritoneum in a patient in whom the
lower lobe of the lung was already partially deflated.
This inflammation caused the pain and the pyrexia and, he
contended, put the hemi-diaphragm and its synergic and
antagonistic muscles out of action. Thus complete
deflation of the corresponding lung or portion of lung
resulted. The infection came possibly by way of the
lymphatics from the abdominal cavity or from an infected
wound. In cases characterized by a frequent unproductive
cough, secretions had accumulated in the larger bronchi.
The lower lobes being deflated, there was no opportunity
for a volume of air to accumulate behind the mucus. When
normal respiratory function was restored by the patient
becoming more active, the deflated lung re-expanded.

Displacement of the heart and mediastinum was not mentioned.

The first account of post-operative massive collapse of
the lung in the medical literature of the United States of
America appeared in 1921 when Scrimger (1921) published
details of seven cases, six following operations on the
lower abdomen and one after haemorrhoidectomy.   His
descriptions of signs and symptoms conformed to those of
previous publications but he had made more frequent
radiological and fluorescent screen observations than had
hitherto been detailed.   Scrimger noted that the actual
size of the lung in massive collapse was notably smaller
than any expiratory state of the chest wall or diaphragm
would explain.   This was shown by the greatly arched hemi-
diaphragm which, in two instances, was seen to be higher
than it normally would be after section of one of the
phrenic nerves.   Fluoroscopy also showed in two instances
the homolateral diaphragm to be high and immobile days after
the corresponding lung, except for a small portion at the
base, had cleared and re-expanded.   The heart also might
not return to its normal position for some time after re-
inflation.   The theory was hazarded that operative
abdominal interference, acting through the vagus nerve,
caused a contraction of the muscular elements of the lung;
subsequently a collection of mucus produced bronchial
obstruction with resultant absorption of alveolar air.

American interest was now stimulated and several

writers described cases.   Scott (1925) reviewed the
literature.   Apart from war wounds he found 64 cases
of post-operative massive collapse, only 36 of which were
given in sufficient detail for analysis.   To these he
added four cases of his own showing little variation from
those previously described.   The most striking post
mortem feature he considered to be the extreme pulmonary
congestion and, as a result, he favoured an initial reflex,
probably vasodilative in character, as the primary cause
of the condition.

Towards the end of the same year, Jackson and Lee
(1925) described bronchoscopic aspiration of the bronchial
tree, a notable contribution in regard to treatment of
post-operative pulmonary collapse.   Jackson's famous
phrase "the cough is the watchdog of the lung" gained much
popularity.   They called attention, as had Scott (1925)
and others, to the inaccuracy of the term collapse when
applied to this condition.   Collapse implied deflation
of the alveolar tissues and bronchi from a positive
intrapleural pressure as was present in conditions such as
pneumothorax.   They considered the term atelectasis to be
more descriptive as implying an airless state of the
pulmonary tissue; this has come to be the popular view and
the term atelectasis will be used in the remainder of this
work.

Manges (1924) had shown that non-opaque foreign bodies

obstructing a large bronchus could be diagnosed
radiologically by the resultant atelectasis. Jackson
was the first to call attention to the similarity of the
radiological picture before and after removal of foreign
bodies through the bronchoscope, to the picture of massive
atelectasis encountered in pulmonary complications
following operation. He suggested that a bronchoscopic
examination might not only determine the aetiology of
this condition but also serve as a therapeutic procedure.
He had successfully treated atelectasis complicating
diphtheria by bronchoscopic aspiration of diphtheritic
membrane from the bronchi and thus tended to provide an
explanation for the early cases of Pasteur (1890).

In this paper by Jackson and Lee (1925) there appears
an account of the first instance of post-operative
atelectasis to be treated by bronchoscopic aspiration.
The case was one of atelectasis of the right middle
and lower lobes following appendicectomy. The
manoeuvre was executed by Dr. Tucker and it is of interest
to note the following among his findings:- there was
marked inflammatory reaction in the bronchi of the right
lung which contained a tenacious mucus entirely closing
up some of the air passages and marked restriction of the
bronchial movements on the affected side; the inspiratory
lengthening and opening of the bronchi was very slight,
particularly in the lower and middle lobe bronchi. A

subsequent bronchoscopy was performed following recovery
four days later when it was noted that the bronchi
lengthened and dilated in the normal manner during
inspiration.

Jackson and Lee stressed the need for early treatment,
by bronchoscopic aspiration if need be, of post-operative
atelectasis.   In the untreated case purulent bronchitis,
pneumonia or pleurisy might ensue.   They called attention
to the less obvious lobular or patchy forms and were
convinced that the great majority of post-operative
pneumonias were really atelectatic in origin.

It might be convenient at this juncture briefly to
take stock of the progress so far made by the actors in
this historical account.   The clinical picture of post-
operative atelectasis has been established with accuracy
but it is not clear why some cases should present a
diminished or absent respiratory murmur over the affected
area while in others the signs are those of pneumonic
consolidation.   Clinical types recognized are acute or
latent in character and these may be classified as
massive (multilobar), lobar and lobular (patchy)
atelectasis.   The condition usually appears within the
first few days of operation and resolution may occur
dramatically or be delayed for a period of days or weeks.
Pneumonia is the complication to be dreaded.   As regards
treatment periodic deep breathing unrestricted by tight

abdominal binders, change of posture, the administration of
oxygen, aids to expectoration and bronchoscopic aspiration
have been advocated.   It is generally agreed that
abdominal surgery, general anaesthesia, and previous
respiratory infection are important, but not necessarily
constant, aetiological factors.   The weight of evidence
seems to favour the mechanism of simple bronchial
obstruction by retained secretions with subsequent
absorption of the entrapped alveolar air as the primary
cause.   An active deflation or reflex spasm of the
muscular elements of the lung have, however, received
consideration and Scott has further complicated the
picture with his suggestion of a vasomotor reflex.

The obscurity that obviously clouded the pathogenesis
of post-operative atelectasis seems to have been accepted
as a challenge by American authors.   The year 1927 was
to see many additional reports and discussions on the
subject, each contributor propounding his own favourite
theory of causation.   Thus we have Sante (1928)
supposing an inhibition of the cough reflex by some toxic
or reflex stimulus associated with an impairment of the
respiratory muscles.   Reporting on, and reviewing the
literature of the incidence of contralateral atelectasis,
Scott and Joelson (1927) felt justified in concluding that,
when the patient had been consistently lying on one side,
both at operation and subsequently, if atelectasis

developed it would occur in the dependent lung.   They
further enlarged on the postulate of a vasomotor reflex
mechanism originally proposed by Scott (1925) and
suggested that air might be expelled by a reverse,
check-valve mechanism.   Bergamini and Shepard (1927)
reported a case of sudden death during operation where
post mortem examination revealed massive atelectasis of
both lungs.   There was no apparent bronchial obstruction
but the capillaries, arterioles and venules of the lungs
were uniformly dilated and filled with blood.   Their
theory was that the cause of the atelectasis was reflex
in origin and probably akin to angioneurotic oedema.
Churchill and Holmes (1927) thought the combined effect
of obstruction of the bronchioles by inflammatory exudate
and reflex immobility of the diaphragm to be the most
likely factors involved.   Mastics, Spittler and McNamee
(1927) in an excellent paper on the subject again called
attention to the cough reflex.

Mastics et al. (1927) considered that atelectasis
accounted for 70 per cent of all post-operative pulmonary
complications and that partial atelectasis was more common
than the massive variety.   It could occur after spinal,
local, regional or general anaesthesia and many of the
cases were missed for lack of a thorough post-operative
examination.   A rise of temperature and pulse rate
constantly presented and, in the uncomplicated case, only

a mild leucocytosis occurred.   The burden of heat
dissipation that the lungs were able to assume in
atelectasis was limited, hence the pyrexia.   The
proportion of males to females was three to one.   The
site of election in order of increasing frequency was
the posterior portion of the right lower lobe, the right
lower and middle lobes together, the left lower lobe, the
right upper lobe and the left upper lobe. (Holt and
Howland (1922) had previously drawn attention to the fact
that the posterior portion of the right lower lobe was
the area of the lung most commonly involved in the
atelectasis of premature or weak infants).   A typical
radiograph showed that the ribs converged on the affected
side and diverged on the opposite side.   The most marked
deviation of the trachea was seen when the upper lobes,
and particularly the right upper lobe, were affected.
Churchill (1925) described the radiological appearance
of an atelectatic right lower lobe as roughly that of a
right-angled triangular opacity, the base of which rested
on the diaphragm, the apex was at the hilum of the lung
and the hypotenuse showed a sharply defined border
extending from the hilum to the diaphragm near the costo-
phrenic angle.   Later Churchill and Holmes (1927)
described how the opacity on the left side was largely
obscured by the heart shadow.   Sante (1928) described a
method of treatment by rolling the patient back and forth

on his unaffected side.   This manoeuvre, which bears the
originator's name, frequently resulted in coughing spasm
with the expectoration of retained secretions.   Farris
(1925) induced an artificial pneumothorax on the affected
side of the chest to relieve the embarrassment resulting
from the lowered isolateral intrapleural pressure.   He
reported that benefit accrued and he urged the use of this
procedure in established cases of atelectasis.   Elkin
(1927) advocated the diagnostic value of estimating the
intrapleural pressures.   In one of his cases of post-
operative massive atelectasis he found a concomitant but
lesser increase in the negativity of the intrapleural
pressures on the contralateral side.

        It will be convenient here to recount three further
observations, although not in strict chronological order,
before embarking upon an account of experimental studies
in pulmonary atelectasis.   Brown (1930) reported
bronchoscopic observations in post-operative atelectasis
and following therapeutic hyper-ventilation with oxygen
and carbon dioxide.   The effects of this hyper-
ventilation as seen through the bronchoscope were: (a) an
increased rate and depth of ventilation; (b) violent
movements of the tracheo-bronchial tree and alterations
in the shape of the lumen of the bronchi, thereby tending
to free adherent mucus; (c) distinct blanching of the
mucus membrane of the trachea and bronchi.   He also

noticed that bronchial oedema was present in atelectasis.
In the same year Overholt, Pendergrass and Leopold (1930)
reported that in a series of 25 cases submitted to upper
abdominal surgery, the vital capacity on the first post-
operative day averaged 33 per cent of the pre-operative
figure. Overholt (1930) later suggested that the
entrance of air into the peritoneal cavity at laparotomy
might be responsible in part for the reduction in
pulmonary ventilation following operation by causing
elevation and partial limitation of the motion of the
diaphragm.

With so many aetiological factors apparently involved
in the production of post-operative atelectasis it was
natural to expect that experimental studies would be
carried out in an effort to elucidate the problem. Many
carefully planned experiments have indeed been performed
mostly on dogs.   It is debatable to what extent such
experiments are vitiated by the fact that in the dog the
thorax is functionally one cavity (Wright, 1945); also
in the quadruped the trachea is horizontal and the
mechanics of respiration and particularly of coughing
cannot be compared with those of the human subject (Maxwell,
1938).

The following experiment by Lee, Ravdin, Tucker and
Pendergrass (1928) was the first of its kind. Seven cubic
centimetres of sputum from a human case of post-operative

atelectasis were inserted into the right main bronchus of an anaesthetised dog by means of the bronchoscope; after the peritoneal cavity had been opened through a right rectus incision, the wound was closed by continuous layer sutures of silk, and the abdominal wall encircled by broad adhesive plaster. Intraperitoneal sodium amytal produced deep narcosis and eliminated the cough reflex. Thus the conditions of laparotomy were simulated in the dog. The animal was kept lying on its side for three hours. Signs of respiratory distress soon appeared, movements of the right chest almost ceased and distinct increase in size of the left chest became obvious. A radiological examination after three hours showed total atelectasis of the entire right lung with retraction of the heart.

This was the forerunner of many similar and more elaborate experiments; those worthy of note were conducted by Coryllos (1930), Coryllos and Birnbaum (1930,1933), Van Allen and Adams (1930) and Val Allen and Lindskog (1930). Throughout these experiments, the operators experienced considerable technical difficulty in occluding the selected bronchus or bronchi of their dogs. In fact it was found to be almost impossible to produce obstructive lesions if the animal was not completely and deeply anaesthetised for several hours after the obstruction. Coryllos and Birnbaum found it necessary to use a small piano wire spring device attached to their obstructing balloon to avoid its expulsion.

Coryllos and Birnbaum in their voluminous writings emphasised again and again their belief that there was only one final mechanism in the production of post-operative atelectasis, namely complete bronchial obstruction by secretions with absorption of the alveolar gases by the circulating blood. They advocated that the term apneumatosis (airlessness) be substituted for atelectasis. So anxious did they appear to have their ideas accepted that they conducted several hundreds of experiments of a most elaborate nature. With both pleural cavities widely open they kept dogs alive in an oscillating negative pressure box. The animals' lungs were submitted to pressure variations equal to those recorded in the individual dog's intrapleural spaces before operation. The rate of oscillation was made equal to the dogs' normal rate of respiration. Obstructing a main bronchus and ingeniously effecting gas analysis of the air in the obstructed alveoli, they observed the various stages of atelectasis developing. On one occasion they secured a moving film of the process. They found that immediately after successful obstruction the lung stopped "breathing" whereas the normal lung increased in size. Little by little the volume of the occluded lung decreased as a whole without conspicuous change in its general shape or colour and it gradually sank towards the costovertebral sinus. The heart was displaced towards the obstructed lung.

Except for a slight cyanotic hue there was no other change until the volume of the affected lung had decreased to about one fifth or one seventh of its normal size. There then appeared dark, bluish-brown patches scattered all over its surface without any special predilection for the hilum or periphery as was noted by Van Allen and Adams (1930). After the great mass of gas was absorbed, the atelectatic process advanced more rapidly and after about one hour was complete, taking about six to eight hours in all. If the branch of the pulmonary artery corresponding to the obstructed lung was ligated, atelectasis did not occur. If the respiratory membrane was rendered oedematous and therefore non-permeable to gases by the injection of one hundred per cent ether, again atelectasis did not develop. If oxygen or carbon dioxide at atmospheric pressure were used to re-inflate the atelectatic lung, absorption took place with great rapidity until the partial pressures of these gases approximated to those of the venous blood. Pure nitrogen required as much as sixteen hours for complete absorption. An interesting and unexpected observation was that the changing intrapulmonary pressures in the obstructed lung were constant with the intrapleural pressures. These pressures, of course, were measured in dogs with closed pleural cavities and the findings corresponded with those of Loewy and von Shroetter (1905) who had made similar observations in four human cases.

The experiments with which Van Allen's name is associated are important. They are responsible for establishing the theory of a collateral air circulation between adjacent lobules of the lobe of a lung.

Van Allen and Adams (1930) using silver nitrate destroyed a length of two centimetres of the medial division of the right lower lobe bronchus of a dog. No atelectasis resulted. Forty-five days later the dog was killed. Complete occlusion of the medial division of the right lower lobe bronchus and the absence of atelectasis were confirmed at autopsy. Repeating such lobular as opposed to lobar bronchial obstructions, identical findings resulted. From the data obtained from their experiments Van Allen and Adams (1930) and later Van Allen and Lindskog (1930) developed what has come to be called Van Allen's theory of collateral air circulation between the lobules of the lung. The substance of Van Allen's theory is as follows:-

(1) The partitions that divide one alveolus from another and one lobule from the next in a single lobe of a lung permit air, fluids and finely particulate matter to pass.

(2) This exchange is a result of very slight differences in pressure within two adjoining lobules. (The difference may be as low as one centimetre of water pressure.)

(3) These differences are such as are commonly generated between obstructed and free parts of the bronchial tree during quiet breathing.

(4) An obstructed portion of one lobe "breathes" by means of this communication through adjacent free parts, and the amount of this respiratory interchange may be as much as 3,600 cubic centimetres per hour.

(5) The interchange is found to occur in man by tests made in vitro.

(6) The interchange fails to occur between lobes.

Van Allen et al. showed that one portion of a lobe could become isolated by a multiplicity of mucous plugs obstructing the bronchioles. The result was that the imprisoned alveolar air was absorbed in a manner similar to that of lobar obstruction. They thought that this was the probable explanation for lobular or patchy atelectasis.

Before leaving this period of feverish American activity with its perhaps characteristic mixture of theory, controversy, and animal experimentation, there is one further observation worthy of note. Corrylos and Birnbaum (1933) in a paper entitled "The Syndrome of Pneumococcal Bronchial Obstruction" adduced evidence, both clinical and experimental, to show that lobar pneumonia was really "pneumococcic lobar atelectasis". They reported cases of lobar pneumonia treated successfully by bronchoscopic aspiration. In post-operative lobar atelectasis they

contended, pneumococci, and especially Group IV pneumococci,
were constantly found in the sputum.   Bronchial obstruction
was particularly liable to follow an infection with the
Group IV pneumococcus because of the copious, viscid sputum
that was characteristically produced.   Both lobar pneumonia
and post-operative lobar atelectasis were accidents in the
course of a pneumococcic bronchitis and were due to the
obstruction of bronchi by secretions or exudate.   If the
staphylococcus, the streptococcus or the bacillus of
influenza should happen to be the infecting organism then
lobular or patchy atelectasis or bronchopneumonia were the
sequelae.   Lastly if the occluding agent should be a
spetic foreign body or a piece of tonsillar tissue,
atelectasis could be followed by anaerobic infection with
necrosis, gangrene or anaerobic abscess of the lung.

The British medical literature, although not neglecting
the subject of pulmonary atelectasis, was not productive of
a parallel volume of publications during the years 1925 to
1930.   Featherstone (1925) recorded the results of an
enquiry into the cause of post-operative pneumonia.   In
the light of present day knowledge and judging from the
signs and symptoms displayed by many of his patients, the
majority of the cases described as pneumonia appear to
have been post-operative atelectasis.   He suggested that
their more adequate costal breathing permitting of coughing
without serious pain was the reason for fewer pulmonary

complications following abdominal surgery in women.   It
is but one indication of the arduous trials that beset
the surgeon and anaesthetist of not so long ago and a
measure of the role played by present day methods of
treatment with chemotherapeutic agents and antibiotic
drugs, that, in Featherstone's series of 106 "clean"
stomach operations, post-operative pneumonia occurred in
21 patients and of these 13 died as a result.

Soltau (1925) described the brick red colour that
sometimes characterises the facies in the presence of
post-operative atelectasis.   He thought that the massive
or multilobar variety was caused by inhibition of the
diaphragmatic action, either from reflex or inflammatory
causes.   Kletz (1927) published a case which he suggested
was the reflex type of massive atelectasis.   In the
following year Livingstone (1928), a pupil of Briscoe,
described how the supine position was associated with a
marked reduction in the vital capacity of the chest.

Boland and Sheret (1928a), in a little quoted but
excellent article on the subject stressed the rarity of
pneumonia as a post-operative complication and the
frequency of atelectasis.   The latter might or might not
be accompanied by displacement of the heart and, in the
absence of this sign, a lateral radiograph was of the
greatest diagnostic assistance.   Males were more frequently
affected than females possibly because their respiratory

passages were in a constant state of mild irritation from smoking.   Prophylactic treatment was of the utmost importance and should consist of postural drainage of the lower lobe bronchi effected by nursing the patient in a Trendelenburg position for 24 hours following operation. They favoured the theory of bronchial obstruction by retained secretions.

This publication prompted Wilkinson (1928), through the medium of the correspondence columns of the Lancet, to take issue with Boland and Sheret on the question of the pathogenesis of pulmonary atelectasis.   Wilkinson wrote that as a result of injury applied to the body a reflex constriction of the bronchioles of the lungs was set up.   In some cases this reflex affected only one lung, not necessarily on the side of the injury, and massive atelectasis resulted from an active process of pulmonary deflation.   Referring to the lowered isolateral intrapleural pressures and the value of artificial pneumo- thorax treatment he made the following interesting observation.   When a pleural effusion and atelectasis co-existed, aspiration of the effusion resulted in the appearance of cyanosis, dyspnoea and marked thoracic pain. Boland and Sheret (1928b) in reply referred to Dr. Curl's observation that even non-penetrating wounds of the thorax were followed by bronchial hypersecretion which was often almost confined to the opposite lung.   In Dr.

Curl's opinion this was due to a contrecoup mechanism such as occurred in cranial injuries. Whiteford (1928) advocated premedication with atropine sulphate to be repeated if need be until the patient became so dry that he could scarcely smack his lips. The author had encountered practically no pulmonary complications after many hundreds of abdominal operations.

Band and Hall (1932) experimenting on dogs found that intrabronchial content of a definite viscosity (they used gum acacia of varying degrees of viscosity) was necessary to produce massive atelectasis. Lobular atelectasis resulted from a lowering of the viscosity of the obstructing medium. They made a plea for a better standard of anaesthesia and recounted the dangers of cyanosis and asphyxia which caused accumulation of secretions in the respiratory tract. Abdominal distention was a factor in diminishing the diaphragmatic excursion and should be treated early in the post-operative period. A late result of unrelieved atelectasis was bronchiectasis.

The inadequacy of the physiological methods of draining the lung when secretions were present in considerable quantity led Nelson (1934) to investigate the effects of continuous postural drainage. He showed how gravity could assist in emptying the lungs of retained secretion if a posture was maintained where the bronchus leading from the affected area was dependent. It was

also necessary that the bifurcation of the trachea and not the mouth should be the centre to which drainage was directed. Cough with consequent expulsion of sputum resulted. Basing his method on investigations which he carried out on the bronchi and their distribution in the lung, Nelson evolved different postures for adoption according to the situation of the pulmonary lesion. He insisted that for efficient drainage the appropriate posture must be maintained continuously for several hours. His adjustable "Nelson bed" ensured that the patient was provided with the maximum possible amount of comfort during this procedure. The application of this study to the treatment of post-operative atelectasis demands that the lateral Trendelenburg position be adopted when the main bronchus is the site of obstruction. If the middle lobe and anterior basic area of the lower lobe are affected the patient lies flat on the back with the foot of the bed raised about twelve inches. The appropriate posture for draining the area supplied by the axillary basic branch of the lower lobe bronchus is attained by lying on the opposite side in the lateral position with the foot of the bed raised about twelve inches. In the case of the posterior basic area the patient lies prone with the foot of the bed raised as above. If different situations require drainage then the postures are altered accordingly. Nelson's postures, although frequently employed in the treatment of post-

operative atelectasis, are unfortunately rarely used with
the accuracy and thoroughness they deserve; as a prophylactic
measure they are much too frequently ignored.

The mode of treatment known as percussion postural
drainage is a refinement of Nelson's method.   Although
Nelson is frequently credited with the introduction of the
manoeuvre, this does not appear to be the case and the
writer has not been able to trace the originator.   The
technique consists of deliberate slapping with the palm
of the hand the area of the chest wall beneath which the
atelectatic lesion lies, the patient assuming the position
appropriate for postural drainage.   It is claimed that
more effective expulsion of sputum results.

About this time the practice of inducing basal
narcosis before the administration of inhalation anaesthesia
became popular.   The press issued glowing accounts of this
new method and patients tended to demand its use before
operation. Dawkins (1936) reported that owing to the
injudicious use of prolonged basal narcosis the incidence
of post-operative atelectasis showed a definite increase.
The rapidly eliminated sodium evipan and sodium pentothal,
however, were not implicated.

King (1933) published the first comprehensive series
of figures relating to post-operative pulmonary complications
to appear in the literature.   The statistics he presented
were based upon two years personal observation of the post-

operative pulmonary complications which had occurred in
the Massachusetts General Hospital during the years 1930
and 1931.   The study was confined to the type of
complication which manifested itself during the first
three or four days after operation and which was almost
always accompanied by fever, leucocytosis, cough and
purulent sputum - in King's words, "the type that is
diagnosed in the literature as bronchopneumonia,
pneumonitis, or collapse (atelectasis)."   His figures
referred to 7,065 operative procedures; they were
tabulated separately for the two years and showed
strikingly similar percentages in most instances.   The
conclusions that King drew from his study have been
corroborated time and again by subsequent observers; their
importance is such as to merit reproduction here.   A
table (Table I) referring to the incidence of post-
operative pulmonary complications in relation to type of
operation and sex of the patient is also reproduced below.
This table has formed a basis for comparison for many
subsequent studies of a similar nature.

(1) The most important factors in the occurrence of post-
operative pulmonary complications were type of operation
and sex of patient.   The incidence among men was at least
twice that among women; it was twelve times as frequent
following laparotomy and herniorrhaphy as after operations
in the non-abdominal group; it was most frequent in

TABLE I.

Pulmonary complications for the year 1930 in relation to type of operation and sex of the patient.

Reproduced from King (1933).

OPERATION ON	No. OF OPERAT-IONS.	COMPLI-CATIONS.	COMPLI-CATIONS %	MALES.			FEMALES.		
				OPERAT-IONS.	COMPLI-CATIONS	COMPLI-CATIONS %	OPERAT-IONS.	COMPLI-CATIONS	COMPLI-CATIONS %
STOMACH AND DUODENUM.	107	41	38.3	85	37	43.5	22	4	18.1
INTESTINES.	124	30	24.2	75	23	30.7	49	7	14.2
GALL BLADDER.	158	38	24.1	43	17	39.5	115	21	18.2
APPENDIX.	472	54	11.4	244	36	14.7	228	18	7.8
MISCELLANEOUS LAPAROTOMY.	120	13	10.8	69	9	13	51	4	7.8
GYNAECOLOGICAL LAPAROTOMY.	265	13	4.9	–	–	–	265	13	4.9
HERNIA.	260	26	10	223	23	10.3	37	3	8.1
ABDOMINALS AND HERNIA.	1506	215	14.3	739	145	19.5	767	70	9.1
ALL OTHERS.	1963	24	1.2	1030	16	1.5	933	8	0.8
TOTAL.	3469	239	6.8	1769	161	9.1	1700	78	4.5

operations on the stomach and duodenum, gall bladder and intestines and especially so when intra-abdominal sepsis was present.   In the group of non-abdominal operations 50 per cent of the pulmonary complications followed operations on the thyroid gland.

(2) Pre-operative respiratory infection of an acute or chronic nature carried a definite predisposition to post-operative pulmonary complications.

(3) A poor, preoperative, general condition of the patient was a predisposing factor.

(4) Fewer complications (12.4%) followed general anaesthesia than followed spinal (16.7%) or local (18.4%) anaesthesia. Poor risk cases, however, were apt to be included in the spinal and local anaesthesia group.

(5) Seasonal incidence was not a factor of definite significance but a slightly larger number of complications occurred during the winter months.

Brock (1936), who has advanced the knowledge of so many aspects of pulmonary disease, applied his logical reasoning to the subject of post-operative atelectasis. His own experience tallied closely with that of King. He thought that the greatest factor in obtaining free expectoration was active effort on the part of the patient accompanied by change of posture.   The most efficient posture for productive coughing was the lateral one, with the body turned slightly over toward the prone position.

He advocated that the patient be encouraged to cough while an attendant supported the region of the operation wound, small doses of morphine sulphate being given to ease the pain.   An expectorant mixture containing potassium iodide was useful in helping to render sputum less viscid. Commenting on the controversy that surrounded the pathogenesis of this condition, Brock stated the obvious but apparently hitherto unappreciated fact that the incidence of post-operative atelectasis depends on a combination of several factors but rarely on one factor alone.

Lander and Davidson (1938a) prepared artificial tenacious sputum in the shape of a viscid solution of gum acacia.   They introduced this mobile obstruction into the main bronchi of cats.   As Lander (1936) had suggested two years previously, this obstructing plug, as a result of the ensuing atelectasis, was sucked down the bronchial tree, split at each bronchial division and obstructed every branch until finally the obstruction came to rest in the finer bronchi.   Iodized oil was then introduced and radiography showed dilated patent bronchi in the atelectatic lobes.   Here at last was the most likely explanation to account for the changing character of breath sounds over atelectatic areas which had puzzled clinicians for so long. Absence of breath sounds was encountered only when the obstructing medium was immobilized in the main bronchus of

a lung or lobe.   If the increased isolateral negative
pressure consequent upon air absorption was sufficiently
powerful and the obstructing secretions sufficiently
mobile, the latter could be sucked peripherally as the
experiments of Lander and Davidson (1938a) had
demonstrated.   With patent bronchi and homogeneous
airless lung acting as a conductor, increased vocal
fremitus, bronchophony, whispered pectoriloquy and
bronchial breathing would be elicited.   It is not
surprising, therefore, that atelectasis has been, and
still is, so often mistaken for pneumonic consolidation.

In spite of the above contributions by Brock (1936)
and Lander and Davidson (1938a), enthusiasm for
describing new theories to account for post-operative
atelectasis had by no means waned.   Lindskog (1940)
referred to the work of Best, Dale, Dudley and Thorpe
(1927) who had previously demonstrated that lung tissue
had many times more histamine content than any other
tissue.   The action of inhaled anaesthetic vapours,
Lindskog contended, would liberate perfusates of histamine
and cause bronchial constriction and bronchial hypersecretion.
These effects in a lung where the respiratory mechanism was
already depressed by sedation and various mechanical factors
might well lead to lobular and lobar atelectasis.   In the
following year de Takats, Fenn and Jenkinson (1942) adduced

evidence, both experimental and clinical, to show that
vagal stimulation frequently resulted in bronchial spasm
and hypersecretion of the bronchial mucous membrane. It
is considered that the findings of the above workers have
an important bearing upon the causation of post-operative
atelectasis and are discussed in Chapter III. Hilding
(1944), drawing his conclusions from experimental work on
a hen's trachea, postulated that ciliary action was
indirectly responsible for the removal of air from the
atelectatic portions of the lung. The bronchial hyper-
secretion resulting from inhalation anaesthesia and
operation formed occluding masses of tenacious mucus in
the air passages of a lung lobe. The movements of a
succession of these masses upwards in the respiratory
tract by ciliary action caused removal of interposed
columns of air somewhat on the principle of a mercury
vacuum pump. Progressive shrinkage of the affected lung
lobe occurred as its air was removed. Ballenger (1950),
experimenting with rabbits, was unable to confirm the work
of Hilding. Xalabarder (1949) recently preferred to
think that an active contraction of the muscular elements
of the lung played a vital role in the production of
atelectasis. This contraction might be related to the
chemical transmission of the nervous impulse as he had
been able to produce areas of atelectasis in the human
lung by the topical application of a solution of 1 in

1000 acetyl choline during thoracoscopy.

During recent years several additions to the armamentarium of treatment have been described. Smart (1940), taking advantage of the enhanced mobility and lowered density of oxygen when mixed with helium, used such a gaseous mixture to lessen respiratory efforts and to relieve dyspnoea and anoxia during the acute phase of post-operative atelectasis. Waters (1942) claimed good results for the method which he called tracheo-bronchial toilet. He passed, for suction purposes, a small catheter down the lumen of a Magill tube previously introduced into the trachea under local anaesthesia. This manoeuvre was less complicated and had a wider application than bronchoscopic aspiration. Accumulated secretions could be aspirated from either main bronchus and post-operative atelectasis had been successfully treated. Grandstaff (1945), by the simple expedient of applying a 5 to 10 per cent solution of cocaine hydro-chloride to the region of the pyriform fossa, achieved speedy cure in a proportion of his cases. Good results were also claimed by Kruger, Marcus and Hoerner (1947) for their method of rapid introduction of ether vapour into the inspired gases during hyperventilation with carbon dioxide and oxygen. The sudden spasm of coughing produced was frequently productive of profuse expectoration.

In the sphere of prophylaxis, a decreased incidence
of atelectasis after abdominal surgery has been claimed
for the following:- (a) immediate post-operative nursing
in the Both Respirator (Mushin and Faux.1944); (b) early
post-operative ambulation (Blodgett and Beattie.1946);
(c) intercostal nerve block with nupercaine in oil
(McCleery, Zollinger, and Lenahan.1948); (d) intravenous
procaine hydrochloride (Brittain.1949).

There remains to be described post-operative
atelectasis in relation to thoracic surgery. It has
already been seen that the so-called massive collapse
was a frequent complication of gunshot wounds, not
necessarily penetrating, of the chest wall. Holst, Semb,
and Frihmann-Dahl (1935) found that atelectasis occurred
in 50 per cent of their thoracoplasties, the incidence
increasing with the extent of the operation. Atelectasis
of the remaining lobe or lobes of the isolateral lung
following the operation of lobectomy was early recognised
as a most unfavourable occurrence by the pioneers of
surgery within the thorax. Particularly was this the case
following partial resection of the lung for bronchiectasis.
Published figures have given the incidence as high as 40
per cent (Gray.1946). Indeed it is not surprising that
such should be the case if one reflects that in these cases
most of the factors considered to predispose to post-
operative atelectasis are likely to be present, namely:-

trauma, mucopurulent sputum, extreme pain on coughing,
reduced vital capacity, oedema of the hilar stump and
deflated lung tissue.   Gray classified these cases
into (a) temporary atelectasis, in which the lobe re-
expanded in a few days and (b) permanent atelectasis,
in which a progressive pneumonitis occurred in the
atelectatic lobe.   Permanent atelectasis left the
patient very much worse off than before operation and,
out of 14 such cases, Gower (1941) reported that nine
had died as a result.   Belsey (1937) analysed the
lobectomies done at the Brompton Hospital by Roberts
and Brock. He found a significantly increased incidence
of lobar atelectasis when the interval between the latest
bronchogram and operation was less than eight weeks.   The
importance of ridding the lungs of iodized oil by postural
drainage before lobectomy is now generally recognized.
Rink, Helliwell and Hutton (1948) reported that following
the operation for the relief of congenital pulmonary
stenosis chest complications were common, atelectasis and
sometimes pleural effusion having occurred in 20 cases out
of 36 who had survived the operation.

Comment:

     There is general agreement as to the clinical picture
and radiological findings of post-operative massive
atelectasis.   Lobar atelectasis is also readily recognized.

Although there may be considerable variability in the order and severity of the signs and symptoms presented by individual cases the displacement of the heart and mediastinum towards the side of involvement makes the diagnosis unequivocal. There is, however, a decided divergence of opinion as to the pulmonary status of lobular or patchy atelectasis and unfortunately the task of establishing diagnostic criteria to distinguish this condition from bronchopneumonia is by no means an easy one. In spite of the many claims that both conditions are identical the marked improvement that follows re-aeration strongly suggests that lobular atelectasis is a definite clinical entity.

Results will accrue from any form of treatment, whether prophylactic or active, which is directed towards the elimination of secretions from the bronchial tree and towards the improvement of pulmonary ventilation. Until the precise nature of the pathogenesis of post-operative atelectasis is ascertained however, it seems a safe assertion that this condition will continue to be the cause of considerable post-operative morbidity and even, on occasions, of mortality.

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Chapter II.

Physiological considerations in relation to post-operative atelectasis.

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I. Expulsion of secretion from the tracheo-bronchial tree.

A leading article in the Lancet of 1939 noted that expectoration was so commonplace that its physiological mechanism had been neglected. Standard works on physiology are apt to give but scant attention to the subject, yet the removal of secretions from the bronchial tree constitutes one of the most important bodily functions without whose protection life becomes endangered.

It is generally agreed that secretions and fluids in the respiratory airway are brought up the tracheo-bronchial tree by a combination of three motor mechanisms, namely ciliary action, cough and bronchial movements. Each of the above plays an important part in the expulsion of secretion; the failure of one renders the whole mechanism defective, though not inoperative because their activities overlap (Gunn,1927).

1. The ciliary mechanism.

The trachea divides into two main bronchi which in turn divide and subdivide into smaller bronchi. The smallest tube in this conducting portion of the bronchial tree is the terminal bronchiole and from it there arise respiratory

bronchioles.   Each respiratory bronchiole leads into an
expansion (the vestibule) from which arise ducts (the atria)
leading into the air sacs and alveoli.   It is in this last
portion of the lung that gaseous exchange with the blood
takes place.

Ciliated columnar or cuboidal epithelial cells extend
upwards to the nose from near or about the junction of the
terminal and respiratory bronchioles (Boyd, Perry and
Stevens, 1943).   In ciliated cells, the cilium appears as
a permanent projection with the power of altering its shape
(Negus, 1949).   In ciliary activity a whip-like motion
occurs leading to an active backward fling of the cilium
followed by a passive recoil.   The direction of ciliary
movement in the air passages is towards the larynx and
under normal conditions the beating frequency of the cilia
has been found to vary between 160 and 250 beats per
minute (Frenckner and Richtner, 1940).

In the normal bronchial tree, the glands of the
bronchial mucosa secrete sufficient mucus to keep the air
passages moist (Roberts, 1938).   Thus a thin continuous
film or blanket or mucus is formed composed of an outer
stratum of mucus resting on the tips of the cilia and an
inner fluid layer of low viscosity, forming a suitable
medium for the vibrating cilia (Lucas and Douglas, 1934).
Many writers (Gunn,1927; Hill,1928; Hilding,1944; Negus,
1949 and others) have described how throughout the bronchial

tree this sheet of mucus is steadily moved by ciliary action towards the larynx from the periphery. In addition, another sheet of mucus is pushed towards the larynx from the upper air passages by a similar method. Naso-pharyngeal and bronchial secretions collecting in the interarytenoid area are normally swallowed and only elicit the assistance of cough when present in abnormal amount. Negus has compared the whole mechanism to a conveyor belt, the platform on which the conveyed packages rest corresponding to the sheet or blanket of mucus; the propulsive agent of underlying pinions is represented by the beating cilia; deleterious particles or excessive secretions present in the air passages correspond to the conveyed packages. The entire lining film of mucus may be removed by ciliary action one to three times each hour and is as often replaced by the bronchial mucous glands. (Hilding,1944).

In health, mucus is raised by the ciliary action alone (Negus,1933) and the reserve capacity of the cilia is very great (Boyd et al), being capable of carrying 100 times the normal load without difficult (Hilding). It has been calculated that ciliary movement can propel solid particles at the rate of 12 feet per hour (Coope,1948). Hilding maintains that a normal ciliary mechanism would move the quantities and types of secretion found in post-operative atelectasis readily and with dispatch if there were not

other factors involved.

(a) <u>Factors affecting ciliary action</u>. The factors which are now detailed as affecting ciliary action have been culled from the writings of the authors quoted above. Cilia work best in a medium which is neutral or only slightly acid or alkaline. Gravity has no appreciable effect on the rate of the ciliary streams. Ciliary function is impaired but not necessarily lost in the presence of congestion, oedema or leucocytic infiltration of the mucous membrane. Temperature per se has no direct influence but factors which alter the viscosity of the mucous covering such as a too dry or a too humid atmosphere or excessive medication with atropine sulphate may have a deleterious effect. Under these circumstances, however, there is a compensatory alteration in the consistency and the amount of secretion of the mucous glands. The cilia are apparently not under direct nervous influence, the rate of beat being an inherent attribute of the cell and will continue after death if the medium is suitable. Neurogenic impulses in the bronchi and trachea affect the rate of motion of the cilia. Ciliary activity carries on during sleep and most authorities agree that general anaesthetics, with the possible exception of chloroform and ethyl chloride, have but little effect on the cilia. Sedatives on the other hand, and particularly morphine sulphate, have a

depressant effect.   Oily substances, e.g. iodised oil, by
clogging the cilia interfere with their activity.   Certain
local anaesthetics, e.g. cocaine hydrochloride in
concentrated solution, inhibit but 2.5 per cent cocaine
hydrochloride is almost harmless.   Mechanical obstruction
and the presence of granulation tissue in the mucous lining
of the bronchi cause interruption of the ciliary streams.

2. Cough.

It has been seen that the ciliary mechanism alone
normally deals with removal of bronchial secretion.   When
secretion in excess of normal reaches certain areas
(especially the larynx, the carina and the major bifurcations
of the bronchi) cough is excited.   Cough is thus an
exceptional and reserve mechanism.   Direct stimulation of
the lung proper or of the finer ramifications of the bronchi
does not excite cough (Ballon,1939).

(a) The Mechanism of Cough.   While cough may be a
voluntary act, it is generally a reflex manifestation.
When reflex in character its production depends upon local
irritation in the air passages eliciting nerve impulses
which are transmitted by afferent fibres in the vagus nerve.
The efferent portion of the reflex arc is completed through
motor nerve fibres innervating the intrinsic and accessory
muscles of respiration (Brown and Archibald,1927).
Stimulation of these motor nerves results in contraction of

the muscles involved.   The sequence of events is as follows:-
Following upon a preceding inspiration, the glottis closes
and the abdominal muscles contract causing an increase in
intra-abdominal pressure.   This increased pressure is
transmitted through the semi-rigid diaphragm into the
thoracic cage.   The intercostal muscles, aided by the
abdominal and lumbar group, tend to elongate the bony
framework of the thorax whose average diameter is thereby
markedly reduced.   The accompanying contraction and
fixation of the diaphragm further narrows the chest cavity.
The sum total effect is a sudden reduction of the intra-
thoracic capacity with a corresponding concentric rise in
intrathoracic pressure. Brown and Archibald have
demonstrated that this rise in pressure is uniform throughout
the thorax.   Finally the glottis relaxes, the compressed air
is violently expelled through the mouth and the extraneous
material is moved a variable distance along the air passages.

(b) Factors affecting the efficacy of cough. It follows
from the above considerations that the effectiveness of the
expulsive effort of cough will vary directly with the
amplitude of the inspiration which precedes it.   In other
words the greater the volume of air distal to the
accumulation of secretions, the greater the force applied
towards their expulsion in the act of coughing.   This
volume of "available air" (a term applied to it by Van Allen
and Lindskog,1930) and consequently the effectiveness of

cough is less in the presence of low vitality of the patient,
pain from thoracic and abdominal wounds, paralysis or
inhibition of the hemi-diaphragm, rigidity of the abdominal
muscles and abdominal distention.   Lack of muscular power
when the thoracic cage has lost its stability (Nosworthy,
1944) and paradoxical respiration may be important factors
in rendering cough ineffective following the operation of
thoracoplasty.   The unproductive cough so typical of
established post-operative atelectasis is in large part due
to the lack of available air in the respiratory passages
beyond the point or points of obstruction.   Narcotics
depress the cough reflex.   Extraneous material in the finer
divisions of the bronchial tree excite less cough than is
noticed under similar conditions in proximal portions.
(Gius, 1940).   Material remaining in contact with the same
area of mucous membrane for any length of time renders that
area insensitive and the urge to cough disappears (Sellors,
1944).   Wall (1928) considers that cough is most productive
when the sitting-up posture is assumed; Brock (1936) on the
other hand favours the lateral position with the patient
turned a little over on the face.

3. Bronchial movements.

   (a) Structure of the bronchial tubes.   In order to
appreciate the nature and effects of the bronchial movements
it is necessary to consider the structure of the bronchial

tubes.   Essentially the tubes consist of a myo-elastic
network with the larger branches stiffened by fibro-
cartilage.   In the trachea and main bronchi the fibro-
cartilage is in the form of incomplete hoops or rings,
open posteriorly where smooth-muscle fasciculi bridge the
gaps.   In the region of the smaller stem bronchi the
cartilages lose their regular horse shoe shape, becoming
irregular crescentic plaques, and the bronchial muscle
extends to embrace the entire tube and lies internal to
the plane of cartilage.   This arrangement is continued
into the terminal bronchioles and the circular disposition
of smooth muscle has been compared to the circular muscle
of the intestine (Macklin, 1929).   The mass of bronchial
muscle becomes relatively greater as we pass from the larger
to the smaller branches of the bronchial tree indicating that
in the latter areas its function is proportionately more
important.   The terminal bronchiole, which contains no
cartilage, is capable of obliterating completely its lumen
by muscular contraction (Macklin).   There has been much
discussion as to how far muscle extends towards the
periphery of the lung.   Macklin, who carried out a
monumental and exhaustive review of the subject, found the
prevalent opinion to be that muscle fibres occurred
everywhere throughout the lung with the exception of the
wall of the alveolus proper.   The bronchial muscle is

undoubtedly under nervous control (Macklin) and reacts to impulses carried in the vagus (broncho-constrictor) and sympathetic (broncho-dilator) nerves. The broncho-dilator nerves are found to run side by side with the constrictor fibres (Dixon and Brodie, 1903).

A network of elastic tissue closely follows the arrangement of the muscle fibres. Although apt to occur as a separate layer in the larger tubes, this network of elastic tissue coalesces with the muscle fibres of the smaller bronchioles. Ellis (1938) considers that these two systems of muscle and elastic tissue can throughout be regarded as a morphological and functional unit, - the myo-elastic sheet. Another set of elastic fibres occurs in the adventitial or outer coat of fibrous tissue which surrounds the bronchial tubes and strands pass to it from the myo-elastic layer. (The adventitial coat eventually forms the stroma of the alveoli). Both the adventitial and myo-elastic layers contain many blood vessels and nerves. Numerous elastic fibres are also found running longitudinally in the mucous membrane which lines the bronchial tubes. The elastic fabric is outwith nervous control and acts passively (Macklin) being responsible in large part for the recoil of the whole lung during expiration (Best and Taylor, 1945).

A study of the structure of the bronchial tree with its incomplete cartilaginous rings, its myo-elastic tissue and

its loosely attached mucosa reveals that it is admirably
adapted to alterations in length and in calibre.   Bronchial
movements may be classified according to the role played by
the bronchial musculature.

(b) <u>Movements in which the bronchial musculature is not</u>
<u>actively implicated.</u>   These consist of oscillations and
indentations of the bronchial tubes which arise from
transmitted pulsations of the heart, aorta and pulmonary
arteries.   Movements transmitted from the oesophagus as a
result of swallowing are of the same type.   Kieth (1909)
has described how the excursion of the diaphragm causes a
rhythmic displacement of the main bronchi and their branches,
downwards and forwards during inspiration and upwards and
backwards during expiration.

(c) <u>Movements in which the bronchial musculature is</u>
<u>actively implicated.</u>   The bronchial tubes elongate and
widen during inspiration and contract and narrow during
expiration.   Ellis (1938) believes that these rhythmic
changes in bronchial calibre are produced passively by the
respiratory movements of the thorax and that the chief
function of the bronchial muscle is the maintenance of tone,
thereby preventing obliteration of the lumen of the smaller
bronchi.   It is generally agreed, however, that changes in
bronchial calibre are the result of movements in which
muscular activity, co-ordinated under nervous influence, is
an important factor (Reinberg,1925; Macklin,1929; Morrison,

1930; and Ballon, 1939). The above authorities assume that the bronchial muscle during the inspiratory phase of dilatation and elongation is under the influence of the broncho-dilator nerves whose action at this juncture is to relax the muscle and so permit of its extension without resistance. Subsequently during expiration, the broncho-constrictor nerves stimulate contraction of the bronchial muscle.

In addition to the above, there exists in the bronchial tree a wave like motion resembling peristalsis. Indeed the term bronchial peristalsis is applied to this phenomenon which has been described by many authors (Reinberg,1925; Gunn,1927; Macklin,1929; Van Allen and Lindskog,1930; Ballon, 1939 and others). The presence of abundant sensory nerve endings in the bronchial wall and the existence of Remak's ganglion shows that an apparatus for peristaltic action exists. The peristaltoid movements are deemed to be dependent entirely upon the bronchial muscles and they seem to be called strongly into play only under abnormal conditions; namely, for the ejection of masses of thick exudate or other foreign material. Reinberg, who has made a minute study of the phenomenon, has observed violent expulsion of radio-opaque material from the tracheo-bronchial tree quite independent of cough. Indeed when cough super-vened it has had the effect of dissipating the material to

remote parts of the lung.   This effect of cough has also been noticed by Brown and Archibald and is an argument against the use of local anaesthesia for the operation of thoracoplasty in pulmonary tuberculosis.

Without the protection of bronchial peristalsis it is obvious that the smaller branches of the bronchioles which lead to the lung alveoli would be in a parlous condition - they possess no cilia and are unable to excite cough. Gunn considers that the peristaltoid movement of the smaller bronchi is especially well developed, a reasonable deduction when the relatively large proportion of muscle possessed by these tubes is recalled.   Opium and its derivatives paralyse peristaltic activity in the bronchi in the same way as they do in the digestive system. (Reinberg).

To sum up, it is the concensus of opinion that in the expulsion of secretions and foreign substances from the lung, ciliary action and bronchial peristalsis are together responsible for shepherding these substances to a point whence they may be expelled by coughing.   The upper airway possesses all three of these motor mechanisms, the intermediate airway has two (ciliary action and bronchial peristalsis) while the lower airway has only one (bronchial peristalsis).

II. Absorption of the occluded air in atelectasis.

1. Absorption of the occluded air in massive and lobar atelectasis.

Complete obstruction, no matter the cause, of the main bronchus of a lung or lobe of a lung inevitably results in absorption of the air distal to the point of obstruction provided the pulmonary circulation is intact and the alveolar epithelium not greatly altered. That this is the case has been demonstrated repeatedly by Lichtheim, 1879, Coryllos and Birnbaum, 1930, Van Allen and Adams, 1930, and others. The absorption of the occluded air into the circulating blood is purely a physical process (Henderson, 1938) and takes place in accordance with the laws that govern the diffusion of gases. It is one of the fundamental laws of gases that each gas diffuses according to its own partial pressure regardless of what other gases may be present (Henderson). Furthermore, in a mixture of gases at a certain pressure, the total pressure is divided between the different gases in proportion to their relative volumes (Wright, 1945).

The following table (Table I) shows in round figures the comparative partial pressures and volumes per cent of the gases in alveolar air and venous blood. (The gases in arterial blood are in pressure equilibrium with those in the alveolar air).

Table I. Partial pressures in millimetres of mercury and percentage volumes of the gases in alveolar air and venous blood.

Gases.	Alveolar Air		Venous Blood	
	Partial Pressure in m.m. Hg.	Volume per cent.	Partial Pressure in m.m. Hg.	Volume per cent.
Oxygen	100	14	40	6
Carbon Dioxide	40	5.6	46	7
Nitrogen	573	80.4	573	87
Water Vapour	47		47	
Total:	760	100	706	100

The total gas pressure in the venous blood (706 millimetres of mercury) is substantially less than that in alveolar air (760 millimetres of mercury) because the former has suffered a large fall in oxygen pressure but has only received a small increase in carbon dioxide pressure. Nitrogen pressure in the venous blood is the same as that in alveolar air. As a result of these pressure differences, gaseous diffusion between the alveoli and the venous blood continues when bronchial obstruction in a previously distended lung occurs. The imprisoned alveolar air loses more oxygen than it gains carbon dioxide. There ensues, therefore, an overall reduction in the volume of the

alveolar air with an increase in the percentage volumes and consequently the partial pressures of its carbon dioxide and nitrogen. Furthermore, because of the elasticity of the alveolar membrane and the outside intrathoracic pressure, the alveoli retract and the total pressure of its gases remain practically constant at 760 millimetres of mercury (Best and Taylor). Further diffusion thus takes place and so the process is continued until complete absorption of the imprisoned alveolar air results. Air is absorbed from the pleural cavity or any other closed cavity in precisely the same way (Henderson). The absorption of 1,000 cubic centimetres of alveolar air is examined in detail in appendix I.

2. Absorption of the occluded air in lobular or patchy atelectasis.

It is more difficult to conceive of a straightforward explanation of the physiological mechanism underlying post-operative lobular or patchy atelectasis. The condition may be quite symptomless or may resemble clinically a mild bronchopneumonia and this has led some authorities including Mann (1949) to doubt its existence as a definite clinical entity. Strong presumptive evidence for the existence of lobular atelectasis has been postulated by Adams (1942), Marshall (1944), Dripps and Deeming (1946), Goodwin (1946), Beard (1948) and others and Fleischner, Hampton and

Castleman (1941) would appear to have settled the issue conclusively. The last named investigators, describing linear shadows in radiographic studies of the lung, were able to corroborate at autopsy that the shadows were caused by small areas of lobular atelectasis.

(a) <u>Definition of a lobule</u>. Each lung lobule consists of a terminal bronchiole and its air cells and of the ramifications of the pulmonary and bronchial vessels, lymph vessels and nerves. Although distinct structures, the lobules are closely connected by areolar tissue. The muscle in the terminal bronchiole is more highly developed than in any other part of the bronchial tree and, when fully contracted, can exert a sphincter-like action cutting off completely the supply of air to the air chambers.

Reference has already been made to the collateral air circulation that exists between adjacent lobules of a single lung lobe. When bronchiolar obstruction occurs in part of a lobe it has been found that the alveoli of the obstructed lobules may be well ventilated by the communications (pores of Kohn) that exist with the unobstructed parts. How then can lobular atelectasis occur when scattered aggregations of terminal bronchioles are obstructed with secretion? To answer this question it becomes necessary to postulate interference with the free circulation of air between ventilated and unventilated lobules. To explain post-operative lobular atelectasis such interference with the

collateral air circulation would require to be operative during the early post-operative period.

(b) Factors which interfere with collateral air circulation.   It has been found that collateral air circulation is restricted or prevented during shallow respiration (Van Allen and Lindskog, 1930), in the presence of inhibition of the homolateral diaphragm, (Alley and Lindskog, 1948), in the presence of inflammatory exudates or secretions in the air passages and in circumstances in which the alveolar walls are not adequately ventilated (Best and Taylor, 1945).   In the early post-operative period we may have pulmonary hypoventilation secondary to rigidity of the abdominal muscles, wound pain, restraining dressings, pneumoperitoneum, and abdominal distention with resultant elevation of the diaphragm. Rees-Jones (1941) and Hawkins (1948) describe that in most instances the hemi-diaphragm is elevated and inhibited following abdominal operation.

It is obvious, therefore, that the factors which interfere with collateral air circulation are frequently operative during the early post-operative period.   It follows that under these circumstances scattered areas of bronchiolar obstruction will lead to absorption of the imprisoned alveolar air in precisely the same manner as in lobar obstruction and lobular atelectasis will result.

III. Pulmonary stresses in atelectasis.

Normally the mediastinal structures form a flexible, easily-moveable partition separating the lung and pleural cavity on the one side from the lung and pleural cavity on the other. The central position of the mediastinum in the thorax is in large part determined because the hemithorax, the lung volume, the elastic tension of the lung and the negative intrapleural pressure are approximately equal on both sides.

In atelectasis, as a result of obliteration of air chambers, the affected lung suffers a diminution in volume which becomes very considerable when a large portion of parenchyma is involved. The remainder of the lung must accommodate itself to a hemithorax almost the same size as before. As the surface of the lung cannot retract from the chest wall owing to the molecular cohesion between the visceral and parietal pleura (Brewerton, 1945), and the rigidity of the bony thorax permits only of slight inward retraction and crowding of ribs, the lost space must be re-occupied by adjoining mobile structures. If, however, compensation is not effected through these agencies there must inevitably occur an over-expansion of the remaining lung tissue. Such over-expansion leads to a state of elastic hypertension within the affected lung and consequently a lowering of the isolateral intrapleural

pressure. (The normal negative pressure of the potential intrapleural space is caused by, and is a direct measure of, the elastic recoil of the lung (Wright, 1945).)

1. Direction of Stresses with Atelectasis.

The pulmonary stresses resulting from the elastic hypertension developed in atelectasis have been described by Andrus (1937). Figs.I and II show schematically the quantitative distribution of such stresses in atelectasis of the right lower lobe. (The cross-lined section represents the atelectatic lobe, MED. refers to mediastinum and BR. refers to bronchus).

Fig.I.
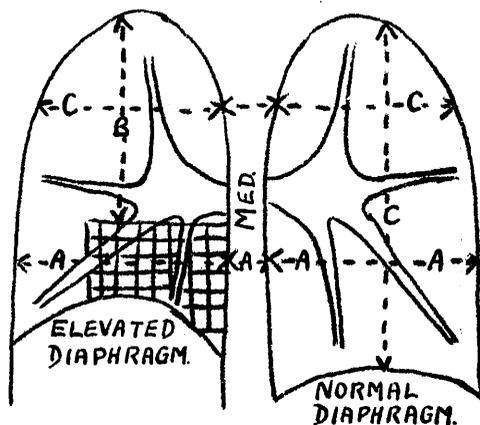
Fig.II.
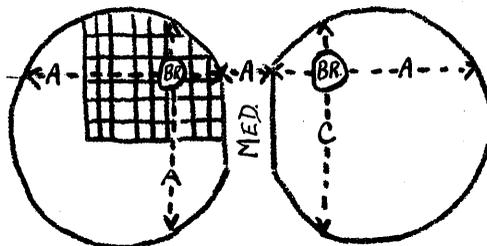
Direction of stresses with atelectasis (after Andrus 1937).

A. Maximum abnormal pull.
B. Intermediate abnormal pull.
C. No clearly abnormal pull.

The dimensions of the atelectatic right lower lobe suffer reduction in all three planes. It follows that compensatory replacement must be effected in three planes, i.e. in vertical, lateral and antero-posterior directions. Shortening in the vertical dimension of the lung, represented in the diagram at B, is in large part compensated by elevation of the homo-lateral diaphragm and by emphysema of the remaining aerated lung. At the position marked C, no direct pull results from the atelectatic shrinkage. Loss of lung volume in the lateral dimension is partly compensated by dislocation of the related mediastinal structures to the affected side and overstretching of the opposite lung, hence the lowered contralateral intrapleural pressure (see fig.I). In the antero-posterior plane, however, but little relief is afforded by crowding of ribs and narrowing of intercostal spaces. It is in this plane, and to a lesser degree in the lateral plane, that the maximum abnormal stress occurs. Vertically disposed bronchi are thus submitted to a considerable dilating force as shown in fig.II. Elevation of the dome of the hemi-diaphragm lessens the dilating stress upon bronchi the main axis of which is horizontal. That this explanation of the relationship between bronchiectasis and atelectasis is correct is strongly suggested by the fact that bronchiectasis is encountered most frequently in the more vertically placed basal bronchi.

The experiments of Tannenberg and Pinner (1942) show
that main-bronchial occlusion leads to absorption of air
from the bronchi distal to the point of obstruction.   In
the absence of a large volume of secretion within the lumen,
the distal bronchi collapse as far as their structure
permits.   When atelectasis exists as a result of
bronchiolar occlusion, bronchiectasis invariably presents
in the patent proximal bronchi of the atelectatic lobe
(Lander and Davidson, 1938a).   Thus it would appear that
the pulmonary stresses in atelectasis cause bronchial
dilatation when the obstruction is bronchiolar; when the
main bronchus is the seat of obstruction; the pulmonary
stresses engendered do not cause bronchiectasis.   It has
already been described, however, that in unrelieved
atelectasis obstructing secretions may be moved distally
by the imbalance of intrathoracic pressures, leaving the
larger bronchi patent.   When early resolution of atelectasis
occurs the dilated bronchi rapidly revert to normal giving
rise to the phenomenon which has been called "reversible
bronchiectasis".   Permanent bronchiectasis may follow delayed
resolution of atelectasis, the walls of the dilated bronchi
being overstretched to such an extent that their elasticity
is lost beyond recovery.

IV. **The blood flow through an atelectatic lung.**

The immediate mortality rate from post-operative
atelectasis is practically nil.   The few cases of acute
uncomplicated atelectasis that have come to autopsy have
been of the massive variety and in these the capillaries,
arterioles and venules of the affected lung were dilated
and filled with blood.   This appearance of engorgement
led Scott (1925) and later Bergamini and Shepard (1927)
to suggest that post-operative atelectasis might be caused
by a vasomotor disturbance of reflex origin.   Lucas (1950)
believes that the high negative pressure developed in
atelectasis encourages an increased pulmonary circulation
through the involved portion of the lung.   Blood flowing
through non-ventilated parts does not become oxygenated
and so dilutes the remainder of the pulmonary blood
returning to the heart.   The systemic circulation thus
becomes deficient in oxygen and cyanosis may develop.
Cyanosis tends to disappear later in spite of non-resolution
of the pulmonary lesion suggesting that stasis of the
circulation has occurred in the atelectatic area.

Bjork and Salen (1950), with the aid of angiography
have studied in dogs the effects of atelectasis on the
pulmonary circulation.   In the early stages the capillary
bed of the atelectatic lung remains open but in the course
of a few days the volume of blood passing through it

diminishes.   The general arterial oxygen deficit
diminishes at the same rate as the blood flow through the
atelectatic lung decreases.   In about one month's time
practically no blood passes through the non-ventilated
parts of the lung, the blood flow appearing to be shut
off at the capillaries.

Summary.

In this chapter some of the less well known aspects
of physiology in relation to post-operative atelectasis
are described.   A full account is given of ciliary action,
cough and bronchial peristalsis, the mechanisms employed in
expelling secretions from the tracheo-bronchial tree.   The
importance of these functions is stressed and factors which
may affect them adversely are detailed.   Emphasis is
especially laid upon the efficiency and reserve power of
the cilia, a fact that is seldom considered when the
aetiology of post-operative atelectasis is discussed.   There
is a brief description of the principles underlying the
absorption of alveolar air by the pulmonary circulation.
Reasons are given in support of the view that in the early
post-operative period the collateral air circulation may
be so reduced as to permit of absorption of air from
obstructed lobules.   There is an account of the pulmonary
stresses as described by Andrus that develop within the
thorax as a result of atelectasis.   The little information

available regarding the blood flow through the atelectatic
lung tends to provide an explanation for the symptomatic
improvement that occurs in spite of non-resolution of the
pulmonary lesion.

Conclusion.

Any theory of the pathogenesis of post-operative
atelectasis, especially when the condition occurs in a
previously healthy lung, must explain the failure of
ciliary action and bronchial peristalsis to prevent the
accumulation of secretions within the bronchial tree.

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Chapter III.

Bronchial constriction and post-operative atelectasis.

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1. Aetiology of post-operative atelectasis.

While many theories have been evolved to account for post-operative atelectasis it is the popular conception that the condition is caused by plugs of mucus or mucopus which accumulate because of weakened respiratory force and suppressed cough and that air distal to the point of obstruction is absorbed by the blood stream. In the opinion of the writer the above explanation leaves much to be desired. It has already been noted that no account of this type of atelectasis can be complete if it does not explain the failure of the mechanism normally made use of by the lung to rid itself of accumulated secretions. It is now proposed to advance the theory that bronchial constriction or spasm is a prime factor in the causation of post-operative atelectasis and to trace the reasoning process by which this conclusion was reached. The argument presented is the outcome of several years of thought based on clinical impressions and must of necessity be discussed in general terms.

2. Observations on post-operative atelectasis in service cases.

The fact that the incidence of post-operative pulmonary

complications is significantly high in service cases has
been recorded by Harris (1942), Campbell and Gordon (1942),
Bird, Kilner and Martin (1943), Lucas (1944) and Holmes (1948).
The writer had first hand experience of this high incidence
while serving as anaesthetist to a large military hospital.
For two years he administered anaesthetics in approximately
equal numbers for orthopaedic and general surgical cases.
It soon became apparent that the incidence of the so-called
"chest" was alarmingly high in the general surgical wards
but the condition was practically unknown in the orthopaedic
department.   It seemed odd that a bronchitic patient,
submitted to the extensive and prolonged surgery of spinal
fusion by tibial bone graft, the victim of protracted pre-
and post-operative immobilization in a plaster shell with
consequent lowering of muscle tone and stagnation of
bronchial secretions, should escape a pulmonary complication.
On the other hand it was common experience for a vigorous
soldier in perfect health and with no respiratory infection
to develop an acute lobar atelectasis within twenty-four or
forty-eight hours of a simple herniotomy lasting but fifteen
or twenty minutes.   Several examples of this apparent
paradox were encountered.

In spite of minimal sedation, frequent changes of
posture, encouragement to cough, deep breathing exercises,
early ambulation and the prophylactic use of sulphonamides
and penicillin, the incidence of atelectasis after simple

herniotomy remained disappointingly high. On the other hand, despite an ever-increasing number of major orthopaedic procedures in poor risk cases given an identical anaesthetic technique by the same anaesthetist, from the same anaesthetic machine and often in the same operating theatre, no single case of atelectasis developed.

It is well established that in the development of post-operative atelectasis the site of operation is the most important single factor (King, 1933; Brock, 1936; Sircar, and Boston, 1940; Henry, 1943; Gusterson, 1945; Beard, 1946; and many others) and that abdominal, thoracic and thyroid surgery provide the very large majority of the cases. The reason usually advanced is that sedation, wound pain and muscle spasm interfere with pulmonary ventilation, cough is voluntarily suppressed, bronchial secretions stagnate and atelectasis develops. Pre-existing respiratory infection is considered a most important predisposing factor - Rink (1938) goes so far as to state that it is probably present in every case.

The site of operation apart, several of the patients submitted for spinal fusion appeared to present most, if not all, of the factors likely to predispose to post-operative atelectasis yet no such complication developed. The hernia cases could not be considered more likely to develop atelectasis; no patient with acute respiratory infection was submitted for hernial repair. Indeed the

large majority were in excellent physical condition with
no signs or symptoms referable to the respiratory tract,
and in several instances spinal anaesthesia only was
administered.   As a rule the patients co-operated well
during the early post-operative period.   When reassured
that no harm would accrue from frequent change of posture,
periodic deep breathing and coughing provided firm
pressure was applied to the wound area, they were able to
indulge in these activities with surprisingly little
discomfort.   In spite of all the prophylactic measures
adopted secretions accumulated within the bronchi,
stagnated and atelectasis developed.

The puzzling features of the hernia cases may be
summed up in the following questions:-
(1) Why, in a previously healthy lung and especially when
spinal anaesthesia was administered, should secretions
accumulate with such rapidity and in sufficient quantity
to cause bronchial obstruction and atelectasis?
(2) Why should the motor mechanisms for ridding the
bronchial tree of mucus and other secretions suddenly
prove inadequate?

The lung is normally one of the best drained organs
in the body with a highly developed mechanism for expelling
secretions, possessing great reserve power and capable of
dealing with very large volumes of mucus and mucopus.
During the operation of thoracoplasty, performed under

general anaesthesia, mucopus, unassisted by cough or
gravity, frequently accumulates in the region of the
larynx, presumably carried there by ciliary action. It
has already been noted that even under deep anaesthesia
and profound narcosis investigators in experimental
studies experienced the greatest difficulty in preventing
expulsion of elaborate obstructing devices from within the
bronchi.

For a long time the irritating qualities of
inhalation anaesthesia, especially ethyl chloride and di-
ethyl ether anaesthesia, have been held responsible for
stimulating the production of mucus within the bronchial
tree. Premedication with atropine sulphate and
scopolamine hydrobromide is designed to obviate excessive
accumulation of mucus during general anaesthesia. Post-
operative atelectasis, however, occurs with at least equal
frequency following local and spinal anaesthesia as studies
by King (1937), Dripps and Deeming (1946) and Chivers (1946)
amply testify. Ciliary activity alone should be capable of
dealing with the quantity and types of secretion found in
post-operative atelectasis. Furthermore bronchial
peristalsis can be called into action when the cilia are
overburdened and it has been seen that the hernia patients
made free use of cough.

3. Development of the bronchial constriction theory.

It was argued therefore that some hitherto unconsidered factor or factors might influence the development of post-operative atelectasis. It seemed possible that in abdominal, thoracic and thyroid surgery there might be some factor operative that not only caused production of mucus secretion but also determined its retention within the bronchi. Such a factor presumably was not encountered in surgery outwith the regions mentioned. The distribution of the vagus nerve lay within the scope of the operations notoriously complicated by atelectasis and as Gunn pointed out as long ago as 1927: "It is a characteristic feature of vagal reflexes generally that the efferent side of a reflex can be discharged by stimulation of afferent branches of the vagus other than those normally concerned with the reflex, provided that this abnormal stimulation is of sufficient intensity and duration." With this in mind the argument was further developed that stimulation of the vagus can cause reflex bronchial constriction; bronchial constriction stimulates mucus secretion; bronchial constriction could conceivably interfere with ciliary action, bronchial peristalsis and the normal rhythmic length and calibre changes of the bronchi during respiration. The result would be retention of secretion, bronchial obstruction, absorption

of alveolar air and atelectasis.

Further examination of the hypothesis so far
developed seems to explain other features characteristic
of post-operative atelectasis.   It accounts for the
presence of crepitations and sibilant rhonchi heard for
the first time post-operatively in a lung later to become
the seat of atelectasis.   It can explain the unproductive
cough, the stagnant mucus stimulating a sensitive area of
bronchial mucous membrane and the constriction holding the
mucus firmly and thus preventing accumulation of available
air for its expulsion.   Depending upon the extent and
severity of constriction and the viscosity of secretion,
bronchial constriction can determine whether the obstruction
will remain in the main bronchus or be sucked down the
bronchial tree with the development of increased negative
pressure.   In the former case instant relief follows
removal of the obstruction by bronchoscopic aspiration or
other means; in the latter the results of bronchoscopic
aspiration are most disappointing and slow resolution of
the atelectasis is to be expected.   Bronchial constriction
may account, at least in part, for the high splinted hemi-
diaphragm and reduced vital capacity so frequently
encountered following abdominal operation.   Altschule
(1943) and Pooler (1949) considered that wound pain caused
the reduction in vital capacity.   Pooler treated his
patients with intravenous procaine hydrochloride, abolished

the pain but little improvement in vital capacity resulted. He concluded that other causes for the lowered vital capacity after abdominal operation needed investigation. McCleery et al. (1948), believing reflex muscle spasm to be the factor involved, interrupted the reflex arc on the afferent side by inducing prolonged local anaesthesia of the sixth to the eleventh intercostal nerves in the mid axillary line. Again the effect on vital capacity was disappointing. It seems that constricted bronchi might account for the reduction in vital capacity by reducing air entry. The hemi-diaphragm would naturally occupy the position of rest and x-ray examination reveal it as occupying a higher position than normal.

Lastly our hypothesis can provide an explanation for the rapid production of mucopurulent sputum in the presence of a previously healthy bronchial mucosa, a feature that has puzzled very many investigators in the field of post-operative atelectasis. Stagnant mucus held by constricted bronchi will form an excellent nidus for pathogenic organisms present in the upper respiratory tract especially during periods when respiratory infections are prevalent. Under these circumstances mucopus may conceivably be produced with unusual rapidity.

4. Bronchial constriction in relation to hypoventilation and infected bronchial mucosa.

Granted that bronchial constriction and mucous secretion constitute the all important first step, conditions favouring hypoventilation and accumulation of intrabronchial secretions, although insufficient in themselves, will assist in the development of post-operative atelectasis.   Patey (1939), investigating the effects of deficient ventilation of the lung bases following abdominal surgery concluded that any effect that respiratory sub-efficiency has in the development of atelectasis is of a subsidiary or predisposing nature only and that other factors of an exciting nature are necessary for the development of this complication.   Atelectasis is a rare occurrence in chronic bronchitis (Erwin, 1939) but it is by no means uncommon in acute bronchitis (Gairdner, 1850; Erwin, 1939).   Christie (1951) describes spasm of the bronchial musculature in acute bronchitis and adds "the parasympathetic nerve supply to the (bronchial) muscles, through the vagus causes bronchial constriction......" The last observation taken with the occurrence of atelectasis in acute bronchitis tends to support the contention that bronchial constriction acting through the vagus nerve is an important factor in post-operative atelectasis.   One is tempted to speculate, although it

is quite outwith the scope of this work, that in whooping
cough the spasmodic nature of the respiratory symptoms
is suggestive of spasm of the bronchial musculature
which may possibly be associated with the development of
atelectasis in this condition.

5. Bronchial constriction in relation to treatment of post-
operative atelectasis.

The standard methods of treatment of post-operative
atelectasis are largely aimed at: (a) improving pulmonary
ventilation by encouraging increased diaphragmatic excursion
with carbon dioxide inhalations and breathing exercises, by
instituting changes of posture, by avoiding over-sedation
and by relieving wound pain with intravenous procaine
hydrochloride; (b) Preventing conditions that favour secretion
formation within the bronchial tree and encouraging removal
of secretions that have accumulated.   Thus cigarette
smoking is discouraged before and after operation and
surgery is postponed in the presence of acute respiratory
infection.   Non-irritating general anaesthetics or local
and spinal anaesthesia are used in the presence of chest
disease.   The patient is encouraged to cough, postural
drainage is instituted and tracheo-bronchial suction
directly aspirates mucus or mucopus.

Numerous investigations have been conducted to show
that one or all of the predisposing factors already detailed

play significant roles in the development of post-
operative atelectasis.   As many surveys claim that the
incidence of atelectasis can be reduced by instituting
one or more of the methods of treatment already described.
In spite of the most vigorous prophylactic and active
treatments no investigator has been able to do more than
reduce the incidence of post-operative atelectasis. The
most ably conducted and controlled series of cases one has
found in the literature is described by Dripps and Deeming
(1946) who analysed the pulmonary complications which
followed 990 upper abdominal operations.   Their patients
were admitted to a hospital where two surgical teams
operated on alternate days.   One team preferred inhalation
anaesthesia, the other spinal anaesthesia.   The only
outstanding variable was the method of anaesthesia;
otherwise the patients were treated in the same operating
theatre, in the same wards and by the same nursing staff.
The authors followed each case during the entire post-
operative period and the patients were treated by the
following regime.   Each case was given carbon dioxide
inhalations for two to three minutes every half hour, he
was turned every hour, secretions were aspirated, sedation
was reduced to a minimum, cough encouraged and early
ambulation practised.   Chemotherapy and antibiotic agents
were administered prophylactically.   In spite of such
intense treatment the incidence of atelectasis and pneumonia

was 5 per cent after spinal anaesthesia and 4.1 per cent after inhalation anaesthesia. (The diagnosis of pneumonia, a very small proportion of the cases, was made where a febrile reaction was protracted or x-ray signs of consolidation developed).

In view of the foregoing it does not seem unreasonable to postulate that there remains something to be desired in our knowledge of the aetiology and treatment of post-operative atelectasis. The present investigator considers that the fundamental underlying cause of this type of atelectasis is bronchial constriction induced reflexly by surgical interference in the area of distribution of the vagus nerve. The bronchial constriction, although apt to be bilateral in distribution, is much more pronounced on the side of operation. Depending upon the severity and duration of the reflex constriction and the quantity and tenacity of the resultant mucus secretion, bronchial constriction and atelectasis can develop in spite of the absence of an infected mucous membrane, depressed cough, over sedation, hypoventilation and all the other factors that have been given a prominence quite outwith their importance. Accepted methods of treatment are able to reduce the incidence of atelectasis only in the cases where the effects and severity of the bronchial constriction are slight enough to permit an active regimen to restore the normal changes in length and calibre and peristaltic action

of the affected bronchi and thus to allow respiration to proceed in the normal manner.   It is true that dramatic resolution of post-operative atelectasis may follow bronchoscopic aspiration in acute cases.   As spectacular results however follow the mere topical application of a local anaesthetic agent in the region of the pyriform recess.   Grandstaff (1945) who describes this latter method of treatment attributes its success to relaxation of the musculature of the bronchial tree - an action that can only take effect through the vagus nerve.

6.   Further observations on bronchial constriction and atelectasis.

At first sight there appeared an obvious criticism of the theory so far developed.   Asthma is a malady characterised by bronchial spasm.   Atelectasis, although not unknown as cases described by Sante (1928b),Peshkin and Fineman (1931), Priest (1950) and Howie (1950) indicate, is a rare complication of asthma.   Further reflection and interrogation of asthmatic subjects met this criticism. In asthma the bronchial spasm is expiratory in type, inspiration is not impeded and respiratory difficulty occurs only on expiration.   Emphysema, due to this check-valve type of obstruction, and not atelectasis constitutes the obvious complication.

A degree of bronchial spasm is a not uncommon feature

of the induction period of general anaesthesia particularly
when ethyl chloride or di-ethyl ether are the agents used.
For many years one has noticed that spasm of this nature
might be either purely inspiratory or purely expiratory in
type or, on occasions, a combination of both.   When a
purely inspiratory spasm presents expiration is seen to be
unimpeded and vice versa.   When the spasm relaxes to return
later it usually returns in relation to the phase of
respiration originally affected.   Occasionally a total
spasm is encountered when respiration ceases abruptly at the
end of expiration, the lungs deflate with alarming rapidity,
profound cyanosis quickly develops and, for a time, the
lungs resist all efforts at inflation.   In this type of
case, in spite of adequate premedication with atropine
sulphate, much mucus secretion invariably accumulates within
the respiratory passages.   Spasm of the bronchial
musculature can be readily initiated in some patients while
in others no such tendency is exhibited.   In the former
group are found nervous individuals and fit vigorous young
men.

It would thus appear that the threshold for spasm of
the bronchial musculature varies considerably with the
individual and that there is an individual predisposition
to develop spasm which may be either inspiratory or
expiratory in type.   It is suggested that this may be due
to a degree of autonomic imbalance and may account for the

increased incidence of post-operative atelectasis which
is found, especially during time of war, among service
personnel.

7. Observations from the literature on bronchial
    constriction and atelectasis.

    Although the above conclusions in regard to the
aetiology of post-operative atelectasis are based on
personal experience and arrived at independently, the
conception that reflex vagal effects are operative in
atelectasis is not new. Bradford (1920) and Wilkinson
(1928) consider the possibility of a reflex spasm of the
bronchioles; Kletz (1927) also refers to the "reflex type"
of atelectasis. Scrimger (1921) entertained the theory
that abdominal operation acting through the vagus nerve
might cause a muscular contraction of the lung, accumulation
of mucus and post-operative atelectasis. Morrison (1930)
also supports this view. Nosworthy (1944) mentions that
spasm of the bronchial musculature, by closing down the
lumen on a small collection of mucus, may be the determining
factor in converting a partial into a complete obstruction.
However, the theory that bronchial constriction or spasm is
a factor in the causation of post-operative atelectasis
does not appear to have been seriously entertained until de
Takats, Fenn and Jenkinson (1942) produced experimental
evidence to show that in the dog various intra-abdominal

manipulations, e.g. traction on the cystic duct and pulling on the mesentery, give rise to reflex bronchial constriction and an associated stimulation of bronchial secretion. Similar results followed experimentally produced pulmonary embolism and trauma to the chest wall. These effects were not encountered in the presence of bilateral vagal section and presented in a proportion of the dogs only when an adequate dose of intravenous atropine sulphate or of an atropine-papaverine combination was administered beforehand. (They claimed that papaverine had a direct action on the smooth muscle of the bronchi and inhibited the cardiac effects of atropine overdosage.) Table I gives a summary of the experiments conducted by de Takats et al. It will be seen that the results of these experiments lend support to the views already expressed in regard to the aetiology of post-operative atelectasis.

Table I.   Reflex Bronchial Spasm - Summary of Experiments.
(de Takats et.al.1942).

Method of bronchial spasm production.	No.of dogs.	Method of bronchial spasm prevention.	Bronchial spasm %.
1. Pulmonary embolism.	12	None.	100
	37	Atropine or atropine-papaverine.	42.5
	4	Bilateral vagotomy.	0
2. Abdominal manipulation.	10	None.	100
	10	Atropine.	30
3. Trauma to chest.	10	None.	60
	10	Atropine.	30
Total animals used:	93		

Applying their experimental findings to the pathogenesis of post-operative atelectasis de Takats et al. make the following observation: "It is our feeling that the initiation of the bronchial obstruction (in post-operative atelectasis) may be due to early bronchomotor and bronchosecretory phenomena which may subsequently result in mechanical obstruction." In other words they arrive by a different route at the same terminus as the present writer.

The experimental findings of de Takats and his colleagues and the fact that atropine sulphate or atropine-papaverine prevents the occurrence of bronchial spasm in half the animals, suggest that drugs which paralyse the action of the parasympathetic system should be effective in the treatment of post-operative atelectasis. No published figures relating to such a method of treatment have been found. Lucas (1950), who has observed bronchial constriction in human subjects following direct traction of the stomach and stimulation of the splanchnic plexus, mentions in a recent publication that he is at present observing the effects of ephedrine hydrochloride in the prevention of post-operative atelectasis. Lack of suitable facilities and the pressure of other work have up to the present made it impossible for the writer to conduct an investigation of this nature. It may be observed, however, that de Takats et al. found it necessary to administer grain 1/6th of atropine sulphate to prevent bronchial spasm in their dogs. It is unlikely that

human subjects would tolerate such a large dose of the drug.

Spontaneous Atelectasis.

Brief reference will now be made to a comparatively rare and little publicised type of atelectasis which may occur during anaesthesia and which, on account of the dramatic suddenness with which it sometimes appears, may suitably be termed spontaneous atelectasis.   It seems probable that some of the cases described by Bradford as complicating trivial gunshot wounds of the chest and abdomen and referred to in Chapter I. may have been examples of spontaneous atelectasis.   It will be recalled that de Takats et al. observed bronchial spasm in 50 per cent of their dogs subjected to chest trauma.

Instances of atelectasis occurring during anaesthesia have been described by Bergamini and Shepard (1927), Schotz (1943), Cassels and Rapoport (1944), and Forregger, Rettig and Conroy (1949).   The cases encountered by Bergamini and Shepard and Schotz followed the following pattern: Suddenly during the course of anaesthesia for abdominal operation the patient became cyanosed, the heart ceased to beat, all efforts at resuscitation proved unavailing and death occurred on the operating table.   Autopsy findings revealed massive atelectasis of both lungs with no evidence of any obstruction within the air passages.   Forregger et al. report a case which differs from the above only in that

it was possible to inflate the lungs under considerable
positive pressure.   Death occurred after eight hours in
spite of continuous efforts at artificial respiration.
Post mortem examination revealed massive bilateral
atelectasis with no evidence of tracheo-bronchial
obstruction.   Examples of spontaneous unilateral
atelectasis with recovery are described by Cassels and
Rapoport.   The present author has experience of two
instances with recovery of this type of atelectasis
presenting suddenly in the opposite lung during the course
of pneumonectomy.   In each case the lung resisted
vigorous efforts at inflation and the patient appeared on
the point of death, when suddenly the bronchial tree
relaxed and complete recovery ensued.

1. Aetiology of spontaneous atelectasis.

Atelectasis occurring during anaesthesia is usually
regarded as arising through a rapid absorption of
anaesthetic gases from the lung without their being
sufficiently replenished by adequate respiratory exchange.
Lucas (1950) points out the fallacy of this conception.
If the alveoli are becoming depleted of gases by too rapid
absorption a negative pressure will develop which will draw
in more gases from the reservoir provided in all anaesthetic
machines.   The exceedingly rapid development of atelectasis
which characterises these cases suggests some underlying

neurogenic mechanism.   It is the view of Scrimger, Morrison, Schotz and Xalabarder that the lung is a muscular organ capable of undergoing active contraction. That there is a vagal origin for this neurogenic mechanism is strongly suggested by the following cases of unilateral spontaneous atelectasis.   Case 1. is described in detail.

2.   Two cases illustrative of spontaneous atelectasis.

Case 1.   R.A., a fit man, aged 30. B.P. 140/90.

11.4.'51. Operation proposed: Repair of a large hernia of
          the left hemi-diaphragm.

2.30 p.m. Anaesthetic commenced.

          (a) Induction: Intravenous thiopentone o.5 G.,
                         cyclopropane, di-vinyl ether,
                         gallamine triethiodide (flaxedil)
                         80 mg. Oral intubation and throat
                         pack.
          (b) Maintenance: Cyclopropane and di-vinyl ether
                         (closed circuit). Intravenous dual
                         drip transfusion of 1 pint 0.1 per
                         cent procaine hydrochloride and 1
                         pint whole blood.

2.50 p.m. Operation commenced.   Left postero-lateral thoracotomy was performed and access gained to the chest through the bed of the eighth rib.   The left lung deflated in the usual manner.   The left side of the thorax was found to contain the stomach, the spleen, most of the small intestine, the transverse colon, the splenic flexure and part of the descending colon.   Considerable manipulation was required to return the abdominal contents, during which

time the spleen was damaged and had to be removed.   A
tear in the left hemi-diaphragm, extending from the central
tendon to the tip of the ninth costal cartilage was repaired.
4.p.m.   Total atelectasis of the left lung was noticed.   The
lung was very small, about one sixth of its normal size and
lay close to the hilum.   It was of a deep purple colour and
of the consistence of the liver.   The right lung was
respiring adequately and the patient's condition was fair.
B.P. 100/70; pulse 120 per minute; respirations 30 per
minute.   Attempts to inflate the left lung were unavailing.
Considerable positive pressure was used but there was no
sign of air entry.   Repeated attempts at inflation were
attempted at intervals during the next twenty minutes but
without success.
4.20 p.m.   Three C.C's. of 1 per cent procaine hydrochloride
were injected into the left posterior pulmonary plexus.
Almost immediately the lower lobe of the atelectatic lung
could be inflated with very slight positive pressure; the
left upper lobe re-inflated easily about thirty seconds later.
4.30 p.m.   The chest was closed and the left lung was seen
to be respiring freely.
4.31 p.m.   Endotrachneal suction applied with negative
results.
12.4.'51.   Portable radiography revealed complete re-
expansion of the left lung.

Comment on Case I.   The dramatic manner in which inflation
of the left lung became possible after the injection of
three cubic centimetres of procaine hydrochloride in the
region of the left posterior pulmonary plexus strongly
suggests that the atelectasis was neurogenic in type.   The
bulk of the vagal fibres supplying the bronchial musculature
of the left lung was effectively blocked by the local
anaesthetic.   This resulted in relaxation of the related
bronchial muscles previously in a state of profound spasm
produced reflexly as a result of manipulation of abdominal
viscera.

Case 2.   B.T., Female, aged 15 years in good general
           condition suffering from bronchiectasis of the
           lower lobe of the left lung.

Anaesthesia: The technique of anaesthesia and the agents
           used were the same as for Case I.

Operation: Left lower lobe lobectomy was performed with the
patient in the "face down" position as described by Brown
(1948).   After one hour's operating time the left upper
lobe and lingula were seen to be almost completely atelectatic.
Inflation under positive pressure resulted in re-aeration of
the inferior division of the lingula only.   Two c.c's of 1
per cent procaine hydrochloride were injected between the
left vagus nerve where it crossed the arch of the aorta and
the posterior wall of the main bronchus.   After approximately

two minutes the remaining portion of the lingula and the upper lobe of the left lung could be easily and completely inflated with slight positive pressure.

Comment on Case 2. It was necessary to inject the procaine solution more distally in this case as the anterior pulmonary plexus is not readily accessible at lobectomy, being covered by the pulmonary artery and superior pulmonary vein. Resolution of the atelectasis, although less dramatic, was none the less complete. An interesting feature was the exclusion of the inferior division of the lingula from the atelectatic process, suggesting that spontaneous atelectasis may have a segmental as well as a lobar and total distribution. (That scattered areas of lobular atelectasis may occur during thoracotomy is well known and was described by Lilienthal in 1930. Such areas are seen to be plainly marked out by pittings several centimetres in diameter and occur chiefly on the surface of the lung, resembling cicatrices. Positive inflation alone is usually sufficient to restore the lung to its normal size and colour.)

3. The Relationship between spontaneous atelectasis and post-operative atelectasis.

Cases I. and 2. thus provide further evidence in support of the contention that spasm of the bronchial musculature results from vagal stimulation. It is suggested that the

difference between post-operative atelectasis and
spontaneous atelectasis is merely one of degree, the
fundamental aetiological factor being the same in both
conditions.

Summary.

At the beginning of this chapter doubt is cast on
the adequacy of the explanation generally advanced to
account for post-operative atelectasis.   An argument,
couched in general terms, is developed in favour of
bronchial constriction or spasm, produced reflexly by
stimulation of the vagus nerve, being an all-important
factor in the production of post-operative atelectasis.
It is claimed that constricted bronchi so produced can
account for: (a) the rapid accumulation of mucus or
mucopus in sufficient quantity to cause bronchial
obstruction in a previously healthy lung irrespective of
the type of anaesthesia used; (b) the failure of ciliary
action and bronchial peristalsis to expel secretions;
(c) the unproductive cough; (d) the reduction in vital
capacity following abdominal operation; (e) the
disappointing results obtained with standard methods of
treatment.   Reasons why atelectasis is not more common
in asthma are given and it is suggested that the increased
incidence of post-operative atelectasis among service
personell may be the result of predisposition to spasm of

the bronchial musculature. Confirmation of the bronchial
constriction theory is to be found in the experimental
work of de Takats and his colleagues. There is a brief
description of a type of atelectasis that may occur during
anaesthesia and the term spontaneous atelectasis is
suggested for this condition. Two cases illustrative of
spontaneous atelectasis are described and reasons are
given for assuming that this type of atelectasis also
results from reflex vagal activity.

* * * * * *

AN INVESTIGATION OF ATELECTASIS COMPLICATING

THORACOPLASTY IN PULMONARY TUBERCULOSIS.

INTRODUCTION:

It is not surprising that atelectasis frequently complicates the operation of thoracoplasty performed on the victims of pulmonary tuberculosis. In this disease many of the conditions that predispose to post-operative atelectasis may be present, namely (a) mucopurulent sputum, often copious; (b) bronchial stenosis due to the pressure of enlarged hilar glands or to the presence of endo-bronchial tuberculosis; (c) endobronchial tuberculosis may lead to the formation of granulation tissue within the lumen of the affected bronchi with consequent loss of ciliary action and stagnation of secretion. (Keers, 1950); (d) the operation of thoracoplasty disrupts the integrity of the hemithorax and impedes the efficiency of expectoration; (e) post-operative pain tends to give rise to suppression of cough and a reluctance on the part of the patient to expectorate accumulated secretions; (f) too active movements are not encouraged in the early post-operative period and, even under the most favourable circumstances, a certain immobility of the patient is to be expected; (g) the effects of a pre-existing paralysis of the isolateral hemi-diaphragm, with or without associated pneumoperitoneum, may still further add to the hypoventilation already present.

In Chapters II and III material is presented to show
the incidence of post-operative atelectasis which
complicated thoracoplasty performed on 175 patients
suffering from pulmonary tuberculosis with cavitation.
A total of 180 consecutive thoracoplasties were
investigated but, in the final analysis, five cases, in
whom there existed pre-operative atelectasis, have been
omitted.   The material comprises an unselected series of
cases drawn from the Glasgow area and the surrounding
districts.   The operations were performed in Mearnskirk
Hospital by the surgeons of the Thoracic Unit; the
anaesthetics were administered by the author and his
assistants.   The period under review extends from
December, 1946 to October, 1950 and for part of this time
(October, 1947 - 49) the author was serving with His
Majesty's Forces.   The investigation has not suffered
greatly as a result since complete records of the data to
be presented later were kept during this period.   There
is a certain lack of detail concerning the clinical
findings in some of the patients with post-operative
atelectasis but this has no bearing on the conclusions
finally reached.

1). <u>Method of Investigation.</u>

Stringer (1947) stresses the value of radiography
within 24 hours of operation in the observation and diagnosis

of post-operative atelectasis.   It is the writer's opinion that no investigation into the incidence of this type of atelectasis can be complete without the assistance of routine post-operative radiography.   In many hospitals and sanatoria in this country such a procedure is virtually impossible owing either to a lack of suitable facilities or to an insuffiency of x-ray staff.   The present investigation has not suffered from this disadvantage as there exists for the sole use of the Mearnskirk Thoracic Unit an x-ray plant complete with facilities for portable radiography.   It has been possible therefore to secure in every case a radiograph of the chest within 24 or 48 hours of operation.   From a study of these radiographs together with the clinical findings, the diagnosis of atelectasis was made.   Subsequent films were taken at intervals of several days in order to observe the course of the pulmonary lesion.

The incidence of atelectasis has been analysed statistically in relation to sex, age, operative risk, site and extent of disease, size of cavity, the operation of phrenic crush, number of ribs resected, apicolysis, the anaesthetic and season of the year.   Details of each patient submitted to thoracoplasty are to be found in Appendix II.   Appendix III deals with the cases of post-operative atelectasis and gives details of the day of onset, the degree of post-operative reaction and the maximum

sustained temperature, pulse and respiratory rates that were recorded. Wherever possible the patients with atelectasis have been followed up for periods varying from six months to three years. In Appendix IV there is tabulated the time of occurrence of the atelectasis in relation to the stage of thoracoplasty together with the end result, both immediate and remote.

As far as I am aware no similar study in regard to the incidence of atelectasis complicating thoracoplasty has been conducted. It is unfortunate, therefore, that the results obtained cannot be compared with the findings of others. In certain respects the published figures of Semb (1937), Millar (1948) and Gray (1948) are available for comparison and wherever possible such comparison has been made.

My thanks are due to Mr. Robert S. Barclay, Consultant Surgeon to the Thoracic Unit, Mearnskirk Hospital, for his encouragement and unfailing willingness to discuss problems in regard to the assessment of patients and the inter-pretation of radiographs. I am indebted to Dr. John G. Stevenson, Senior Registrar, Mearnskirk Hospital, who carried out the bronchography in the cases of post-operative atelectasis.

Chapter I.

Background of the Investigation.

1) Definition of Thoracoplasty.

Thoracoplasty is an operation in which ribs or segments of ribs are removed to permit the soft tissues of the chest wall to collapse inwards (Donaldson, 1947). Thoracoplasty as performed in the treatment of pulmonary tuberculosis is a form of compression or collapse therapy and is based upon the premise that the tuberculous lung is more liable to heal when its respiratory excursions are diminished and its diseased areas partially or completely collapsed.

The large majority of patients selected for operation are those in whom a cavity or cavities have failed to close as a result of other simpler methods of treatment. In many, the induction of an artificial pneumothorax has proved impossible because of pre-existing obliterative pleurisy or ineffectual because of an extensive system of pleural adhesions. In others phrenic crush, with or without a supplementary pneumoperitoneum may have been tried in an effort to effect cavity closure.

2) Development of the operation:

The operation of thoracoplasty has developed from an original and independent proposal by Quincke and Spengler (1888) that rib resection over the site of a rigid walled cavity should encourage its healing and resolution. There

were many and varied attempts at implementing this suggestion but it was not until Sauerbruch (1913) developed and perfected the technique of paravertebral thoracoplasty that satisfactory results were achieved.   Sauerbruch's operation aimed at reducing the capacity of the hemithorax and effecting a relaxation of the whole of the diseased lung by resecting posterior segments of the first to the eleventh ribs.   He preferred a single stage operation the indications for which were unilateral, predominantly fibrotic tuberculous disease in a patient who was non-toxic and apyrexial.

Sauerbruch's thoracoplasty undoubtedly provided the first great stimulus to major surgery in pulmonary tuberculosis.   In those early days, however, selection of cases was more restricted and the present day insistence on cavity closure was not greatly emphasised.   Paravertebral thoracoplasty, which compressed healthy as well as diseased portions of the lung, was frequently attended by a severe degree of surgical shock.   It provided lateral but no vertical relaxation of the underlying lung and cavity closure was affected only in 25 per cent of cases. (Thomas and Cleland, 1942).

The disadvantages of the Sauerbruch operation were in large part overcome when Semb (1937) and his associates of the Norwegian School developed the procedure of upper selective thoracoplasty with apicolysis.   This operation was

designed, by limiting the extent of rib resection, to affect mainly the diseased area of the lung and to conserve as much as possible the uninvolved pulmonary tissue.   Apicolysis or mobilisation of the apex of the lung involved freeing of the apex from its attachments at the root of the neck and thus allowed relaxation in the vertical as well as in the horizontal plane.   As approximately 95 per cent of cavities in pulmonary tuberculosis are situated in the apex of the lung the direct result of this manoeuvre was an increased incidence of cavity closure.   Originally Semb performed a one-stage operation combining apicolysis with wide resection of the upper five ribs. (In extensive disease he resected as many as seven ribs in a single stage). This proved to be a very severe procedure mainly due to the large area of chest wall that became mobile thus subjecting a considerable portion of the lung to paradoxical respiration.   Later he preferred a multiple stage thoracoplasty and by so doing reduced the fatality rate considerably.

The operation has been little modified in recent years but its indications have widened.   It is not unusual for thoracoplasty to be performed in the presence of bilateral tuberculous disease or when an artificial pneumothorax is partially compressing the contralateral lung.   Occasionally

apical or even partial thoracoplasty is performed on both sides of the chest.

3). Classification of Thoracoplasty.

The operation of thoracoplasty may be classified according to the total number of ribs resected. Apical thoracoplasty involves resection of the first three or four ribs only. In partial thoracoplasty varying portions of more than four but less then seven ribs are removed. In complete thoracoplasty seven or more ribs are resected.

4). Operative technique of Thoracoplasty.

The modern thoracoplasty as performed in the treatment of pulmonary tuberculosis is carried out in stages in order to lessen surgical shock, to prevent too sudden a drop in vital capacity and to diminish the danger of paradoxical respiration which follows too wide removal of the chest wall support. Ideally the interval between stages should not exceed two or three weeks - this interval permits of a certain stabilisation of the chest wall structures and of the mediastinum. A longer period may result in sufficient regeneration of ribs to interfere with the collapse process. It is generally agreed that not more than three ribs should be resected at any one stage. At the first stage, the whole of the first rib and its cartilage are resected
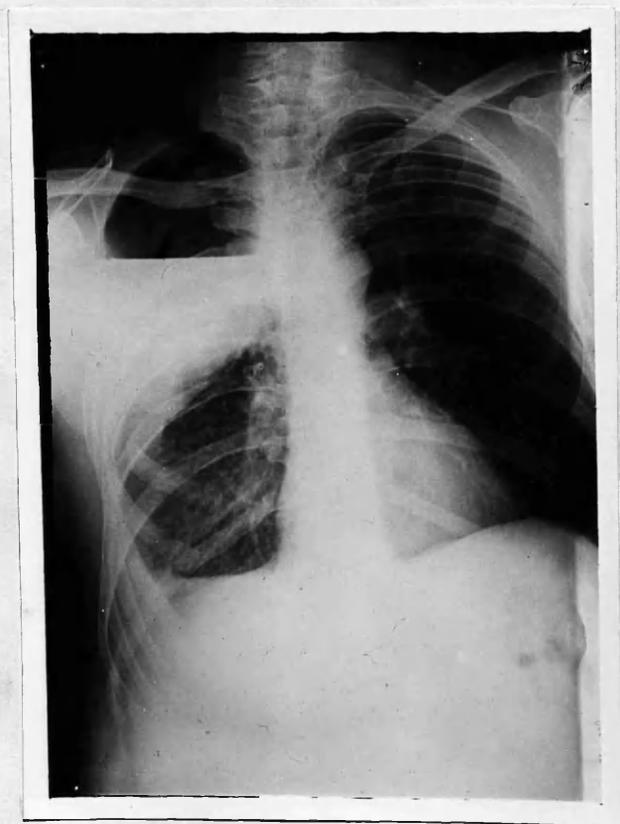
Radiograph I   Case 162

together with the whole of the second and the major part
of the third rib.   The apex of the lung is freed by
dividing its attachments (Sebileau's bands and Sibson's
fascia) to adjacent structures and the lung is mobilised
in the extrapleural or extrafascial plane as far as the
level of the hilum.   As a result of apicolysis a space,
Semb's space, is created extending from the freed surface
of the lung to the root of the neck.   A considerable sero-
sanguinous effusion frequently occurs into Semb's space
during the early post-operative period following extensive
apicolysis.   Absorption of this effusion usually occurs
within a matter of one or two weeks but it may take
considerably longer to disappear.   Radiograph I of Case
162 shows that an extensive apicolysis has been effected
following thoracoplasty.   There is a considerable
collection of fluid and air in Semb's space which is
having the beneficial effect of causing excellent
compression of the underlying lung.

Depending upon the degree of cavitation and the
extent of pulmonary involvement varying numbers and lengths
of ribs are excised at a subsequent stage or stages of the
operation.   In general when the fourth and fifth ribs are
dealt with approximately two-thirds of their lengths are
removed.   Smaller portions are excised from any additional
ribs that may require resection.   Occasionally the standard

thoracoplasty procedure fails to effect complete cavity closure and at a later date it may be necessary to perform a supplementary or revisionary stage of the operation. This procedure is designed to effect still further inward collapse of the chest wall. According to the needs of the situation the costal cartilages and remaining portions of a varying number of upper ribs are resected.

5). Thoracoplasty as performed at Mearnskirk Hospital.

The thoracoplasties performed on the 175 patients concerned in this study conformed substantially to the technique outlined above. It was usually decided pre-operatively, from a study of the extent of the pulmonary involvement and the degree of cavitation, the number of ribs likely to require resection in order to control the disease. If, for example, it was considered that an eight rib thoracoplasty was necessary, the routine procedure was to resect three ribs at the first stage, three at the second and two at the third. In certain cases where the disease appeared to be unusually chronic and the mediastinum fixed more than three ribs were resected at one stage. Furthermore the number of ribs resected at one stage was apt to vary with the surgeon, one operator favouring the resection of a maximum number of ribs in a minimum number of stages. A total of 461 stages of thoracoplasty were performed. (See Table I).

TABLE I.

Operation stages in 175 cases of thoracoplasty.

Stage of Thoracoplasty.	Number of Cases.
First	175
Second	164
Third	111
Revisionary	11
Total:	461

When dense fibrotic disease was encountered in the region of the lung apex, apicolysis was sometimes dispensed with on account of the difficulty involved and the fear of an infected Semb's space. In certain patients, usually poor-risk, toxic cases, it was considered that partial mobilisation of the apex only was justified. In others, partial mobilisation was sufficient to produce the desired effect. The majority of the patients however were given the benefit of lung mobilisation as far as the hilum. The extent of apicolysis also tended to vary with the operator, one surgeon having more enthusiasm for the procedure. The degree of apicolysis in 175 cases of thoracoplasty is shown in Table II.

TABLE II./

TABLE II.

The degree of apicolysis in 175 cases of thoracoplasty.

Apicolysis.	No. of patients.
Extensive	104
Partial	42
None	29
Total:	175

6). Fatality.

There was no anaesthetic death in the series. Four patients, all after first stage thoracoplasty, died within three months of operation giving an operative fatality rate of 2.29 per cent. Case 2. and Case 50. died of unrelieved atelectasis on the 24th and 8th post-operative days respectively. Case 4. succumbed on the 9th post-operative day of a spontaneous pneumothorax in the contralateral lung. Another patient, Case 103., had a fatal cerebral haemorrhage on the 10th day following operation. Comparative fatality rates quoted by others are Semb (1937), 3 per cent; Sellors (1947a), 2.7 per cent; Millar (1948), 5.3 per cent; Gray (1948), 3.6 per cent; Goldman, Segal and Sherman (1950), 5.8 per cent; Hagn-Meincke (1950), 3 per cent.

7). Indications for Thoracoplasty.

It is not usual for thoracoplasty to be adopted as a

primary procedure and in general only patients in whom
treatment with artificial pneumothorax or other more
conservative methods of collapse therapy have been
attempted without success are treated by this procedure.
Broadly the indications for thoracoplasty are unilateral
or predominantly unilateral pulmonary tuberculosis with
cavitation. Disease, if present in the contralateral
lung, should be either stationary or controlled. The
best results are obtained in the so-called stable chronics,
i.e. patients in whom the disease has been present for some
time and is in the productive or fibrotic phase. Such
cases are in good general condition, often apyrexial,
showing little or no signs of toxaemia and their only
symptoms are cough and sputum which is infected with the
tubercle bacillus. Eighty or 45.71 per cent of the cases
under review were considered to belong to this group. A
second less favourable category of patient is the relapsing
chronic. The disease in this type of case although mainly
productive shows a certain amount of exudative infiltration
and the patient has intermittent periods of pyrexia, toxic
symptoms, increase in cough and sputum and loss of weight.
Fifty-seven or 32.57 per cent of the cases submitted to
thoracoplasty belonged to this category. The lower end of
the scale is occupied by the slipping chronics, poor-risk
cases who have shown no improvement with general measures.

The disease in these patients is often bilateral, usually there is evidence of fresh exudation and surgery offers the only chance of survival.   Of the 175 cases under review, 38 or 21.71 per cent were considered to be slipping chronics. Thoracoplasty in the slipping chronic was by no means a measure of despair as many, especially since the advent of streptomycin therapy, were saved from developing into the category of the hopeless.   Indeed it was not a rare experience to achieve sputum conversion in such cases and to return what was previously a public menace to some form of useful occupation.

The presence of an active non-pulmonary tuberculous lesion must be given consideration when fitness for thoracoplasty is assessed.   Tuberculous laryngitis, when not sufficiently far advanced to cause pain that interfered with effective coughing, was not considered a contra-indication and usually disappeared following healing of the pulmonary lesion.   Mild infection of the genito-urinary tract does not rule out thoracoplasty but the operation is contraindicated in the presence of intestinal tuberculosis. A high blood sedimentation rate has not necessarily been a contraindication but if serial readings showed that the blood sedimentation rate was rising, postponement of the operation was sometimes considered.

8). <u>Preparation of the patient for Thoracoplasty.</u>

(a) <u>General measures</u>:   As thoracoplasty is an
operation of election ample time is afforded to institute
methods of therapy to improve the general condition of the
patient.   A period of preoperative sanatorium treatment
is therefore highly desirable and, with a view to improving
their general condition, many of the less fit patients in
this series were under observation for several months
before operation was eventually undertaken.   When it became
obvious that preliminary general measures were having little
effect, operation was not further delayed.   Whenever possible
the patients were ambulant for part of the day during the
preoperative period in order to improve vasomotor and muscle
tone.   Vigorous breathing exercises were not encouraged but
tuition by physiotherapists in the correct use of the
diaphragm and abdominal muscles was instituted as a
routine and in many instances a very desirable improvement
in vital capacity resulted.   It was found that when the
bases of the lungs were used to the best advantage there
was less respiratory distress and less paradoxical breathing
during the immediate post-operative period.   Patients
likely to benefit from oxygen therapy following operation
were instructed in the use of the B.L.B.* mask.   Those
with copious sputum were encouraged to expectorate as
much as possible on the morning of operation but postural
drainage was considered harmful and was not practised

* Boothby, Lovelace and Bulbulian.

practised.   When the immediate preoperative radiograph
showed a recent spread of disease or in the event of a
new rise of temperature thoracoplasty was postponed
until a more favourable time.

Patients about to undergo thoracoplasty appreciated
a simple explanation of the principle underlying the
operation with reasons for the varying number of stages
that might be required.

It must be admitted that, although the above measures
were adopted later as a routine procedure, in the early
cases preoperative preparation was not adequate.   When
the Thoracic Unit opened at Mearnskirk Hospital the
waiting-list for thoracoplasty was so great and beds at
such a premium that it was necessary to submit the patients
to thoracoplasy soon after admission to hospital.   Further
the patients in some instances were discharged to sanatoria
all too soon after completion of thoracoplasty.   This
system worked reasonably well for institutions where the
standard of preoperative preparation and post-operative
care was high.   Such, however, was not always the case.
As the work of the Thoracic Unit progressed and results were
assessed it was decided as far as the waiting-list would
permit to admit the patients for one or two weeks before
operation and to retain them longer after completion of
thoracoplasty.   It will be seen later that improved results

accrued from this procedure.

(b) <u>Drug therapy</u>:   Until recently drug therapy played
no specific role in the treatment of pulmonary tuberculosis.
The sulphonamide drugs and penicillin were prescribed in the
early cases of thoracoplasty but no results can be claimed
from their use.   Early in 1949 streptomycin and para-amino
salicylic acid (P.A.S.) became available and the later cases
were given the benefit of treatment by these drugs.   The
routine employed has been:-

> (i) <u>Streptomycin</u> - 1 gramme per day commenced 1 week
>   before operation and continued until a maximum of
>   90 grammes has been administered.
>
> (ii) <u>P.A.S.</u> - an average of 15 grammes per day given for
>   2 weeks before operation. Therapy with P.A.S. was
>   discontinued during the course of thoracoplasty
>   and recommenced 1 week after the completion of the
>   final stage when it was continued for approximately
>   3 months thereafter.   If signs of intolerance such
>   as urticarial rash, vomiting and diarrhoea appeared,
>   P.A.S. therapy was discontinued.

9. Anaesthesia for Thoracoplasty.

Anaesthesia for thoracoplasty is a somewhat contentious
subject.   As Mushin (1948) puts it, "Anaesthesia for
thoracoplasty is a bone of contention among those who gather
round the tubercular patient."   There is considerable

difference of opinion as to whether local or general anaesthesia is the method of choice for thoracoplasty, each technique having its supporters and detractors. On occasion one finds an advocate for spinal anaesthesia, one such being Hewer (1944). A review of the recent literature on the subject finds Semb (1937), Thomas and Cleland (1942), d'Abreu (1947), Davies (1948), Weinstein (1948) and Millar (1948) advocating local anaesthesia for thoracoplasty while a general anaesthetic technique is favoured by Roberts (1947), Mountford (1947), Tubbs (1947), Watt (1947), Sellors (1947b), Gillies (1947), Gray (1948) and Pinkerton (1948).

Beecher (1941), Lindskog (1941), Burstein and Alexander (1947) and Mushin (1948) agree that equally good results may be obtained from either local or general anaesthesia provided the technique is carried out with skill. Whatever the anaesthetic method employed certain conditions must be provided to ensure the best results for thoracoplasty. There must be quiet respiration during the entire operating period in order to reduce to a minimum paradoxical movement of the apex during mobilisation. The anaesthetic agents used must be non-irritating to the lungs and provide for rapid recovery of reflexes. Adequate oxygenation must be maintained throughout if the dangers of anoxia are to be avoided. The bronchial tree must be kept free of sputum during operation.

It is not proposed to take part here in the controversy
regarding the relative merits of local and general anaesthesia
for thoracoplasty.   The point is made, however, that general
anaesthesia with cyclopropane as the principal agent together
with intermittent aspiration of sputum and secretions during
operation, provides all the essentials mentioned above.
Furthermore the mental stress, trauma and uncontrollable
bouts of coughing sometimes met with in the patient under
local anaesthesia are not encountered.   Cyclopropane
anaesthesia however does present certain disadvantages.   It
precludes the use of diathermy, there is apt to be an
increased oozing of blood at the operative site, disorders
of cardiac rhythm are sometimes encountered and there may be
a fall of blood pressure at the conclusion of anaesthesia.
In regard to the present investigation both surgeons and
anaesthetists agree that the advantageous conditions provided
by cyclopropane anaesthesia outweigh any disadvantage that
might occur from its use.   At Mearnskirk Hospital
diathermy is not used for arresting haemorrhage and some
post-operative oozing into Semb's space is considered helpful
in preventing re-expansion of the mobilised apex.   No
untoward effects seem to follow cardiac irregularity under
cyclopropane anaesthesia and in fact cyclopropane is often
the agent of choice for cardiac surgery (Rink, Helliwell and
Hutton, 1948, and Evans, 1949).   The fall in blood pressure
at the conclusion of the anaesthetic, the so-called

"cyclopropane shock" of Dripps (1947) does not prove

troublesome provided a gradual reduction in the concentration

of cyclopropane is effected towards the end of operation.

10). The Anaesthetic technique for Thoracoplasty.

The anaesthetics in this study were administered by

several anaesthetists and in general the technique employed

was as follows:-

Premedication with omnopon grain 1/3rd and scopolamine

grain 1/150th was given by hypodermic injection one hour

before operation. (Poor risk patients were more lightly

premedicated with morphine sulphate grain 1/6th and atropine

sulphate grain 1/100th). Anaesthesia was induced with

intravenous thiopentone, the average dose being 0.5 gramme.

The anaesthetic was maintained in the first plane of

surgical anaesthesia with a mixture of cyclopropane and

oxygen administered from the circle absorption type of

closed circuit apparatus. When the induction phase was

completed a muscular relaxant (d-tubocurarine chloride or

gallamine triethiodide) was injected intravenously in

sufficient amount to obtund the laryngeal reflex. (The

doses required for this purpose varied between 10 to 15

millegrams for d-tubocurarine chloride and 60 to 80 mille-

grams for gallamine triethiodide). Oral intubation with a

number 7 or 8 Magill tube was effected under direct vision

using a Macintosh laryngoscope, a pharyngeal airway was

inserted alongside the endotracheal tube and the face piece held in position by means of Clausen's harness. Sputum was aspirated from the bronchial tree during and at the end of operation by means of catheter suction. Towards the end of operation the concentration of cyclopropane was decreased and the gaseous mixture diluted with air.

In order to compare the incidence of post-operative atelectasis when a semi-open method of anaesthesia was employed a certain number of patients were anaesthetised by substituting a sequence of nitrous oxide, oxygen and trichlorethylene for cyclopropane. Otherwise the anaesthetic technique was identical with that described above. The semi-open method was found to be less satisfactory in that respiration was frequently not sufficiently peaceful to allow of satisfactory operating conditions and a degree of anoxia was inclined to develop in patients with a low vital capacity. The results of this investigation are found in Chapter III.

11). Spread of the tuberculous disease following operation.

The advocates of a particular type of anaesthesia for thoracoplasty are wont to claim a decreased incidence of spread of the tuberculous disease following operation in support of their method. It is rarely, however, that precise figures are given. In the 175 cases under review there were 11 instances of spread or re-activation of

disease during the immediate post-operative period, an incidence of 6.29 per cent. In six of these patients there was merely an extension of the pulmonary involvement downwards in the thoracoplasty lung. Spread to the opposite side appeared in five cases (2.86 per cent). Millar (1948), reporting a series of 204 thoracoplasties performed under local anaesthesia quotes contralateral spreads only, the incidence being 2.94 per cent. Overholt (1937) in 253 patients under general anaesthesia encountered 18 instances of spread of the disease (6.28 per cent). Harrison and Berry (1943) found spread of the tuberculous disease in 16 (10.7 per cent) out of 150 cases of thoracoplasty performed under various methods of anaesthesia.

Thomas and Cleland (1942) consider that spread of the tuberculous disease is associated with post-operative atelectasis. In the present series spread was not significantly related to post-operative atelectasis, occurring in three only of the 37 instances of atelectasis, an incidence of 8.11 per cent.

12). Post-operative management.

The post-operative management of the cases of thoracoplasty as it is practised at Mearnskirk Hospital is now briefly described. Liberal dressings applied over the area of rib resection and held firmly in position by adhesive strapping tends to limit paradoxical respiration.

As soon as the patient has receovered from the anaesthetic
he is placed in the semi-upright position in order to cause
a minimum of respiratory embarrassment and to facilitate
coughing.   Oxygen is administered freely during the early
post-operative period as it is desirable to have quiet
respiration in order to minimise pain and discomfort and the
risk of spread of the disease.   The rare occasions when
blood transfusion is necessary occur after first stage
thoracoplasty in the event of an unusual degree of
haemorrhage into Semb's space.   There is usually an increase
of sputum after operation, especially following the first
stage of thoracoplasty.   It is important that sputum be
voided as expeditiously as possible as its accumulation in
the bronchial tree will precipitate atelectasis or
bronchogenic spread of disease.   Expectoration is aided by
medication with expectorant mixtures and frequent inhalations
of the compound tincture of benzoin.   Small doses of morphine
sulphate (grain 1/8th to 1/6th) help to allay the pain of
coughing without depressing the cough reflex.   The nursing
staff are instructed to apply firm digital pressure in the
region of the unsupported apex of the lung; this manoeuvre
greatly assists the patient in his efforts at voiding sputum.
More recently (dating from Case 117.), posturing has been
practised in all patients not suffering from acute respiratory
distress.   The method is as follows: On the evening of
operation the patient is laid down flat without pillows for

ten minutes.   He is then turned on each side for a similar
period.   This process of posturing, which is repeated twice
daily for one week, usually results in expectoration of
unexpected amounts of sputum.

On the day following operation the patient is visited
by the physiotherapist who corrects faulty posture and
ensures that he is breathing diaphragmatically.   Passive
movements of the shoulder girdle and arm are instituted on
the second or third day.   Later active movements are
practised and usually by the tenth day both arms can be
completely elevated.   Patients who are ambulant for two
or three hours are given graduated class exercises to
ensure that good posture and arm movements will be maintained.
Some degree of scoliosis inevitably follows thoracoplasty
but thanks to the art and skill of the physiotherapists
the large majority of patients achieve an extraordinary
good posture in spite of the destructive effects of
thoracoplasty.

It has been seen that ideally the stages of
thoracoplasty are performed at intervals of two to three
weeks.   The patient is confined to bed between stages and
the average case is but little upset by the operation.
Subsequent stages however may have to be postponed on
account of a poor general condition of the patient or in
the presence of wound infection, spread of disease or post-

operative atelectasis.   If the interval between stages is delayed longer than one month regeneration of ribs may interfere with the adequate compression of lung tissue that is so desirable.

Four weeks after the final stage of thoracoplasty the patient is allowed up for gradually increasing periods. An elevation of temperature or of the blood sedimentation rate suggests that rehabilitation has been too rapid and a return to bed rest regime is indicated.   The average case may be discharged from hospital about six months after thoracoplasty.   It is impressed upon the patient that his operation is not in itself a cure but merely a measure employed towards the ultimate healing of the tuberculous lesion.   He is instructed to rest as much as possible, to restrict his activities to walking in the fresh air and to return at frequent intervals to the out-patients department.   If progress be maintained it is often possible to return the patient to light work within nine or twelve months of operation.

*   *   *   *   *   *   *

Chapter II.

Post-operative Atelectasis following thoracoplasty.

1) Incidence of post-operative Atelectasis.

Out of 175 cases of pulmonary tuberculosis with cavitation subjected to thoracoplasty, post-operative atelectasis occurred in 37 patients, an incidence of 21.14 per cent.

TABLE I.

The incidence of atelectasis following thoracoplasty.

Cases of Thoracoplasty.	Cases of Atelectasis.	Atelectasis per cent.
175	37	21.14

Reference to Appendix II will show that 15 instances or 50 per cent of atelectasis complicated the first 30 thoracoplasties performed in the series. (In the remaining 145 patients there were 22 instances of post-operative atelectasis, an incidence of 15.17 per cent). Early in the investigation it was considered that the period during which the staff of the Thoracic Unit was gaining experience of the condition together with the initial inadequacy of preoperative preparation and post-operative care for reasons already stated was reflected in the high incidence of post-operative atelectasis at the outset. A subsequent decrease in the frequency of atelectasis tended to confirm this impression. That such was not the case however is

shown in Chapter III where analysis indicates that the high
incidence of post-operative atelectasis in the first 30
cases of thoracoplasty was due to the relatively high
proportion of predisposing factors which characterised these
cases.

2) First appearance of atelectasis in relation to the stage
   of thoracoplasty.

   Twenty-eight cases of atelectasis occurred after first
stage, 6 appeared for the first time following second-stage
and 3 appeared similarly after third-stage thoracoplasty.
When the condition resolved in whole or in part between
stages of the operation, the atelectasis returned in every
instance following subsequent resection of ribs.

TABLE II.

First appearance of atelectasis in relation to stage of
thoracoplasty.

Stage of Thoracoplasty.	No.of Operations.	First appearance of Atelectasis.	Atelectasis per cent.
First	175	28	16.00
Second	164	6	3.66
Third	111	3	2.70
Revisionary	11	Nil	—
Total:	461	37	8.03

It would appear that post-operative atelectasis as such is not recognised by many workers in the field of thoracoplasty. Urquhart (1937), Overholt (1937), Harrison and Berry (1943), Hagn-Meincke (1950) and Goldman, Segal and Sherman (1950), reporting the results of thoracoplasty in large series of cases do not mention the condition. A radiograph reproduced in an article by Aufses (1937) to indicate bronchiectasis following thoracoplasty shows typical massive atelectasis yet this fact is not mentioned in the text.

Thomas and Cleland (1942), who are very much aware of the complication of atelectasis, describe an incidence of 20 per cent following thoracoplasty and admit that some cases are not recognised. Semb (1937) found that the incidence of post-operative atelectasis after 256 first stage thoracoplasties varied from 4 per cent when three ribs only were resected to 62 per cent when seven ribs were removed. Millar (1948) quotes 5.5 per cent of atelectasis in 204 thoracoplasties and Gray (1948) gives a figure of 3.6 per cent in 153 patients.

It will bear repeating that the only way to make certain of the diagnosis of post-operative atelectasis is by routine serial radiography after operation.

3) Clinical Summary.

(a) Post-operative reaction: Whether atelectasis

occurred for the first time or recurred it was manifest on
the first or second post-operative day irrespective of the
stage of thoracoplasty.   The only exception was provided
by Case 113. in whom partial atelectasis was not evident
until the sixth day after second-stage thoracoplasty.   In
general three types of post-operative reaction were
encountered and it was found that these could be roughly
classified as severe, moderate or slight according to the
maximum sustained temperature recorded.   When the reaction
was severe a temperature of over 101° Fahrenheit was the
rule; when moderate the temperature ranged between 99° and
101° Fahrenheit; in a slight reaction there was little or
no rise in temperature.   When the atelectasis occurred for
the first time the post-operative reaction was usually
severe irrespective of the stage of thoracoplasty or the
degree of lung involvement.   As the large majority (28
out of 37 cases) occurred for the first time after first
stage thoracoplasty by far the greatest number of severe
reactions were found at this time.   The onset was usually
heralded by a sudden rise of temperature, pulse and
respiratory rates and the diagnosis was at once suggested
by a glance at the temperature chart.   The patient looked
ill and often complained of pain or a feeling of constriction
in the chest.   Dyspnoea was a common but not a constant
feature.   Cyanosis occurred in a comparatively small
number of cases but it was more usual for the victim of

atelectasis to present a flushed appearance.   In the early
stages cough was usually unproductive and often suppressed
on account of pain.   Later (within a few days in favourable
cases) coughing became easier and productive of considerable
quantities of muco-purulent sputum and marked symptomatic,
clinical and radiological improvement followed.   Whether
the atelectasis resolved or not, the temperature was apt to
return to normal within seven to ten days of operation.

The above picture, but to a lesser degree, was portrayed
by the patients who had a moderate post-operative reaction.
Some of the cases in this group complained only of a feeling
of constriction in the chest and difficulty in voiding
sputum.   Many of the patients with slight reaction were
diagnosed only on routine radiography.

Whether the atelectasis recurred or persisted the
reactions experienced during subsequent stages of
thoracoplasty became progressively less severe.   Details
of the day of onset of atelectasis together with the degree
of post-operative reaction are found in Appendix III.
Table III shows the degree of post-operative reaction that
was encountered in the cases of atelectasis in relation to
the various stages of thoracoplasty.   The total number of
88 post-operative reactions corresponds to the total number
of operation stages following which atelectasis was manifest.
An individual patient might have as many as three post-
operative reactions depending upon the number of stages of

his thoracoplasty after which atelectasis occurred.

TABLE III.

Post-operative reaction following the stages of
thoracoplasty.

Stage of Thoracoplasty.	Severe post-operative reaction.	Moderate post-operative reaction.	Slight post-operative reaction.	Total
First	21	5	2	28
Second	7	12	13	32
Third	3	6	15	24
Revisionary	Nil	2	2	4
Total:	31	25	32	88

(b) Clinical Examination: On clinical examination where
massive atelectasis existed there was dullness to percussion
over the area of atelectasis.   The breath sounds, at first
markedly diminished or absent, within a few days became
bronchial in character and accompanied by numerous coarse
rales and rhonchi.   The adventitious sounds were especially
prominent when resolution of the atelectasis was taking place.
Cardiac displacement is a valuable diagnostic sign but was by
no means constant.

As Thomas and Cleland (1942) point out too much reliance
cannot be placed on the physical signs as examination of the
chest is difficult on account of occlusive dressings.   Further

in the uncomplicated case of thoracoplasty the breath
sounds are often weak or absent as a result of the immobility
of the thorax that usually follows resection of ribs.

4). Classificiation of atelectasis following thoracoplasty.

Massive and partial types of atelectasis were
encountered and in every case the side of operation only
was affected.   In no instance was a single lobe only
(lobar atelectasis) involved.   In the massive variety
approximately two-thirds or more of the affected lung was
involved and a dense homogeneous opacity was evident on
radiological examination.   In the partial type scattered
areas of atelectasis occurred throughout the lung field and
the x-ray film showed areas of opacity in the midst of air-
bearing lung tissue.   The large majority of the cases
were massive in type and in many the entire lung was
involved.   Erwin (1939) and Thomas and Cleland (1942)
report a similar experience and comment on the rarity of
lobar atelectasis following thoracoplasty.   When partial
atelectasis occurred after an early stage of the
thoracoplasty it usually recurred as, or progressed to,
massive atelectasis following the later stages.

TABLE IV./
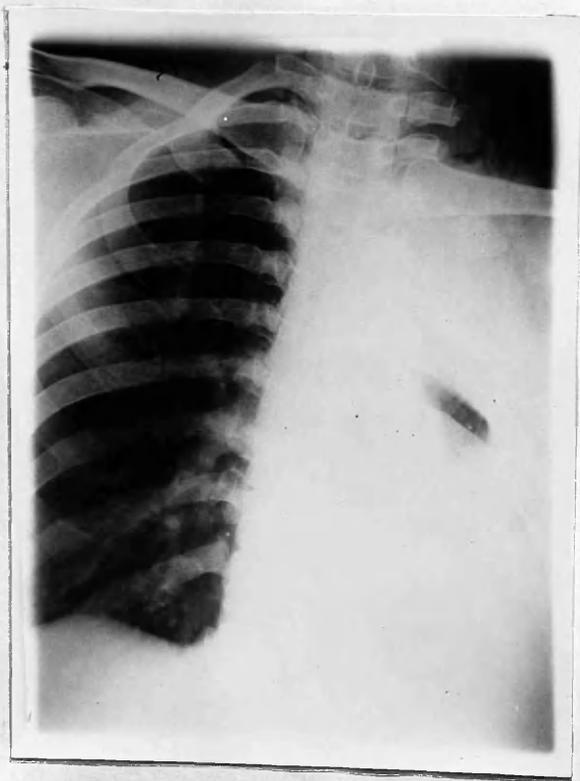
Radiograph II   Case 9

TABLE IV.

Classification of atelectasis and stage of thoracoplasty.

Stage of Thoracoplasty.	Massive Atelectasis.	Partial Atelectasis.	Total.
First	23	5	28
Second	27	5	32
Third	23	1	24
Revisionary	4	Nil	4
Total:	77	11	88

5) Radiography in atelectasis following thoracoplasty.

To arrive at a radiological diagnosis of atelectasis following thoracoplasty is by no means an easy task and considerable experience is required before one can inter-pretate the x-ray findings with accuracy.   Indeed it is often necessary to follow up an opaque lung shadow with serial radiographs before a diagnosis of atelectasis may be made.   It is the exception rather than the rule to find the typical signs portrayed in radiograph II of Case 9., taken three days after first stage thoracoplasty.   This film shows massive atelectasis of the left lung with pronounced cardiac retraction.   There is some air-bearing tissue in the upper part of the lung.   Unfortunately the photographic reproduction does not show crowding of the
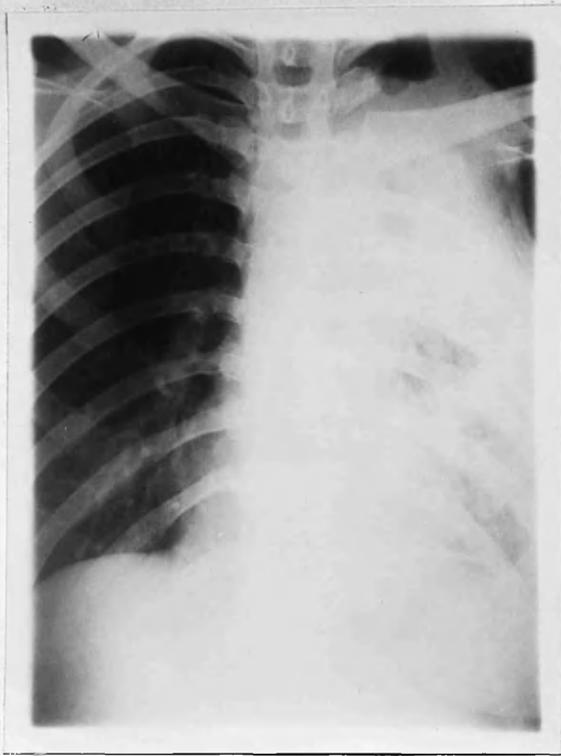
Radiograph III   Case 22
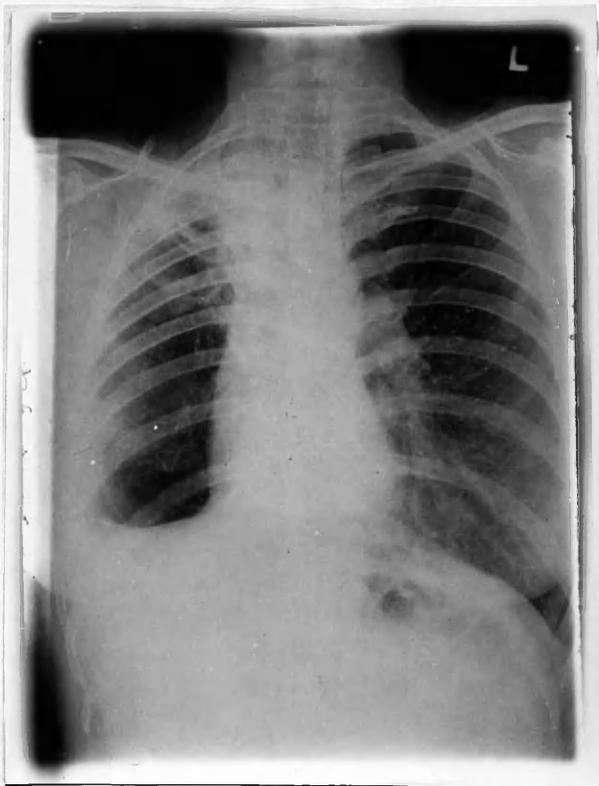
Radiograph IV   Case 131

unresected ribs that was apparent in the original
radiograph.   (The dense opacity that results in
atelectasis has been the cause of considerable technical
difficulty in achieving accurate contrasts in several of
the reproductions of radiographs and bronchograms).
Radiograph III of Case 22., taken on the day following
first stage thoracoplasty gives a typical picture of
partial or patchy atelectasis throughout the left lung
with cardiac retraction and some crowding of the
unresected ribs.   There is some effusion and air in
Semb's space.

Retraction of the heart towards the side of
atelectasis is a most useful guide to diagnosis when such
displacement occurs for the first time post-operatively.
Natural healing in pulmonary tuberculosis however is by
fibrosis which gives rise to a tendency towards retraction.
In chronic unilateral disease it is quite usual to have
crowding of the ribs, elevation of the hemi-diaphragm and
displacement of the trachea and heart shadows evident in
the preoperative radiograph of the chest.   The pre-
operative film (radiograph IV) of Case 131. shows all these
signs of retraction on the right side.   There is also
generalised right-sided pleural thickening with obliteration
of the cardio-phrenic and costo-phrenic angles.   The
crowding of ribs is especially evident in the region of the
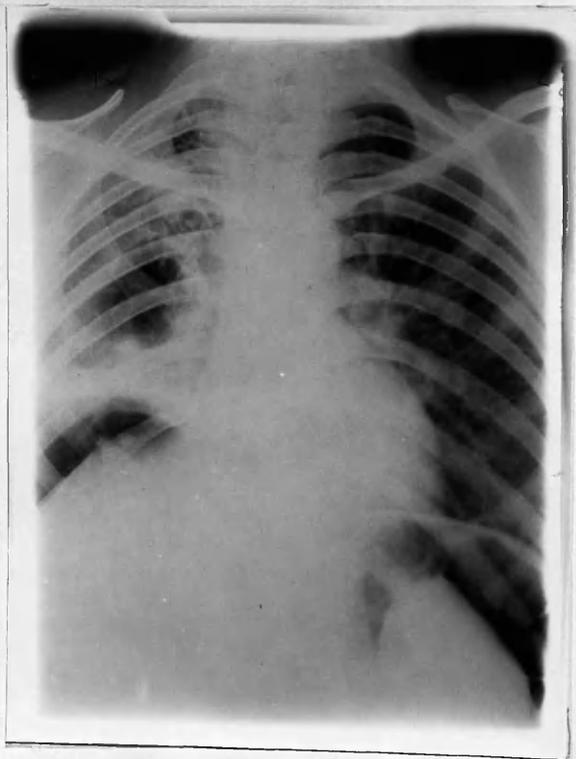
Radiograph V   Case 28
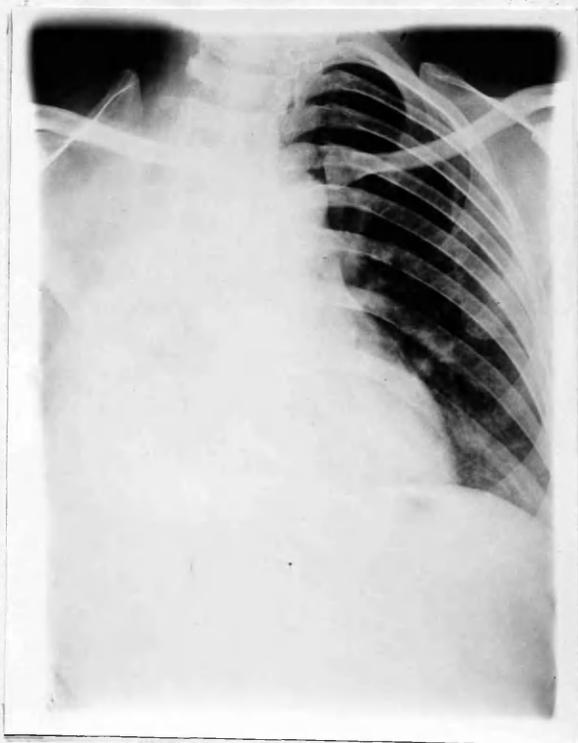
Radiograph VI   Case 28

upper part of the lung which is the seat of tuberculous disease showing much fibrosis with cavitation.   It is obvious therefore that a recent preoperative radiograph must be available for comparison lest undue weight be given to the presence of elevation of the hemi-diaphragm, cardiac and tracheal displacement, crowding of the ribs etc.   On the other hand the absence of such signs does not rule out a diagnosis of atelectasis as the frequent association of pneumothorax, pleural effusion or effusion into Semb's space may result in complete compensation for the decrease in size of the atelectatic lung.

Cardiac retraction may not be present in atelectasis following thoracoplasty when the hemi-diaphragm had previously occupied a high position as a result of the operation of phrenic crush.   The probable explanation is that when atelectasis supervenes no lateral pulmonary stresses are exerted on the lower part of the mediastinum. Radiographs V and VI of Case 28. serve to illustrate this point.   Radiograph V, a pre-operation film, shows tuberculous disease throughout the right lung with cavitation in both apex and base.   There is also healed tuberculosis in the upper half of the left lung.   Pneumo-peritoneum is present along with right phrenic crush resulting in elevation of both domes of the diaphragm, especially the right.   Radiograph VI was taken after a

Radiograph VII   Case 57

seven rib, two-stage thoracoplasty.   It shows massive
atelectasis of the right lung without any marked cardiac
retraction, probably due to the high level of the right
diaphragm.

Displacement of the trachea towards the side of
atelectasis, a prominent feature in massive atelectasis
due to causes other than thoracoplasty, is not usual when
extensive apicolysis has been effected.   In this instance
lateral pulmonary stresses are not exerted in the region of
the upper mediastinum when atelectasis supervenes.   Indeed
the pressure of a massive effusion into Semb's space may
cause displacement of the trachea away from the side of
atelectasis.

A pleural effusion may be mistaken for post-operative
atelectasis.   Radiograph VII of Case 57. shows resection
of the upper seven ribs on the left side and the lower part
of the left chest is opaque.   There is some displacement
of the heart towards the opposite side due to pressure of
the effusion.   Proof of the effusion was procured by
paracentesis thoracis and resultant clearing of the opacity.

When effusion and post-operative atelectasis co-exist
considerable diagnostic difficulty may be presented.
Several such cases were only diagnosed with certainty when
aspiration or absorption of the effusion revealed an
underlying atelectasis or when resolution of the atelectasis
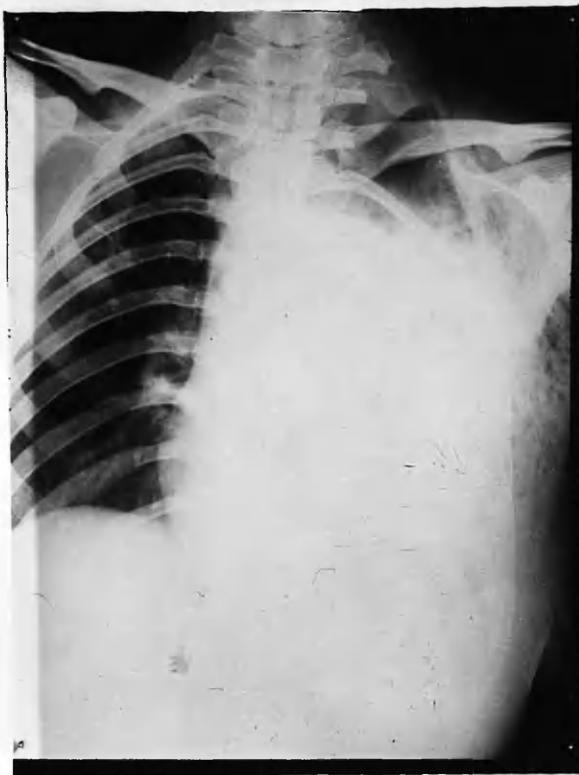
Radiograph VIII   Case 174   Radiograph IX   Case 174
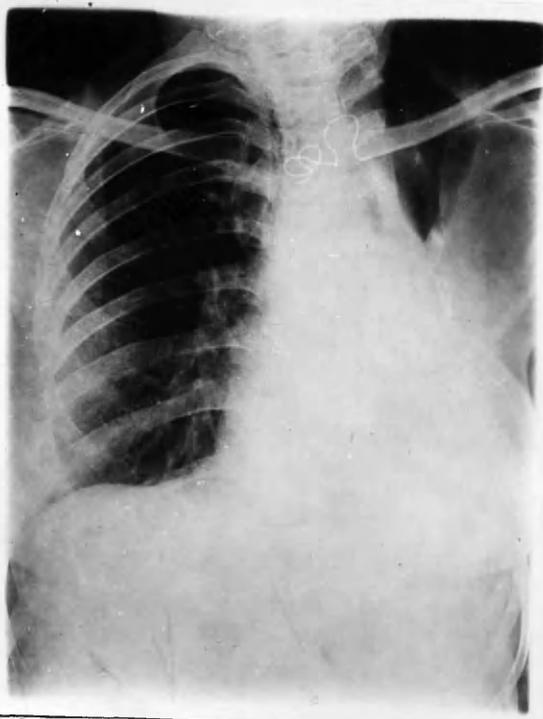
Radiograph X   Case 8   Radiograph XI   Case 8

revealed air-breathing lung in an area previously opaque.
Radiograph VIII of Case 174. would appear to indicate a
simple effusion as it shows a massive opacity on the left
side with displacement of the heart shadow to the right.
A subsequent film (radiograph IX) of the same case shows
that in the previous instance we were dealing not only
with effusion but with massive collapse of the left lung.
This film, taken after considerable expectoration of
mucopus, reveals the presence of air in the left lung
while the opacity due to the fluid in the upper part of
the chest still remains.   Radiograph X of Case 8. shows
an opacity throughout the left lung field.   A subsequent
x-ray (radiograph XI) reveals that the upper part of the
opacity had been caused by an effusion into Semb's space,
now absorbed.   The lower opacity is seen to be due to
massive atelectasis of the left lung.

A thin pleura is sometimes torn at operation giving
rise to pneumothorax and great obscurity in the post-
operative radiograph.   This point is illustrated in
radiograph XII of Case 126.   It shows resection of seven
ribs after second-stage thoracoplasty.   The state of the
underlying left lung is obscured by the presence of air and
fluid in the pleura which was inadvertently torn at
operation, and the presence of air and much effusion into
Semb's space.   Radiograph XIII of the same case shows

Radiograph XII   Case 126
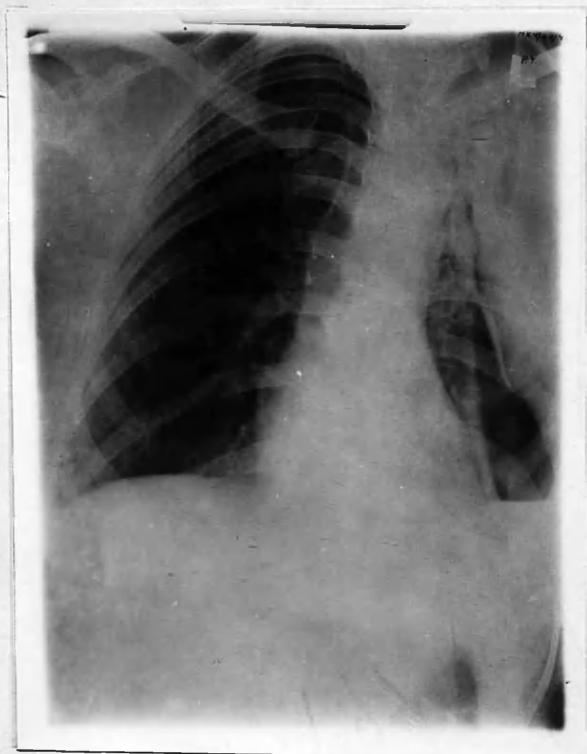
Radiograph XIII   Case 126

some absorption of the effusion in Semb's space, a lesser collection of fluid and air in the pleural space with some re-expansion of the underlying lung, which is obviously air-bearing.

It will be appreciated, therefore, that in the many instances where there is no severe post-operative reaction and where the radiological picture is obscure, a diagnosis of atelectasis may readily be missed unless the condition is sought for with diligence.

6). Treatment of atelectasis following thoracoplasty.

To a large extent the preoperative preparation and post-operative care of the thoracoplasty patient is aimed at the prevention of post-operative atelectasis. It has already been described how every effort is made to prevent the accumulation of sputum in the bronchial tree by the administration of expectorants and inhalations, by posturing and by support of the chest wall during bouts of coughing. Small doses of morphine sulphate, insufficient to depress the cough reflex, ease the pain of coughing and diaphragmatic breathing stimulates ventilation of the lung bases.

In established cases of atelectasis the above measures are more vigorously applied but care is necessary as the patients become easily exhausted. A mixture of five per cent carbon dioxide in oxygen is administered for two to

three minutes every four hours.   The cause of the condition
is explained to the patient and his co-operation regarding
posture and expectoration of sputum is sought.   It is
difficult to decide how vigorously the measures adopted
should be applied.   Post-operative atelectasis calls for an
intensive regime of activity on the part of the patient in
regard to change of posture and pulmonary ventilation.   On
the other hand immobility is desirable in order to obtain
the best results from thoracoplasty and to avoid spread of
the disease.   A mean compatible with both ends must be
found always bearing in mind that a patient in poor general
condition whose vital capacity is low may become completely
exhausted even with the mildest form of postural change.

a) Bronchoscopic aspiration of the bronchial tree.

Erwin (1939) was not impressed with the results of
bronchoscopic aspiration of mucopus from the bronchial tree
in atelectasis following thoracoplasty but Bence (1944) and
Millar (1948) reported good results from the procedure.
Five patients in the present series (Cases 2, 9, 18, 23 and
50) were treated by bronchoscopic aspiration.   These were
instances of massive atelectasis after first stage
thoracoplasty and each patient experienced a severe post-
operative reaction.   The procedure, which was conducted
under local anaesthesia, was originally embarked upon with

some trepidation as it was thought it might prove too much
of an ordeal in patients with acute respiratory distress.
Experience proved our fears to be unfounded for, provided
it is conducted with gentleness and skill, the victims of
massive atelectasis seem to tolerate bronchoscopic
aspiration surprisingly well.   The results however were
not encouraging and the method has been abandoned.   In
view of the fact that bronchoscopic aspiration is often
advocated in atelectasis of this nature, the brief resume
given below of the cases so treated will be of interest.

Case 2. - female, aged 19 years in poor general condition
with tuberculosis and extensive cavitation in the upper part
of the right lung.   Massive atelectasis of the right lung
was diagnosed two days after first stage thoracoplasty at
which the upper four ribs were resected.   Apicolysis was
not carried out.   A severe post-operative reaction and
considerable difficulty in voiding sputum occurred.
Bronchoscopic aspiration under local anaesthesia was carried
out on the 4th day after operation when a considerable amount
of mucopus was aspirated from the right main bronchus.
Slight improvement of air entry on the right side followed
but there was no radiological improvement.   By the 14th
day the temperature had gradually settled but the patient
remained dyspnoeic and radiographs continued to show total
atelectasis of the right lung.   The patient continued to

have attacks of breathlessness and she died on the 24th
post-operative day.   Permission for post-mortem examination
was refused.

In spite of the aspiration of mucopus this patient
died of unrelieved atelectasis.   It may be that repeated
aspiration as advocated by Bence (1947) may have led to a
more satisfactory result.

Case 9. - female, aged 31 years in poor general condition
with a large apical cavity and tuberculosis throughout the
left lung.   Severe post-operative reaction. Massive
atelectasis of the left lung was diagnosed on the second day
following resection of the upper four ribs without apicolysis
at first stage thoracoplasty.   The patient experienced
considerable difficulty in voiding sputum.   Bronchoscopic
aspiration was carried out on the 5th post-operative day
when some mucopurulent sputum was aspirated.   Slight
clinical and radiological improvement resulted.   Temperature,
pulse and respiratory rates remained elevated on the following
day and cough was still unproductive.   Considerable
improvement occurred on the 8th post-operative day when
temperature, pulse and respiratory rates became less and
expectoration became more productive.   The patient
continued to show gradual clinical and radiological
improvement and x-ray on the 19th day after thoracoplasty
showed complete resolution of the atelectasis.

It cannot be said that bronchoscopic aspiration was effective in this case. The atelectasis, as it frequently does in the absence of aspiration, disappeared in the course of time.

Case 18. - male, aged 31 years in fair general condition with a large apical cavity and tuberculosis throughout the left lung. Severe post-operative reaction. Massive atelectasis of the left lung was diagnosed on the day following first stage, three rib thoracoplasty with apicolysis. Bronchoscopic aspiration on the 4th post-operative day resulted in slight clinical and radiological improvement. Temperature, pulse and respiratory rates remained elevated but by the following week had gradually returned to normal. Radiography 28 days after broncho-scopic aspiration showed only partial re-aeration of the left lung.

Result of bronchoscopic aspiration - unsatisfactory.

Case 23. - female aged 23 in poor general condition with considerable apical cavitation and tuberculosis throughout the left lung. Tuberculous disease also present in the upper part of the right lung. Severe post-operative reaction. Massive atelectasis of the left lung was diagnosed on the day following first stage thoracoplasty with apicolysis and resection of the upper four ribs.

Bronchoscopic aspiration on the 4th post-operative day resulted in considerable evacuation of mucopus.   No radiological improvement followed.

Massive atelectasis persisted in this case throughout the course of thoracoplasty.

Case 50. - female, aged 35 in good general condition with tuberculosis and cavitation affecting the apex of the right lung.   The patient experienced a severe post-operative reaction and marked paradoxical respiration. Radiography on the day following first stage thoracoplasty with partial apicolysis and resection of the upper six ribs showed patchy atelectasis throughout the right lung.   The patient was able to cough up sputum fairly well but radio-graphy on the 6th post-operative day showed that massive atelectasis of the right lung had developed.   On the 7th day paradoxical respiration was still present and the patient was very ill but not cyanosed.   Bronchoscopic aspiration on the same day resulted in some clinical improvement but on the following day the patient died. Autopsy was not performed.   Death was considered to be due to unrelieved atelectasis, paradoxical respiration and exhaustion.

Paradoxical respiration was a prominent feature in this case probably due to the larger number of ribs resected in the presence of a mobile mediastinum.   Again no marked

relief of the atelectasis resulted from bronchoscopic
aspiration.

7). Subsequent course of the atelectasis.

The subsequent course of the cases of atelectasis
both immediate and remote is shown in Appendix IV.

(a) Immediate results.

It was only possible to achieve complete re-aeration
of the atelectatic lung in a proportion of the cases during
the interval between stages of thoracoplasty.   As it was
not desirable from a surgical point of view to delay a
subsequent stage unduly in the presence of non-resolution
of the atelectasis, operation was frequently performed
where the affected lung was air-bearing only partially or
not at all.   Tables V, VI, VII and VIII show the degree
of aeration that was effected after the various stages of
operation.   Complete re-aeration of a massive atelectasis
was more liable to occur after the initial stage than after
later stages of thoracoplasty.   The two patients, (Cases 2.
and 50.), who died as a result of unrelieved atelectasis
are included in Table V under the heading of "no re-aeration".
The incidence of non-resolution became progressively greater
as the operation proceeded.   Partial re-aeration of massive
atelectasis was encountered most often following the second
stage of thoracoplasty.   In all but one of the ten occasions

when scattered areas of atelectasis only appeared throughout
the involved lung, complete re-aeration resulted.

TABLE V.

Degree of re-aeration of atelectasis after

1st stage thoracoplasty.

Atelectasis.	No.of cases.	Complete re-aeration.	Partial re-aeration.	No re-aeration
Massive	23	14	5	4
Partial	5	4	1	Nil
Total:	28	18	6	4

TABLE VI.

Degree of re-aeration of atelectasis after

second-stage thoracoplasty.

Atelectasis.	No. of cases.	Complete re-aeration.	Partial re-aeration.	No re-aeration
Massive	27	10	10	7
Partial	5	5	Nil	Nil
Total:	32	15	10	7

TABLE VII./

TABLE VII.

Degree of re-aeration of atelectasis after

third-stage thoracoplasty.

Atelectasis.	No. of cases.	Complete re-aeration.	Partial re-aeration.	No re-aeration.
Massive	23	9	4	10
Partial	1	1	Nil	Nil
Total:	24	10	4	10

TABLE VIII.

Degree of re-aeration of atelectasis after

revisionary thoracoplasty.

Atelectasis.	No. of cases.	Complete re-aeration.	Partial re-aeration.	No re-aeration.
Massive	4	1	1	2
Partial	Nil	Nil	Nil	Nil
Total:	4	1	1	2

(b) Late results.

The late results have been classified according to the final state of aeration of the atelectatic lung. Follow-up of the 35 cases of atelectasis which survived operation produced the following:-

Bronchogram I   Case 39

Bronchogram II   Case 39

Early complete re-aeration	:	12 cases
Delayed complete re-aeration	:	3 cases
Incomplete re-aeration	:	7 cases
No re-aeration	:	9 cases
Death	:	2 cases
Untraced	:	2 cases
Total:		35 cases

i. Early complete re-aeration.

Twelve cases showed complete resolution of the
atelectasis within one month of completion of the operation.
Apart from the morbidity that the condition caused and a
degree of terminal bronchiectasis in some instances it
cannot be said that any permanent effects had accrued as a
consequence of post-operative atelectasis in these patients.
Evidence of the improved results that followed experience
with post-operative atelectasis is provided by this group
of cases.   The favourable outcome of early complete re-
aeration occurred in two only of the 15 instances of
atelectasis that complicated the first 30 thoracoplasties,
an incidence of 13.33 per cent.   The corresponding incidence
for the remaining cases was 45.45 per cent.

Case 39. presented more marked bronchiectasis than was
usual in the patients who had manifested early complete re-
aeration of the atelectasis.   Bronchograms I and II of this
patient taken two years and four months after nine rib,

Bronchogram III   Case 123

Bronchogram IV   Case 123

Bronchogram V   Case 28

Bronchogram VI   Case 28

three-stage thoracoplasty show crowding and kinking of the
outlined bronchi as a consequence of marked compression of
the left lung which resulted from extensive resection of
ribs.   There is a moderate degree of mixed cylindrical
and saccular bronchiectasis present.   The patient remains
well and her sputum is negative for the tubercle bacillus.
In contrast postero-anterior and lateral bronchograms III
and IV of Case 123. taken two years after six rib, two-
stage thoracoplasty show no significant degree of
bronchiectasis.   A more conservative thoracoplasty was
performed on this patient and atelectasis did not develop
during the course of the operation.

ii. Delayed complete re-aeration.

In 3 patients (Cases, 28, 55 and 60) re-aeration of
massive atelectasis was not complete until six, four and
three months respectively after the final stage of
thoracoplasty.   Clinical signs of bronchiectasis exist
in all three instances.   The postero-anterior and lateral
bronchograms V and VI of Case 28. reveal gross bronchiectatic
changes on the side of operation chiefly affecting the right
lower lobe.   Where the field is not obscured by the outlined
bronchi the right lung is seen to be completely aerated.

iii. Incomplete re-aeration.

Incomplete resolution of atelectasis was encountered in

Bronchogram VII   Case 21   Bronchogram VIII   Case 21

Bronchogram IX   Case 31   Bronchogram X   Case 31

7 patients, 5 of whom had varying degrees of permanent
atelectasis of the base of the thoracoplasty lung.   In
the other 2 cases permanent atelectasis was present in the
upper lobe of the operation side.   Again bronchiectatic
change in the affected lung was a feature of all the
patients in this group.   The postero-anterior and lateral
bronchograms VII and VIII of Case 21. taken three years and
eight months after completion of an eight rib, two-stage
thoracoplasty show gross bronchiectasis, mainly cylindrical
and in part saccular, of the lower and middle lobes of the
right lung.   The postero-anterior film (Bronchogram VII)
shows the upper part of the right lung to be air-bearing.
Bronchograms IX and X of Case 31., taken three years and
four months after the completion of a seven rib, three-
stage thoracoplasty show bronchiectatic change throughout
the left lung with marked crowding of the basic bronchi.
In this case the base of the left lung gradually re-aerated
but the left upper lobe remained the seat of permanent
atelectasis.

iv. No re-aeration.

In 9 patients there remained permanent massive
atelectasis on the side of operation and in every instance
marked signs of bronchiectasis were found in the affected
lung.   Case 10. provides an example of permanent total
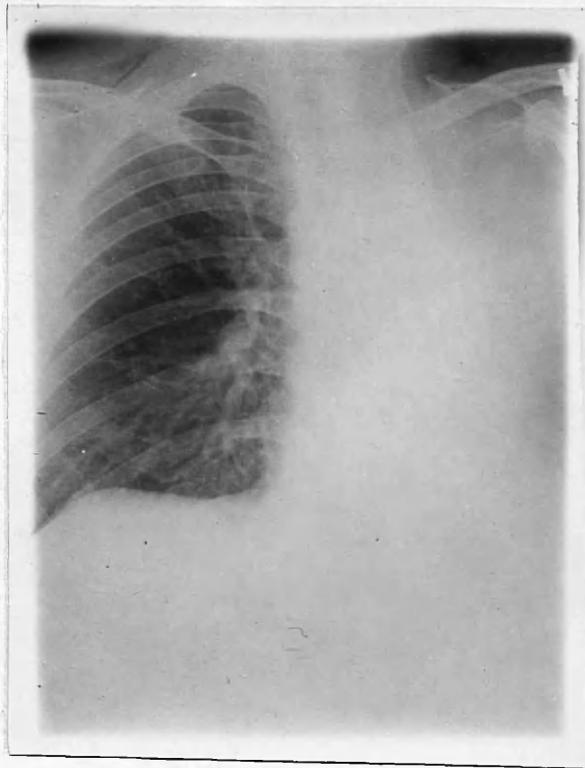
Radiograph XIV   Case 10

Bronchogram XI   Case 10

atelectasis affecting the left lung.   Radiograph XIV
of this patient, taken four years after completion of a
nine rib, three-stage thoracoplasty shows a dense homo-
geneous opacity throughout the left side of the chest
and cardiac retraction.   So dense is the opacity in
this case that it has proved difficult to get photographic
evidence of bronchiectasis.   Close inspection of the
lateral bronchogram XI, however reveals the presence of
gross bronchiectasis.   The patient remains well, he has
gained more than two stones in weight since his discharge
from hospital and the sputum he produces is persistently
negative for the tubercle bacillus.

In spite of permanent massive atelectasis some of
the patients in this group remain in fairly good general
condition and produce sputum which is persistently negative
for the tubercle bacillus.   The reason for this experience,
which is directly contrary to the considered opinion of
Thomas and Cleland (1942) who regard an atelectatic lung
which has not re-aerated within three weeks to be the seat
of an additional tuberculous process, eluded us for some
time.   It was not until the writer came across an account
by Erwin (1939) of massive atelectasis in pulmonary
tuberculosis that an explanation was suggested.   Erwin
reasons that a tuberculous focus in a lung which is the
seat of atelectasis is benefited by the local cessation of

respiration and the consequent complete rest afforded.
Cavitation within the lung will tend to disappear as a
result of absorption of its contained air.   Should the
bronchus draining the cavity remain patent, however,
persistent cavitation in the midst of the atelectasis
will occur.   Further the increased intrapulmonary
tension resulting from atelectasis will have a considerable
distending influence when exerted on the walls of the
cavity.   Radiograph XV of Case 23., taken one year and
four months after revisionary thoracoplasty, is considered
to show a typical example of persistent cavitation in the
midst of massive atelectasis.   It shows massive
atelectasis of the left lung with a large cavity at the
level of the fifth and sixth transverse processes.   This
large cavity remains in spite of the excellent collapse of
the chest wall that has resulted from resection of the
anterior ends of the third, fourth and fifth ribs at
revisionary thoracoplasty.   Bronchograms XII and XIII of
the same patient show the presence of gross bronchiectasis
in the left lung.   She was too ill to permit of an attempt
to instil lipoidal into the persistent cavity but
bronchogram XII shows that a small quantity of lipoidal
actually found its way into the most dependent part of the
cavity.   When last seen this patient was in very poor
general condition and producing sputum which was positive

for the tubercle bacillus.

It was not possible to trace two patients, Cases 69.
and 70.   Case 69. was transferred to Law Hospital,
Lanarkshire with unresolved massive atelectasis three
weeks after completion of thoracoplasty at a time when
there was an acute shortage of bed accommodation in the
Thoracic Unit, Mearnskirk.   Reference to Law Hospital
elicited the information that the result of operation had
been unsatisfactory, that the patient was subject to
attacks of breathlessness and cyanosis and that she had
been discharged from hospital at her parents request seven
months after admission.   The doctor who had attended the
patient during this period was of the opinion that she
would not live long.

Case 70. was irregularly dismissed from hospital four
weeks after completion of thoracoplasty.   The most recent
chest radiograph available had been taken on the 14th post-
operative day and showed massive atelectasis on the side of
operation.   A letter from her mother two years later
recorded that the patient was well, was married and had given
birth to a healthy baby.

There were two late deaths in the series.   Case 18.,
a victim of massive unresolved atelectasis with persistent
apical cavitation had a fatal haemoptysis one year after
dismissal from hospital.   The persistent cavitation or

bronchiectasis may have been the cause of haemoptysis in
this case. Case 90., in whom there was also persistent
apical cavitation, developed bronchiectasis as a
consequence of persistent basal atelectasis. She was
admitted to the Brompton Hospital, London, one year and
three months after completion of thoracoplasty where she
died following pneumonectomy.

8). Bronchiectasis following thoracoplasty.

The relationship between bronchiectasis and atelectasis
has been described by Jennings (1937), Andrus (1937), Lander
and Davidson (1938b), Lander (1946), Lees (1950) and others.
The high incidence of bronchiectasis following delayed or
non-resolution of post-operative atelectasis in the series
under review is but another example of this relationship.
Erwin (1939) and Sellors (1947a) have commented on the
frequency of bronchiectasis following thoracoplasty and
they associate its occurrence with the previous development
of post-operative atelectasis. It is doubtful however if
it is generally appreciated that bronchiectasis following
thoracoplasty may be of a non-tuberculous nature. It is
more common for the persistence of sputum and occasional
haemoptyses in these cases to be erroneously diagnosed as
persistence of active tuberculous disease. Cases 10. and
28. provide contrasting illustrations of advanced non-
tuberculous bronchiectasis. In the former it has been seen
that gross bronchiectatic change is present in the midst of

permanent total atelectasis (see radiograph XIV and
bronchogram XI); the latter, in whom re-aeration of massive
post-operative atelectasis did not occur until six months
after completion of thoracoplasty, shows gross bronchiectasis
in the midst of aerated lung tissue (see bronchograms V and
VI).   Both patients remain surprisingly well and sputum
tests have been persistently negative for the tubercle
bacillus.   It is important in such cases to reassure the
patient that the persistence of sputum is not due to active
tuberculosis.

Conclusions.

Atelectasis following thoracoplasty in pulmonary
tuberculosis is an unfavourable development.   It gives rise
to considerable post-operative morbidity; it may cause
delay between stages of operation; it was a direct or indirect
cause of death in three of the cases under review.

Post-operative atelectasis occurs most frequently after
the initial stage of thoracoplasty.   When it appears for
the first time it is usually associated with more severe
symptoms.   .

Once established, post-operative atelectasis either
persists or recurs following any subsequent stage of
thoracoplasty.

Difficulties in diagnosis may be overcome by the
observation of serial radiographs taken at frequent
intervals.

The effects of post-operative atelectasis may be
reduced where there is adequate pre-operative preparation
and post-operative care of the patients submitted to
thoracoplasty.   Bronchoscopic aspiration of sputum from
the bronchial tree would not appear to be an effective
method of treatment in the established condition.

Prognosis is more favourable in (a) the partial
or patchy variety than in the massive type of post-operative
atelectasis and (b) in the cases where early complete
re-aeration of the lung is effected.   Prognosis is least
favourable where the pulmonary stresses engendered within
the lung as a consequence of atelectasis result in
persistent cavitation.

Bronchiectasis is a late development especially in
the cases where delayed, incomplete, or no resolution of
the atelectasis has occurred.   It is important to bear
in mind that such bronchiectasis may be non-tuberculous in
nature.

* * * * * * *

Chapter III.

An assessment of predisposition to
atelectasis following thoracoplasty.

The initial objective of this chapter is to analyse the
incidence of atelectasis which complicated 145 thoracoplasty
operations (Cases 31-175) in respect of certain factors of
possible predisposing significance.   The first 30 cases
of thoracoplasty have not been included in the analysis
lest the high incidence of post-operative atelectasis already
noted in this group should unduly influence the final
conclusions.   It is fortunate that the 145 cases under
review are comprised of approximately equal numbers of males
and females, that post-operative atelectasis occurred
regularly throughout the series and that tuberculosis
affected either lung in comparable numbers.   The figures
presented have been analysed statistically according to the
x^2 or chi-square test as described by Hill (1945) and all
calculations have been made by the writer.   Two methods of
analysis have been employed: (a) the "expected and observed
values" and (b) the "fourfold table" with the Yates'
correction for small numbers.   As a conventional level a P,
the probability, of 0.05 or less is taken to be significant.

I.   Selection of probable predisposing factors.

The published works of others have afforded some
guidance in the selection of factors likely to predispose

to atelectasis following thoracoplasty.   Semb (1937)
has shown that the incidence of post-operative atelectasis
increased with the number of ribs resected at a single stage
of thoracoplasty.   Erwin (1939) and Mitchell (1949) report
that a pre-existing phrenic nerve paralysis has a similar
effect.   Investigations of atelectasis following abdominal
surgery by King (1933), Brock (1936), Mimpriss and
Etheridge (1944) and others indicate that the complication
occurs most frequently after upper abdominal operations on
male subjects in poor general condition and suffering from
preoperative respiratory infection of an acute or chronic
nature.   Morton (1944) has added addiction to cigarette
smoking to the list of predisposing factors and Dripps and
Deeming (1946) have found an increased incidence of
atelectasis after emergency operations within the abdomen.
Varying opinions exist in respect of age and seasonal
frequency in post-operative atelectasis.

In the series under review the factors of cigarette
smoking, acute respiratory infection and emergency operation
can be discounted.   All the patients suffered from pulmonary
tuberculosis with cough and sputum and the operation differed
only in regard to the number of ribs resected and the extent
of apicolysis at first stage thoracoplasty.   The following
variable factors were selected as worthy of investigation
from the point of view of predisposition to atelectasis
following thoracoplasty:-

Sex.
Age.
Operative Risk.
Side and extent of pulmonary tuberculosis.
Size of cavity.
The operation of phrenic crush.
Preoperative vital capacity.
Number of ribs resected at first stage thoracoplasty.
Extent of apicolysis at first stage thoracoplasty.
Anaesthesia for first stage thoracoplasty.
Season of the year.

The analysis in respect of vital capacity, number of ribs resected, extent of apicolysis and anaesthesia is restricted to the 16 instances of atelectasis which complicated first stage thoracoplasty in 145 cases. The remaining factors apply to the completed operation and have been analysed in respect of the total incidence of atelectasis. It is not claimed that the above is an exhaustive list of the variants encountered before and during thoracoplasty. The study for instance does not include observations on the frequency of endobronchial tuberculosis and its relationship to post-operative atelectasis. Such an investigation would have necessitated routine bronchoscopic examination of the bronchial tree, a procedure which was considered to be neither practical nor desirable. Furthermore, although positive proof of the existence of endobronchial tuberculosis may only be provided by bronchoscopy, the method has its limitations and it is doubtful whether more than a porportion of the cases may be diagnosed in this way. It will be appreciated that the factors selected may be easily and quickly evaluated and therefore readily applicable as a

routine procedure in assessing patients for operation.
If, as a result of the investigation, any factor not
hitherto emphasised is found to play a significant role
in the development of post-operative atelectasis a useful
purpose will have been served.

II. Record of Results.

TABLE I.

Post-operative atelectasis in relation to age.

Age in years.	No. of cases.	Atelectasis.	Atelectasis per cent
15-20	37	5	13.51
21-25	47	10	21.28
26-30	23	3	13.04
31-35	18	3	16.67
36-46	20	1	5.00
Total:	145	22	15.17

Statistical analysis: $x^2 = 4.365$

$n = 4.$

P is slightly less than 0.30.

It will be seen from an analysis of the figures presented
in Table I.that the age of the patient has exerted no
significant influence in the development of atelectasis
following thoracoplasty.

TABLE II.

Post-operative atelectasis in relation to sex.

Sex	No. of cases.	Atelectasis.	Atelectasis per cent.
Males	69	5	7.25
Females	76	17	22.37
Total:	145	22	15.17

Statistical analysis: $x^2 = 5.30$

$n = 1$

P is slightly less than 0.02

The 145 cases of thoracoplasty under analysis comprised 69 males and 76 females. Table II. shows that the incidence per cent of atelectasis was three times greater in females than in males, a difference of definite significance. No figures of the relationship of sex to atelectasis following thoracoplasty have been found in the literature consulted but many workers (King, Rink, Morton, Dripps and Deeming to list but a few) have recorded a significantly greater incidence of atelectasis in males after abdominal surgery. The theories advanced to account for this latter relationship of sex to atelectasis have been listed by Morton (1944) and Dripps et al. (1946) and are as follows:-

(a) Women have a higher resistance to infection of the respiratory tract than males and are therefore less liable to develop post-operative atelectasis.

(b) Women tolerate pain better than men and consequently
are more liable to maintain ventilation of the lung
bases in the presence of pain from an abdominal wound.
Men are more apt to splint their respiratory muscles
because of discomfort.

(c) There is a greater incidence of cigarette smoking
among males.

(d) The operations of herniotomy and partial gastrectomy
are much commoner in males and these operations are
complicated by a high incidence of post-operative
atelectasis.

(e) Men make greater use of diaphragmatic breathing and
their normal respiration is thus interfered with to
a larger extent after abdominal operation.

This last is the most popularly held theory to account
for the larger incidence of atelectasis following abdominal
surgery in males than in females.   It may be argued that
if males are at a decided disadvantage after abdominal
surgery because of their greater reliance on diaphragmatic
breathing, then females with their relatively more
pronounced costal respiration should suffer the more marked
disability after surgery of the chest wall.   Thomas and
Cleland point out that costal respiration is severely
disturbed or even non-existant for several days after
thoracoplasty.   The significantly high incidence of post-
operative atelectasis in the present study would seem to

lend support to the contention that the degree of
diminution of pulmonary ventilation after surgery has a
bearing on the development of atelectasis.   It will be
seen later that there is a more marked reduction of vital
capacity in males than in females following first stage
thoracoplasty.   The difference, however, is not
statistically significant.

TABLE III.

Post-operative atelectasis in relation to operative risk.

Risk	No. of cases.	Atelectasis.	Atelectasis per cent.
1	69	7	10.14
2	49	2	4.08
3	27	13	48.15
Total:	145	22	15.17

Statistical analysis: $x^2 = 28.81$

$n = 2.$

P is considerably less than 0.01.

As already described the operative risk or the fitness
of the patient for operation was assessed according to the
stability of the pulmonary disease and the general condition
of the patient.   Three types of patient were recognised:
Risk I. refers to the stable chronic in good general
condition; Risk II. refers to the relapsing chronic in fair
general condition; Risk III. refers to the slipping chronic

in poor general condition.   Too much reliance cannot be
placed on the figures presented in Table III. Classification
was often difficult and there were many border-line cases.
As might be expected Table III. shows a significantly large
incidence of atelectasis complicating thoracoplasty in poor
risk cases.   The patients in this group as already described
were less liable to benefit from the preoperative and post-
operative measures directed at reducing the complication of
atelectasis:

TABLE IV.

Post-operative atelectasis in relation to
side and extent of pulmonary tuberculosis

Side and extent of disease.	No. of cases.	Atelectasis.	Atelectasis per cent.
RA	10	2	20.00
R1	37	1	2.70
R1,2	22	2	9.09
R1,2.3.	10	1	10.00
LA	7	Nil	-
L1	37	6	16.22
L1,2	22	10	45.45
Total:	145	22	15.17

The side and extent of the pulmonary tuberculosis
was determined from a study of the most recent preoperative
radiograph of the chest - in all instances a radiograph

taken just prior to thoracoplasty was available. For the purposes of tabulation four areas of the right lung and three areas of the left lung were delineated. RA denotes tuberculosis affecting the apex of the right lung; Rl, R2 and R3 denote tuberculous disease in the region of the upper, middle and lower thirds respectively of the right lung. LA denotes tuberculosis in the apex of the left lung; Ll and L2 denote tuberculosis affecting the upper and lower halves respectively of the left lung. The side of pulmonary involvement corresponds to the side of operation in every case.

Table IV. shows the high incidence of 45.45 per cent of atelectasis to have complicated 22 thoracoplasty operations performed for tuberculous disease throughout the whole of the left lung. If we take the remaining 123 cases it is found that the corresponding incidence of atelectasis equals 9.76 per cent. When this difference is analysed statistically it gives x^2 a value of 15.84 and P is considerably less than 0.01, indicating pronounced significance.

TABLE V. /

TABLE V.

Post-operative atelectasis in relation to side of operation.

Side of operation.	No. of cases.	Atelectasis.	Atelectasis per cent.
Right	79	6	7.59
Left	66	16	24.24
Total:	145	22	15.17

Statistical analysis: $x^2 = 6.50$

$n = 1.$

$P = 0.01.$

Table V. shows that 6 instances of atelectasis followed 79 operations on the right side of the chest, an incidence of 7.59 per cent; the corresponding incidence of atelectasis for 66 thoracoplasty operations for tuberculous disease affecting the left lung was 16 or 24.24 per cent. This difference is statistically significant. If however the 22 cases tabulated as Ll,2 in Table IV. be excluded from the analysis there is no significant increase of atelectasis affecting the left lung. ($x^2 = 0.005$; $n = 1$ and $P = 0.95$). It would seem justifiable therefore to infer that the incidence of atelectasis is significantly high following thoracoplasty for tuberculous disease throughout the left lung but not so when a lesser extent of the lung is involved.

Again no comparable figures from the literature are available but it is of interest to note that Gower (1941)

and Gray (1946) report that atelectasis occurs more frequently
on the left side following pulmonary lobectomy.   Gower
suggests an anatomical explanation, secretions being more
liable to retention in the left main bronchus which is longer,
narrower and runs more obliquely than the right.

TABLE VI.

Post-operative atelectasis in relation to bilateral
tuberculosis.

Disease.	No. of cases.	Atelectasis.	Atelectasis per cent.
Bilateral	39	4	10.26
Unilateral	106	18	16.98
Total:	145	22	15.17

Statistical analysis: x^2 = 0.004

n = 1.

P = 0.95.

It will be seen from Table VI. that in 145 cases of
thoracoplasty the occurrence of post-operative atelectasis
was not influenced by the presence of bilateral
tuberculosis.

TABLE VII./

TABLE VII.

Post-operative atelectasis in relation to size of cavity

Size of Cavity.	No. of Cases.	Atelectasis.	Atelectasis per cent.
1.	25	2	8.00
2.	55	6	10.91
3.	65	14	21.54
Total:	145	22	15.17

Statistical analysis: $x^2 = 3.83$

n = 2.

P is slightly less than 0.20.

The size of the cavity was measured on the preoperative radiograph.   Size 1. indicates a cavity of less than 2 centimetres in diameter; Size 2. indicates a cavity of more than 2 but less than 4 centimetres in diameter; Size 3. indicates a cavity of more than 4 centimetres in diameter or an extensive system of small cavities.

It will be seen from Table VII. that in the series under review the size of cavity has exerted no significant influence on the development of atelectasis following thoracoplasty.   Unfortunately there are no figures to correlate the amount of sputum expectorated in relation to the extent of cavitation.   If it may be assumed that patients with large cavities tend to produce a greater quantity of sputum it is of interest to note that Gower (1941)

found the amount of sputum expectorated in bronchiectasis
bore no relation to the incidence of atelectasis after
pulmonary lobectomy.

TABLE VIII.

Post-operative atelectasis in relation to
the operation of phrenic crush.

	No. of Cases.	Atelectasis.	Atelectasis per cent.
Cases with phrenic crush	22	5	22.73
Cases without phrenic crush	123	17	13.82
Total:	145	22	15.17

Statistical analysis: $x^2 = .56$

$n = 1.$

P is slightly less than 0.50.

Reference has already been made to the operation of
phrenic crush performed to relax the tuberculous lung in
the hope of effecting cavity closure.   It is not
sufficient to take the presence of an operation scar in
the neck as evidence of paralysis of the related hemi-
diaphragm for two reasons: (a) the operation may have
been unsuccessful; (b) sufficient time may have elapsed
since the operation to permit of regeneration of the
crushed phrenic nerve and restoration of diaphragmatic
function.   When the hemi-diaphragm is paralysed it

becomes more or less fixed in the position of quiet
expiration and radiography taken at full inspiration
reveals the paralysed leaf of the diaphragm occupying a
high position in the chest.   The 22 cases with paralysis
of the hemi-diaphragm in this study were diagnosed in the
presence of such a high position of the diaphragm following
upon a fairly recent phrenic crush operation.

Table VIII shows that paralysis of the hemi-diaphragm
played no significant role in the production of post-
operative atelectasis.   This finding would appear to be
directly contrary to expectation in view of the fact that
considerable hypoventilation of the related lung must ensue
after thoracoplasty from deficient function of the homo-
lateral diaphragm.   Mitchell (1949) reports a significantly
increased incidence of atelectasis in the homolateral lung
after thoracoplasty in the presence of paralysis of the
hemi-diaphragm.   His figures, however, are not presented
in sufficient detail for analysis.   Various opinions exist
as to the efficiency of expectoration after phrenic crush.
Wall (1928) believes that the effect of cough is enhanced
and recommends the operation in the treatment of basal
bronchiectasis.   Ballon (1939) and Mitchell consider that
paralysis of the hemi-diaphragm results in impairment of
expectoration.   The present investigation can throw no
light on the subject.   The 22 cases of phrenic crush
encountered were fairly evenly distributed throughout the

series except in relation to side of operation.   Eight
of the cases were submitted to left sided thoracoplasty
and 14 had an operation on the right side.   The series,
however, is too small for further analysis.

TABLE IX.

Post-operative atelectasis after first stage
thoracoplasty in relation to preoperative
vital capacity.

Average pre-operative vital capacity in c.c's.	No. of cases.	Atelectasis	Atelectasis per cent.
500 - 1499	31	5	16.13
1500 - 1999	45	7	15.56
2000 - 2499	38	3	7.89
2500 - 4500	26	Nil	–
Total:	140	15	10.71

Statistical analysis: $x^2 = 5.48$.

$n = 3$.

P is slightly less than 0.20.

Vital capacity records were available in 140 of the
cases of thoracoplasty under review.   The apparatus used
for this purpose was the Oliver Pell pneumanometer and the
average preoperative vital capacity in cubic centimetres is
shown in Table IX. along with the incidence of atelectasis
that complicated first stage thoracoplasty in these cases.

Although patients with a low vital capacity tended to develop
atelectasis more frequently the difference is not statistically
significant. (See Table IX.). It is of interest to note
that of the two patients who recorded a vital capacity of
over 3500 cubic centimetres, one was employed as a trumpeter
in a dance band while the other played the bagpipes.

Vital capacity readings are apt to be misleading. Some
patients are afraid to blow hard lest the effect be
detrimental to their pulmonary lesion while others never
acquire the trick of applying a steady prolonged expiration
through the mouthpiece of the apparatus. Opportunity
however has been taken to compare the average percentage
reduction in vital capacity which followed first stage
thoracoplasty in a comparable group of male and female
patients. For this purpose the records compared were:
(a) the average preoperative vital capacity and
(b) the vital capacity recorded on the day immediately
following first stage thoracoplasty. Only those patients
were selected who had a preoperative vital capacity of
between 1,000 and 3,000 cubic centimetres, in whom three
ribs only were resected at first stage thoracoplasty and
in whom a partial or extensive apicolysis was performed.
No patient with post-operative atelectasis or effusion,
with spread of tuberculous disease, torn pleura, pneumo-
peritoneum, artificial pneumothorax etc. was included.
Thus as near a comparable series of cases with the figures

available was achieved. The results of this investigation are shown in Table X.

TABLE X.

Average reduction in vital capacity per cent in males and females following first stage thoracoplasty.

Sex.	No. of cases.	Average reduction in vital capacity per cent following first stage thoracoplasty.
Male	29	31.2
Female	32	40.4

Statistical analysis: The difference between the percentage reduction in vital capacity for males and females as shown in Table X., is 9.2 and 12.1 is the standard error of this difference. Thus, although the average reduction in vital capacity per cent following first stage thoracoplasty is greater in females than in males the difference is not statistically significant.

TABLE XI.

Atelectasis in relation to the number of ribs resected at first stage thoracoplasty.

No. of ribs resected.	No. of cases.	Atelectasis.	Atelectasis per cent.
3	127	11	8.66
4	12	2	16.67
5	4	1	25.00
6	2	2	100.
Total:	145	16	11.03

TABLE XI(a).

No. of ribs resected.	No. of cases.	Atelectasis.	Atelectasis per cent.
3	127	11	8.66
4 and over	18	5	27.78
Total:	145	16	11.03

Statistical analysis: $x^2 = 4.08$

$n = 1$

P is slightly less than 0.05.

On account of the small numbers involved when more than three ribs were resected at first stage thoracoplasty the cases concerned have been grouped together in Table XI(a) for the purposes of analysis. It will be observed that a significantly increased incidence of post-operative atelectasis occurred when four or more ribs were resected. This finding is in accord with the experience of Semb (1937).

TABLE XII.

Post-operative atelectasis in relation to

the extent of apicolysis at first stage thoracoplasty.

Extent of Apicolysis.	No. of cases.	Atelectasis.	Atelectasis per cent.
Extensive	87	10	11.49
Partial	40	6	15.00
None	18	Nil	-
Total:	145	16	11.03

Statistical analysis: $x^2 = 2.892$.

$n = 2$.

P is slightly more than 0.20.

It will be seen from an analysis of the figures presented in Table XII. that the extent of apicolysis did not exert a significant influence on the development of atelectasis after first stage thoracoplasty. Thomas and Cleland (1942) report a similar experience.

TABLE XIII.

Atelectasis after first stage thoracoplasty in relation to the technique of general anaesthesia.

Anaesthesia	No. of Cases.	Atelectasis.	Atelectasis per cent.
Semi-open	30	5	16.67
Closed	115	11	9.57
Total:	145	16	11.03

Statistical analysis: $x^2 = 1.51$

$n = 1$

P is slightly greater than 0.20.

The techniques of semi-open and closed circuit anaesthesia have been described in Part II, Chapter I. Table XIII. reveals that the incidence of post-operative atelectasis after first stage thoracoplasty showed no significant change when either technique was used.

TABLE XIV.

Post-operative atelectasis in relation to the

season of the year.

Season.	No.of cases.	Atelectasis.	Atelectasis per cent.
Oct. - Mch.	74	12	16.22
Apr. - Sep.	71	10	14.08
Total:	145	22	15.17

To compare the seasonal incidence of atelectasis following thoracoplasty the year has been divided into two six-monthly periods, namely October to March and April to September, corresponding respectively to the periods of prevalence and non-prevalence of acute respiratory infection. Several writers (e.g. Binning, Sircar and Boston, Stringer, Harris) report an increased incidence of pulmonary complications following abdominal surgery during the winter months. Table XIV. shows that approximately equal numbers of thoracoplasty operations were performed during the periods selected. As might be expected a similar relationship exists between the respective incidence of post-operative atelectasis. Thoracoplasty, being an operation of election, is not undertaken in the presence of acute respiratory infection and coryza is less prevalent in sanatoria than in general hospitals.

*   *   *   *   *   *

It will be convenient at this juncture to enquire if the introduction of routine change of posture and streptomycin therapy have exerted any influence in the occurrence of post-operative atelectasis in the cases under review.

TABLE XV.

Post-operative atelectasis in relation to treatment with routine change of posture after thoracoplasty.

Posture.	No. of cases.	Atelectasis.	Atelectasis per cent.
Change	59	8	13.56
No change	86	14	16.28
Total:	145	22	15.17

Statistical analysis: $x^2 = 0.05$

$n = 1$

P is slightly less than .80

As already described routine change of posture after operation was introduced during the course of the study with the object of stimulating an increased expectoration of sputum.   Although other benefits accrued Table XV shows that no significant effect was exerted on the incidence of post-operative atelectasis.

TABLE XVI.

Post-operative atelectasis in relation to treatment

with streptomycin during the pre and post-operative periods.

Streptomycin.	No. of Cases.	Atelectasis.	Atelectasis per cent.
Treated	33	4	12.12
Not Treated	112	18	16.07
Total:	145	22	15.17

Statistical analysis:　　$x^2 = .08$

$n = 1$

P is slightly more than .08

It will be seen from Table XVI that the introduction of streptomycin therapy has not materially altered the incidence of atelectasis after thoracoplasty.

1. Results of the analysis of predisposition to post-operative atelectasis in 145 cases of thoracoplasty.

The factors which have been shown to be of importance in predispostion to atelectasis following thoracoplasty are as follows:-

Sex.

Operative risk.

Tuberculosis throughout the left lung.

Number of ribs resected at first stage thoracoplasty.

The factors which did not exert a significant influence in the occurrence of atelectasis following thoracoplasty are listed below.

Age.

Size of cavity.

The operation of phrenic crush.

Preoperative vital capacity.

Extent of apicolysis at first stage thoracoplasty.

Anaesthesia for first stage thoracoplasty.

Season of the year.

2. Comparison of the first 30 and subsequent 145 cases of thoracoplasty.

The exclusion of the first 30 cases of thoracoplasty in the study from the analysis just completed was designed to eliminate the effects that the large incidence of atelectasis which complicated those cases might have in assessing predisposition to atelectasis.   Having determined the factors which are of predisposing significance in the subsequent 145 cases of thoracoplasty it will be of interest to compare both series in respect of the factors originally selected for analysis and the respective incidence of atelectasis that occurred.   If it is found that the factors which influenced the development of atelectasis in the larger series appear to have played a similar role in the smaller series, further weight will be added to our conclusions in

regard to predisposition to atelectasis. Comparison in
the manner indicated above has been made in Appendix V.
For the purposes of tabulation and convenience of
description the first 30 cases of thoracoplasty are called
Series A and the subsequent 145 cases are called Series B.
Tables a to f (see Appendix V) refer respectively to the
incidence of atelectasis that occurred in Series A and B
in relation to age, size of cavity, phrenic crush,
apicolysis, anaesthesia and season of the year, i.e. the
"non-significant factors" of Series B. (Records of vital
capacity were available in a small proportion only of the
cases that comprise Series A and this factor has been
omitted). Tables g to j refer respectively to the
incidence of atelectasis that occurred in Series A and B
in relation to sex, operative risk, side and extent of
pulmonary tuberculosis and number of ribs resected at first
stage thoracoplasty, i.e. the "significant factors" of
Series B.

A study of tables a to j shows that there is a definite
similarity between the respective incidence of post-operative
atelectasis in Series A and B in relation to the various
factors originally chosen for analysis. On account of
the number of factors involved Series A is too small for
separate analysis but one may at this stage cautiously
infer that the factors which predisposed to post-operative

atelectasis in Series B appear also to have exerted a
similar influence in Series A.

Our next task is to determine whether the relatively
large incidence of 50 per cent atelectasis which complicated
the first 30 cases of thoracoplasty was due to the
relatively greater number of predisposing factors displayed
by these cases or whether it was due, as originally thought,
to the inadequate preoperative preparation and post-
operative care which operated at the outset of the study for
reasons already described.

TABLE XVII.

Comparison of "predisposing factors" per cent in

Series A & B.

Factor	Series A.	Series B.
Female sex	63.3 per cent.	52.4 per cent.
Operative risk 3	36.7 per cent.	18.6 per cent.
L1, 2 disease	33.3 per cent.	15.2 per cent.
4 + ribs resected	56.7 per cent.	12.4 per cent.

Table XVII. shows that the factors already determined as
having a predisposing influence in the production of post-
operative atelectasis were represented in decidedly greater
proportion in the first 30 as compared with the subsequent
145 cases of thoracoplasty.

At first sight it would appear that the analysis can be carried no further but a method of comparison of "like with like" has been evolved which is particularly applicable to the present study.   The method devised is as follows:-

The number of ribs resected at first stage thoracoplasty apart, 29 of the first 30 cases have exact counterparts among the subsequent 145 cases in respect of the important factors of sex, operative risk and side and extent of pulmonary tuberculosis.   Reference to Appendix II will show that Case 1. is tabulated as male, risk 1 and RA (tuberculosis affecting the right apex).   In the subsequent 145 cases the first patient to show similar characteristics is Case 47.   Similarly the remaining cases in Series A, with the exception of Case 22., can be paired with an exact counterpart in respect of the factors mentioned.   The first case in Series B to show the desired similarity has been chosen and in this way "selection" or bias has been avoided.   It will be observed that Case 13. has similar characteristics to Case 1.   In an example of this kind the next comparable case in Series B has been chosen for comparison and Case 13. has therefore been paired with Case 65.   The result of this comparison of cases appears in Table XVIII.

TABLE XVIII.

Comparison of the incidence of atelectasis in Series A
with a comparable series of cases.

	No. of Cases.	Atelectasis.	Atelectasis per cent.
Series A.	29	14	48.28
Comparable series.	29	8	27.59
Total:	58	22	37.93

Statistical analysis: $x^2 = 1.83$

$n = 1$

P is slightly more than 0.20.

As Table XVIII. shows 14 instances or 48.28 per cent of
atelectasis complicated 29 cases in Series A as compared
with 8 instances of atelectasis or 27.59 per cent in a
comparable series of cases. The difference, however, is
not statistically significant. When it is noted that in
Series A, 17 patients had four or more ribs resected at
first stage thoracoplasty while 8 only of the patients in
the comparable series had this characteristic, the conclusion
that the larger incidence of atelectasis which occurred in
the first 30 cases of thoracoplasty might easily have been
attributed to chance is unavoidable. It is felt, however,
that the lack of statistical significance may be due to the
small numbers involved in the analysis.

3. Statistical analysis of the total series of 175 cases
   of thoracoplasty.

As a check on the accuracy of our conclusions so far
reached a statistical analysis of the total 175 cases of
thoracoplasty in the study similar to that already described
for 145 cases was conducted.   The results are shown in
Table XIX.

TABLE XIX.

Analysis of the incidence of atelectasis in 175 cases of
thoracoplasty.

Factor.	P.	Observation.
Sex	less than 0.01	Significant.
Age	0.20	Not significant.
Operative risk	less than 0.01	Significant.
Ll,2 disease	less than 0.01	Significant.
Size of cavity	less than 0.01	Significant.
Phrenic crush	more than 0.10	Not significant.
4 + ribs resected	less than 0.01	Significant.
Apicolysis	0.98	Not significant.
Anaesthesia	less than 0.70	Not significant.
Season of the year	0.70	Not significant.

It will be seen from the above table that no alteration
in regard to significance has resulted from analysis of the
total number of cases observed except in regard to the factor

of "size of cavity". This difference that has emerged
may be explained by the disproportionately large number of
patients in the first 30 cases of thoracoplasty in whom a
large cavity appeared in association with a poor operative
risk suffering from tuberculosis throughout the left lung.
Atelectasis complicated thoracoplasty in 9 out of 10 such
cases.

III. Interpretation of significant values.

Statistical methods of analysis, as Hill is careful
to point out, involves weighing probabilities and does not
amount to proof in the logical sense. Tests of significance
can give no information as to the origin of the differences
found beyond saying that chance is an unlikely explanation.
There remains for us to consider the final conclusions to
be drawn from the analysis of the incidence of post-operative
atelectasis in the patients who formed the material for the
present study. As already described a method has been
devised for comparing two groups of patients equivalent in
all relevant respects except in their differentiation by
one particular factor. The method labours under the
disadvantage that the numbers of comparable patients are
apt to be small with the result that statistical methods
tend to belittle the importance of differences found. On
the other hand it obviates the possibility of fallacious
conclusions. It is now proposed to examine by the method

described the factors of, (1) number of ribs resected at first stage thoracoplasty, (2) sex, and (3) tuberculous disease throughout the left lung, in the order stated.

(1) Post-operative atelectasis in relation to the number of ribs resected at first stage thoracoplasty.

In the series of 175 cases four or more ribs were resected at first stage thoracoplasty in 35 instances. Thirty-three of these patients can be compared in respect of sex, operative risk and side and extent of tuberculous involvement with 33 patients in whom three ribs only were resected at the same stage of thoracoplasty.   The result of such comparison appears in Table XX.

TABLE XX.

Post-operative atelectasis in two comparable groups of patients in relation to the number of ribs resected at first stage thoracoplasty.

Ribs resected.	No. of cases.	Atelectasis.	Atelectasis per cent.
3	33	6	18.18
4 +	33	12	36.36
Total:	66	18	27.27

Statistical analysis: $x^2 = 1.91$

n = 1

P is more than 0.20.

Although, as Table XX. shows, twice as many instances of atelectasis followed resection of four or more ribs at first stage thoracoplasty the difference is not statistically significant for two reasons: (a) the numbers concerned are small; (b) 5 out of 6 patients who developed atelectasis following resection of three ribs only at first stage thoracoplasty suffered from tuberculous disease throughout the left lung and were therefore especially liable to develop atelectasis as will be seen later. We can, however, conclude that although there is reason to believe from analysis of the cases in this study that an increased incidence of atelectasis follows resection of four or more ribs at first stage thoracoplasty it is necessary to observe a larger series of comparable cases before a definite conclusion may be reached.

(2) <u>Post-operative atelectasis in relation to sex of</u>
    <u>the patient.</u>

For the purpose of determining the influence of sex in the occurrence of atelectasis after thoracoplasty the following procedure was carried out. All the females in whom three ribs only were resected at first stage thoracoplasty were selected, 74 patients in all. Exactly equivalent males in respect of tuberculous disease and operative risk were found in 43 instances. Again selection was avoided by pairing the patients from the beginning of

the study as already explained.   Comparison of the incidence of atelectasis in these cases is shown in Table XXI.

TABLE XXI.

Post-operative atelectasis in two comparable groups of male and female patients.

Sex.	No. of Cases.	Atelectasis.	Atelectasis per cent.
Male	43	8	18.60
Female	43	6	13.95
Total:	86	14	16.28

It will be observed that in 43 comparable male and female patients the incidence of post-operative atelectasis varied but little, as shown in Table XXI.   This finding was unexpected and several test checks from the figures available were made, all with similar results.   We can therefore conclude that sex per se did not influence the occurrence of post-operative atelectasis in the cases under review. Another explanation must be sought for the significantly large incidence of the complication in the female patients.   It may be noted in passing that if care had not been exercised in seeking comparable groups of cases, the apparent increase in the incidence of post-operative atelectasis in female patients might easily have been explained by their increased dependence on costal respiration.

(3) <u>Post-operative atelectasis in relation to tuberculous</u>
    <u>disease throughout the left lung.</u>

There were 32 cases in whom the disease was classified
as Ll,2., i.e. tuberculosis throughout the whole of the left
lung, and 19 of these patients developed post-operative
atelectasis.   Twenty-seven such cases can be paired and the
result appears in Table XXII.

<u>TABLE XXII.</u>

Post-operative atelectasis in relation to the site

and extent of tuberculous disease.

	No. of Cases.	Atelectasis.	Atelectasis per cent.
Ll,2 disease.	27	15	55.56
Comparable Series.	27	7	25.93
Total:	54	22	40.74

Statistical analysis: $x^2 = 3.85$

$n = 1$

$P = .05$

An analysis of the figures presented in Table XXII.
shows that significance is achieved and this in spite of the
fact that no counterpart could be found for 5 cases with
Ll,2 disease, 4 of whom had post-operative atelectasis.   We
may therefore conclude that in thoracoplasty for tuberculosis
throughout the left lung there is a definite predisposition

to develop post-operative atelectasis.

(4) <u>Post-operative atelectasis in relation to operative</u>

    <u>risk.</u>

It has already been seen that there were 22 instances of post-operative atelectasis in 38 patients who were classified in the risk 3 category. Those 38 **patients,** however, included 20 **cases** with tuberculous disease involving the entire left lung and 15 of the latter developed post-operative atelectasis. Operative risk 3 and L1,2 disease therefore cannot be dissociated in the study and in Table XXIII. those factors are considered together.

<u>TABLE XXIII.</u>

Post-operative atelectasis in relation to L1,2 disease

in poor risk patients.

Category.	No. of Cases.	Atelectasis.	Atelectasis per cent.
L1,2 and Risk 3.	20	15	75.00
Others	155	22	14.19
Total:	175	37	21.14

Statistical analysis: $x^2 = 35.72$

                   $n = 1$

                   P is considerably less than 0.01.

We thus reach the most important conclusion (see

Table XXIII.) that emerges from the analysis of the
incidence of post-operative atelectasis in the present
study, namely that when thoracoplasty is performed upon
a patient in poor general condition and suffering from
tuberculosis throughout the left lung there is a marked
predisposition to develop post-operative atelectasis.
This conclusion which has been reached by a careful
process of analysis would seem to be irrefutable.   As
far as the writer is aware no other worker in the field
of thoracoplasty has published a similar finding.

There remains to be explained the high incidence of
post-operative atelectasis among the females of the present
study. Of the 20 patients who have been classified as poor
operative risks with tuberculosis throughout the left lung,
17 were females and 3 only were males and in these the
respective incidence of atelectasis was 13 and two.   When
these cases are excluded statistical analysis gives x^2 a
value of 2.49 and P is less than .20, a result that may
readily be attributed to chance.   Thus in the original
analysis we were not considering comparable groups of male
and female patients.   Sex therefore has exerted a
significant influence in the occurrence of atelectasis only
because a considerably greater number of females with
tuberculosis throughout the left lung were submitted to
thoracoplasty.

Summary and Conclusions:

The cases of thoracoplasty have been investigated with regard to the significance of various initial factors considered likely to have an important bearing on the development of post-operative atelectasis. At the outset the first 30 cases of thoracoplasty were excluded from a statistical analysis because of the relatively high incidence of atelectasis which complicated this group of cases. The results of an analysis of the subsequent 145 cases show that the following factors did not exert a significant influence in the occurrence of atelectasis following thoracoplasty:- age, size of cavity, the operation of phrenic crush, preoperative vital capacity, extent of apicolysis at first stage thoracoplasty, semi-open as opposed to closed circuit anaesthesia and season of the year.

On the other hand the factors of female sex, poor operative risk, tuberculosis throughout the left lung and the resection of more than three ribs at first stage thoracoplasty appear to have exerted a significant effect in the occurrence of post-operative atelectasis.

The introduction of streptomycin therapy and routine change of posture after operation did not reduce the incidence of post-operative atelectasis.

Analysis of the total 175 cases of thoracoplasty did

not materially alter the above findings except in
relation to the size of cavity which for the first time
assumed a significant value.   This latter finding,
however, has been explained by the disproportionately
large number of patients in the first 30 cases of
thoracoplasty in whom a large cavity appeared in
association with a poor operative risk suffering from
tuberculosis throughout the left lung.

Significant values have been carefully checked by
seeking comparable groups of patients equivalent in all
relevant respects except in their differentiation by the
particular factor under consideration.   This method has
led to the final conclusions of the analysis which are as
follows:-

Sex has only exerted a significant influence in the
production of atelectasis in so far as a considerably
greater number of females with tuberculosis throughout the
left lung were submitted to thoracoplasty.

Although it would appear that atelectasis is more
likely to develop when more than three ribs are resected
at first stage thoracoplasty the evidence is not conclusive
and it will be necessary to observe a larger series of cases.

When tuberculosis occurs throughout the left lung
there is a definite predisposition to atelectasis following
thoracoplasty.

In the series of cases under review a poor operative
risk cannot be dissociated from tuberculosis affecting the
whole of the left lung.   When both those factors occurred
together, three out of every four cases developed post-
operative atelectasis and this is considered to be the
most important finding of this part of the investigation.

* * * * * * *

Chapter IV.

Concluding Remarks.

This study of post-operative atelectasis has dealt
with two aspects of the subject, namely (i) the aetiology
of post-operative atelectasis and (ii) post-operative
atelectasis in relation to the operation of thoracoplasty
in pulmonary tuberculosis.

(i) Aetiology of post-operative atelectasis.

In regard to the aetiology of post-operative
atelectasis generally too much emphasis has in the past
been laid on experimental evidence and too little on
clinical observation.   The fact that atelectasis results
from plugging the main bronchus of a dog with infected
secretions from a human case of atelectasis does not
necessarily mean that a similar mechanical obstruction is
present in every case of post-operative atelectasis in man.
One indication of the confusion that surrounds the
pathogenesis of this type of atelectasis is well illustrated
by the following quotations from the literature:-

"It does not appear that previous chest disease
necessarily predisposes to post-operative atelectasis."
(Stringer).

"About half the cases of acute bronchitis and nearly
all those of pulmonary consolidation occurring post-
operatively had shown a predisposition to respiratory
affections.   This was true only in a minority of the

cases developing pulmonary atelectasis." (Holmes).

"It is difficult to believe that the production of mucopurulent sputum within 12 to 18 hours of an operation is possible in the absence of a previously infected bronchial mucous membrane." (Brock).

The fact remains that post-operative atelectasis frequently occurs with remarkable rapidity in a previously healthy lung. An explanation of this phenomenon, based on bronchial constriction arising reflexly through stimulation of the vagus nerve, appears in Part I, Chapter III. It is claimed that constricted bronchi so produced can account for (a) the rapid accumulation of mucus or mucopus in sufficient quantity to cause bronchial obstruction in a previously healthy lung irrespective of the type of anaesthesia used; (b) the failure of ciliary action and bronchial peristalsis to expel secretions; (c) the unproductive cough so characteristic of post-operative atelectasis; (d) the reduction in vital capacity following abdominal operation; (e) the disappointing results obtained with standard methods of treatment of post-operative atelectasis.

The bronchial constriction theory, which receives confirmation from the experimental work of de Takats and his colleagues may be expanded to embrace other aspects of atelectasis but the point is not laboured. The way is

shown, however, for a new approach to what has hitherto
been a confused and baffling problem in the field of
surgery and anaesthesia.   There remains much work to be
done before we can hope to reduce to negligible proportions
the incidence of post-operative atelectasis.

On the other hand an effective method of treating the
dramatic spontaneous atelectasis when it occurs during
operations within the thorax is described for the first
time.   Although two cases only of this condition have been
treated by blocking the vagal fibres supplying the bronchial
musculature of the affected lung the results are most
encouraging.

(ii) Post-operative atelectasis in relation to the
    - operation of thoracoplasty in pulmonary tuberculosis.

Although there is support for the bronchial
constriction theory in Part II. which deals with atelectasis
following thoracoplasty, this section has been confined
purely to a clinical study in order that the issues may not
be confused.

Atelectasis following thoracoplasty in pulmonary
tuberculosis is an unfavourable development, giving rise to
considerable post-operative morbidity and, on occasions, to
fatality.   Diagnosis is by no means easy and the observation
of serial radiographs taken at frequent intervals is necessary
if all the cases are to be recognised.   In the absence of

early complete re-aeration of the atelectatic lung
bronchiectasis has been shown to be a constant feature.
Again it should be emphasised that such bronchiectasis
may be non-tuberculous in nature.   Where persistent
cavitation remains in the midst of permanent atelectasis
the prognosis is bad.

From a preliminary statistical analysis of the
incidence of atelectasis which complicated 175 cases of
thoracoplasty, factors which appeared to have a significant
bearing on the occurrence of this complication were as
follows:-

(a) Sex.

(b) Operative risk.

(c) Tuberculosis throughout the left lung.

(d) The number of ribs resected at first stage
     thoracoplasty.

The analysis might well have terminated at this point
but a method of seeking comparable groups of cases showed
that the only final and irrefutable conclusion that could
emerge from this part of the study was that patients, and
especially those in poor general condition, suffering from
tuberculosis throughout the left lung displayed a
pronounced tendency to develop atelectasis following
thoracoplasty.   This finding is important.   It proves
that in the presence of tuberculous disease affecting the

whole of the left lung thoracoplasty is contra-indicated.
Some other method of surgical treatment, e.g. extrapleural
pneumothorax or pulmonary resection should be adopted in
such cases.

Only when the surgeon and the anaesthetist discuss
together the general physiological state of the patient
as well as the surgical requirements of the case can the
best results be achieved.   In the surgical treatment of
the tuberculous patient team work of this nature is of
paramount importance.   We have gained much experience
over the years at the Thoracic Unit, Mearnskirk Hospital
and we hope that we have benefited by our mistakes.   The
better results that reward our efforts today encourage us
to further endeavour.   It may well be that, as thoracic
units are springing up all over the country, those less
experienced than ourselves may find something of value
from the observations detailed herein.

*   *   *   *   *   *   *

Appendix I.

A mathematical calculation of the absorption of 1,000 cubic centimetres of alveolar air in atelectasis due to bronchial obstruction.   Adapted from Wright (1945).

Notes on abreviations:

c.c.	refers to cubic centimetres.
mm.Hg.	refers to millimetres of Mercury.
O_2	refers to oxygen.
CO_2	refers to carbon dioxide.
N_2	refers to nitrogen.
%	refers to per cent.
p.p.	refers to partial pressure.

Absorption of 1000 c.c's of alveolar air in atelectasis due to bronchial obstruction.

The initial total gas pressure in 1000 c.c. of alveolar air
at body temperature and saturated with

water vapour   = atmospheric pressure

                                      = 760 mm. Hg.

Water vapour pressure   = 47 mm. Hg.

Thus total pressure of the dry gases (O_2; CO_2 and N_2) =

                                  (760 - 47) mm. Hg.

                                  = 713 mm. Hg.

The percentage volumes of O_2, CO_2 and N_2 in
alveolar air are O_2 = 14%; CO_2 = 5.6%; N_2 = 80.4%.

Thus 1000 c.c. of alveolar air is composed of:- 140 c.c. O_2

                                           56 c.c. CO_2

                                         804 c.c. N_2.

The partial pressures of the various gases in a gaseous
mixture are divided between the gases in proportion to their
relative volumes.

Thus the p.p. of O_2 in alveolar air = $(\frac{14}{100} \times 713)$ = 100 mm.Hg.

and " " " CO_2 " "   " = $(\frac{5.6}{100} \times 713)$ = 40 mm.Hg.

and " " " N_2 " "   " = $(\frac{80.4}{100} \times 713)$ = 573 mm.Hg.

The total pressure of O_2; CO_2; and N_2 in the pulmonary venous blood = 706 mm.Hg. wet and (706 - 47) = 659 mm.Hg. dry. (The p.p. of water vapour in alveolar air = the p.p. of water vapour in venous blood).

The percentage volumes of O_2, CO_2 and N_2 in venous blood = 6%, 7% and 87% respectively.

Thus the p.p. of O_2 in the pulmonary venous blood

$$= (\frac{6}{100} \ x \ 659) = \ 40 \ mm.Hg.$$

the p.p. of CO_2 in the pulmonary venous blood

$$= (\frac{7}{100} \ x \ 659) = \ 46 \ mm.Hg.$$

the p.p. of N_2 in the pulmonary venous blood

$$= (\frac{87}{100} \ x \ 659) = 573 \ mm.Hg.$$

Assuming that the pulmonary membrane, though permeable, is rigid and that the alveoli do not retract, then gaseous diffusion will take place between the alveolar air and the venous blood till the total gas pressure and the partial pressures of the individual gases become equal on both sides of the membrane. As the venous blood is constantly being renewed its gas pressures remain constant and the gas pressures in the alveolar air will thus become equal to those in the venous blood. Therefore the original 140 c.c. of O_2 in 1000 c.c. of alveolar air will become

$$\frac{40}{659} \ x \ 100 \ = \ 60 \ c.c.$$

Thus 80 c.c. of O_2 will diffuse into the venous blood.

Similarly 14 c.c. of CO_2 will diffuse from the venous blood into the alveolar air.

No N_2 will diffuse as its partial pressure is the same in alveolar air and venous blood.

Thus the original 1000 c.c. of alveolar air is reduced in volume by 66 c.c. to 934 c.c., made up thus: 60 c.c. O_2

70 c.c. CO_2

804 c.c. N_2.

However, as explained in Part I, Chapter II, the total pressure of the alveolar air will be restored to 760 mm.Hg. wet and 713 mm.Hg. dry due to retraction of the alveoli and the contained gases will come to exert pressures as follows:-

p.p. of O_2 $= (\frac{60}{934}$ x 713) $=$ 46 mm.Hg.

p.p. of CO_2 $= (\frac{70}{934}$ x 713) $=$ 53 mm.Hg.

p.p. of N_2 $= (\frac{804}{934}$ x 713) $=$ 614 mm.Hg.

Thus the partial pressures of all the gases in the alveolar air again become higher than those in the venous blood. As a result further diffusion of alveolar air into the venous blood will take place.   The process continues in this way until the 1000 c.c. of alveolar air is completely absorbed.

*   *   *   *   *   *

Details of 175 cases of pulmonary tuberculosis

submitted to thoracoplasty.

Notes on tabulation:

RISK.   -   refers to the state of the pulmonary disease
and the general condition of the patient.

    1. denotes a stable chronic in good general
condition.

    2. denotes a relapsing chronic in fair general
condition.

    3. denotes a slipping chronic in poor general
condition.

DISEASE.   -   refers to the extent of lung involvement as
seen radiologically.

    RA   denotes tuberculous disease in the apex of the
right lung.

    R1   denotes tuberculous disease in the upper third
of the right lung.

    R1,2   denotes tuberculous disease in the upper and
middle thirds of the right lung.

    R1,2,3   denotes tuberculous disease throughout the
right lung.

    LA   denotes tuberculous disease in the apex of
the left lung.

    L1   denotes tuberculous disease in the upper half
of the left lung.

DISEASE. (Cont.).

     L1,2.   denotes tuberculous disease throughout the

                      left lung.

     H   denotes tuberculous disease in the region of

                      the hilum of the lung.

     T.S.   refers to the thoracoplasty side.

     O.S.   refers to the opposite side.

CAV.   -   refers to the size of cavity as measured on

                      the preoperative radiograph.

     1.   denotes a cavity of less than 2 centimetres

                      diameter.

     2.   denotes a cavity of between 2 and 4 centimetres

                      diameter.

     3.   denotes a cavity (a) of more than 4 centimetres

                      diameter.

                              (b) an extensive system of

                      small cavities.

PH.C. -   refers to the operation of phrenic crush.

VIT.CAP. -   refers to vital capacity (measured in cubic

                      centimetres).

     PRE.   refers to the average preoperative vital

                      capacity.

     POST.   refers to the vital capacity on the day

                      following first stage thoracoplasty.

STAGES and RIBS.   - refers to the stages of operation and the number and sequence of ribs resected at each stage.

ANT.   refers to the resection of anterior portions of ribs at revisionary thoracoplasty.

APICO.   - refers to the degree of apicolysis which was either extensive (denoted by YES), partial or not attempted (denoted by -).

ANAES.   - refers to the anaesthetic administered at first stage thoracoplasty.

P.   denotes pentothal (thiopentone).

Cy.   denotes cyclopropane.

C.   denotes curare (d-tubocurarine chloride).

G.   denotes gas (nitrous oxide).

T.   denotes trilene (trichlorethylene).

F.   denotes flaxedil (gallamine triethiodide).

ATEL.   - refers to post-operative atelectasis.

A.P.   - refers to artificial pneumothorax.

P.P.   - refers to pneumoperitoneum.

* * * * * *

Cases of thoracoplasty.

No.	Sex	Age	Risk	Disease T.S.	Disease O.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Vit. Cap. Post.	Stages and Ribs 1st	2nd	3rd	Ant.	Apico.	Anaes.	Atel.	Remarks.
1	M	34	1	Ra	–	1	–	–	–	1-4	5,6	–	–	–	P.Cy.C.	–	–
2	F	19	3	R1	–	3	–	–	–	1-4	–	–	–	–	P.Cy.C.	Yes	Died 24th Day.
3	F	32	1	Ra	–	1	–	–	–	1-4	5,6	–	–	–	P.Cy.C.	–	–
4	F	20	1	L1	R1	3	–	–	–	1-4	–	–	–	–	P.Cy.C.	–	Died 9th Day.
5	M	28	2	R1	–	3	Yes	–	–	1-3	4-6	7,8	–	Yes	P.Cy.C.	–	–
6	F	30	1	Ra	–	2	–	–	–	1-3	4-7	–	–	Yes	P.Cy.C.	–	–
7	F	31	3	L1	–	3	–	–	–	1-3	4-6	7-9	–	Yes	P.Cy.C.	Yes	–
8	F	20	2	L1	–	3	Yes	–	–	1-3	4-6	7,8	–	Yes	P.Cy.C.	Yes	–
9	F	31	3	L1,2	–	3	–	–	–	1-4	5-7	–	2-4	–	P.Cy.C.	Yes	–
10	M	32	3	L1,2	–	3	Yes	–	–	1-3	4-6	7-9	–	–	P.Cy.C.	Yes	–
11	M	24	2	L1,2	–	3	–	–	–	1-3	4-6	7-9	–	Yes	P.Cy.C.	Yes	–
12	M	22	2	L1	Ra	1	–	–	–	1-3	4-6	–	–	Yes	P.Cy.C.	–	–
13	M	34	1	Ra	–	1	Yes	–	–	1-5	–	–	2-4	Yes	P.Cy.C.	–	Pleura Opened.
14	F	20	3	L1,2	–	3	–	–	–	1-4	5-7	–	–	Yes	P.Cy.C.	Yes	–
15	M	27	1	L1	–	2	–	–	–	1-3	4-6	–	–	Yes	P.Cy.C.	–	–
16	F	41	1	Ra	–	1	Yes	–	–	1-5	–	–	2-4	Yes	P.Cy.C.	–	–
17	F	29	3	L1,2	–	3	–	–	–	1-4	5-8	–	2-4	Partial	P.Cy.C.	Yes	A.P. Left Side.
18	M	31	2	L1,2	–	3	Yes	–	–	1-3	4-6	7,8	–	Yes	P.Cy.C.	Yes	–
19	F	43	3	R1,2,3	La	3	–	–	–	1-4	5-8	–	–	Yes	P.Cy.C.	–	–
20	F	25	3	L1,2	–	3	–	1400	1000	1-4	5-8	–	–	–	P.Cy.C.	Yes	Spread of Disease.
21	F	22	2	R1	–	2	–	1700	800	1-4	5-7	–	–	Yes	P.G.T.C.	Yes	–
22	F	18	1	L1,2	–	3	–	–	–	1-4	5,6	7,8	–	–	P.G.T.C.	Yes	–
23	F	27	3	L1,2	R1	3	Yes	–	–	1-4	5-7	–	3-5	Yes	P.G.T.C.	Yes	Pneumoperitoneum.
24	F	34	1	R1	–	1	–	1800	1000	1-4	5-7	–	–	–	P.G.T.C.	–	–
25	M	19	1	Ra	–	1	–	3300	3000	1-4	5-7	–	–	Yes	P.Cy.C.	–	Small A.P.Right side
26	M	28	1	R1	–	1	–	1900	1500	1-4	5,6	7,8	–	–	P.Cy.C.	–	–
27	F	16	3	L1,2	R1	3	–	1300	1200	1-3	4,5	6,7	–	–	P.G.T.C.	–	Pneumoperitoneum.
28	F	32	3	R1,2,3	L1	3	Yes	700	600	1-3	4-7	–	–	Yes	P.G.T.C.	Yes	Pneumoperitoneum.
29	M	30	2	L1	–	3	–	3400	2500	1-3	4,5	6,7	–	Partial	P.Cy.C.	Yes	–
30	F	25	2	R1	–	3	–	1400	1000	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–

Cases of thoracoplasty (Contd.)

No.	Sex	Age	Risk	Disease T.S.	Disease O.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Vit. Cap. Post.	Stages and Ribs 1st	2nd	3rd	Ant.	Apico.	Anaes.	Atel.	Remarks.
31	F	22	3	L 1,2	–	3	–	1250	900	1-3	4,5	6,7	–	Yes	P.Cy.C.	Yes	–
32	F	16	1	L A	–	1	Yes	1200	1400	1-3	4,5	–	–	Yes	P.Cy.C.	–	–
33	F	18	2	L 1,2	–	2	–	1500	800	1-3	4,5	–	–	Yes	P.Cy.C.	Yes	–
34	M	25	1	R 1	–	2	–	2000	1000	1-3	4,5	–	–	Yes	P.G.T.C.	–	–
35	F	27	2	R 1	–	2	–	1400	900	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	A.P, Left Side.
36	F	21	2	R 1,2,3	–	3	–	1700	1200	1-3	4,5	6,7	–	–	P.G.T.C.	–	–
37	F	20	1	R 1	–	2	–	1500	800	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
38	F	27	1	R A	–	1	–	1900	1200	1-3	4,5	6,7	–	Yes	P.Cy.C.	–	–
39	F	17	1	L 1	–	3	–	1400	800	1-4	5-7	8,9	–	Yes	P.G.T.C.	Yes	–
40	F	27	2	L 1,2	–	3	–	1200	750	1-3	4-7	–	–	–	P.G.T.C.	–	–
41	M	27	3	L 1,2	–	2	–	1400	1000	1-5	6,7	8,9	–	–	P.G.T.C.	–	–
42	M	25	2	R 1,2	L A	3	–	4200	3300	1-4	5-7	8	–	–	P.G.T.C.	–	–
43	M	18	1	R 1	–	2	–	1500	1000	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
44	F	21	1	L A	–	1	–	1600	1100	1-4	5-7	–	–	Yes	P.G.T.C.	–	–
45	F	25	3	L 1,2	–	3	Yes	1800	1300	1-4	5-7	–	–	Yes	P.G.T.C.	–	–
46	F	15	1	R A	L A	1	Yes	1800	600	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
47	M	23	1	R A	–	1	–	–	–	1-6	7-9	–	3-5	Partial	P.G.T.C.	Yes	–
48	F	16	2	L 1	–	2	–	2000	700	1-3	4-6	7,8	–	Yes	P.G.T.C.	–	–
49	M	19	1	R 1	L A	1	–	2300	1700	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
50	F	35	1	R A	–	2	–	1500	500	1-6	–	–	–	Partial	P.G.T.C.	Yes	Died 8th Day.
51	M	31	1	R 1,2	–	3	–	3000	2200	1-3	4,5	6-8	–	Yes	P.G.T.C.	–	–
52	F	34	3	R 1	L A	3	–	1850	600	1-3	4,5	–	–	Partial	P.G.T.C.	–	A.P, Left Side. Spread of Disease.
53	F	22	1	R 1,2	–	2	Yes	1200	800	1-3	4,5	6,7	–	–	P.G.T.C.	–	–
54	M	27	1	L 1	–	2	–	1900	1250	1-4	5-7	–	–	Partial	P.G.T.C.	–	–
55	F	22	1	R 1,2	–	2	–	2100	1050	1-3	4,5	6,7	–	Yes	P.G.T.C.	Yes	–
56	F	24	2	R 1	L A	2	–	2100	1200	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
57	F	15	1	L A	–	1	–	2000	1300	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	Large Effusion.
58	F	24	2	R 1,2,3	–	1	Yes	1400	1100	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	Large Effusion
59	M	24	2	R 1	L A	3	–	2900	2700	1-3	4,5	6,7	–	Yes	P.G.T.C.	–	–
60	F	19	3	R 1,2,3	–	3	Yes	2400	700	1-3	4,5	6,7	–	Yes	P.G.T.C.	Yes	

Cases of thoracoplasty (Contd.)

No.	Sex	Age	Risk	Disease T.S.	Disease O.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Vit. Cap. Post.	Stages and Ribs 1st.	2nd.	3rd.	Ant.	Apico	Anaes	Atel.	Remarks
61	F	15	2	R_1	LA	3	—	2150	1300	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
62	F	17	2	$R_{1,2,3}$	—	2	—	1500	700	1-4	5-7	—	2,3	Yes	P.Cy.C.	—	Pleura Opened.
63	F	20	2	R_1	L_1	3	—	800	800	1-3	4,5	6,7	—	—	P.Cy.C.	—	—
64	M	18	1	R_1	—	2	—	2000	1100	1-3	4-7	—	2,3	—	P.Cy.C.	—	—
65	M	18	1	RA	—	1	—	2300	1200	1-3	4,5	—	—	Partial	P.Cy.C.	—	—
66	F	18	1	R_1	—	1	—	2300	1300	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
67	F	20	3	$R_{1,2,3}$	LH	3	—	—	—	1-3	4,5	6,7	2-4	—	P.Cy.C.	—	—
68	F	23	2	R_1	LH	2	—	2600	1100	1-3	4,5	6-8	—	Partial	P.Cy.C.	—	—
69	F	27	3	$L_{1,2}$	—	3	Yes	1100	500	1-3	4,5	6,7	—	Yes	P.Cy.C.	Yes	—
70	F	20	3	$L_{1,2}$	RA	2	—	1600	900	1-5	6,7	8,9	—	Yes	P.Cy.C.	Yes	—
71	F	19	3	$L_{1,2}$	RA	3	—	1300	800	1-3	4,5	6-8	—	Partial	P.G.T.C.	Yes	—
72	M	21	1	L_1	R_1	2	—	2200	600	1-3	4-6	7,8	—	Partial	P.G.T.C.	—	Paradoxical Respiration+++
73	M	43	3	R_1	—	3	—	1600	1300	1-3	4,5	—	—	Partial	P.Cy.C.	—	Spread of Disease
74	M	35	3	R_1	—	3	—	1900	1400	1-3	4,5	6,7	—	Yes	P.G.T.C.	—	—
75	M	17	2	R_1	—	3	—	2200	1300	1-3	4,5	6-8	—	Yes	P.Cy.C.	—	—
76	M	28	2	L_1	R_1	2	—	2300	1500	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
77	F	23	1	L_1	—	2	—	2000	1200	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
78	F	20	1	LA	—	1	—	2000	1150	1-3	4-6	—	—	Yes	P.Cy.C.	—	—
79	F	22	2	L_1	—	2	—	1300	950	1-3	4,5	6,7	—	Partial	P.Cy.C.	—	—
80	F	20	1	R_1	—	3	Yes	1350	800	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
81	F	27	2	$L_{1,2}$	RA	2	—	1600	1100	1-3	4,5	6,7	—	Yes	P.G.T.C.	—	—
82	F	32	1	R_1	—	2	—	1400	700	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
83	M	27	1	RA	—	1	—	2750	1000	1-5	—	—	—	Partial	P.Cy.C.	—	—
84	M	42	2	$R_{1,2}$	LH	3	—	3000	2500	1-3	4,5	6-8	—	Yes	P.Cy.C.	—	—
85	M	23	2	L_1	RA	2	—	3000	1600	1-3	4,5	6,7	—	Partial	P.Cy.C.	—	—
86	M	19	2	L_1	RA	3	—	2000	1300	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
87	M	22	2	$L_{1,2}$	RH	3	—	3200	2000	1-3	4,5	6,7	—	Yes	P.Cy.C.	—	—
88	F	22	3	$L_{1,2}$	—	3	—	1500	1000	1-3	4-6	7,8	—	Yes	P.Cy.C.	—	—
89	F	24	3	$R_{1,2,3}$	—	2	—	1100	450	1-3	4,5	6,7	—	Partial	P.Cy.C.	—	—
90	F	21	3	$L_{1,2}$	—	3	—	1400	700	1-4	5-7	—	—	Partial	P.Cy.C.	Yes	—

Cases of thoracoplasty (Contd.)

No.	Sex	Age	Risk	Disease T.S.	O.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Post.	1st	2nd	3rd	Ant.	Apico	Anaes.	Atel.	Remarks.
91	M	42	1	L1	Ra	2	–	2000	1100	1-3	4,5	6,7	–	Yes	P.Cy.C.	–	–
92	M	38	2	R1	–	2	–	2000	1700	1-3	4,5	6-8	–	Partial	P.Cy.C.	–	–
93	F	28	2	L1	–	3	–	1500	850	1-3	4,5	6,7	–	Partial	P.Cy.C.	Yes	–
94	F	31	2	L1	–	3	–	1600	900	1-3	4,5	6,7	–	Yes	P.Cy.C.	–	–
95	M	19	2	R1	–	2	–	2200	1000	1-3	4-6	7,8	–	Yes	P.Cy.C.	–	–
96	M	45	1	L1	–	2	–	3500	2000	1-5	6-8	–	–	Partial	P.Cy.C.	–	–
97	M	23	1	Ra	–	2	–	2300	1300	1-3	4-6	–	–	Yes	P.Cy.C.	–	Pleura Opened.
98	M	19	2	R1,2	–	3	Yes	1900	1400	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–
99	F	22	3	L1	–	3	–	1500	1100	1-3	4,5	6-9	–	Yes	P.Cy.C.	–	–
100	F	23	2	L1,2	–	3	Yes	1300	1000	1-3	4-6	7,8	–	–	P.Cy.C.	–	–
101	F	22	1	La	–	1	–	1700	800	1-3	4,5	6,7	–	Partial	P.Cy.C.	–	Pleura Opened.
102	M	20	1	Ra	–	1	–	3300	1800	1-3	4-6	–	–	Partial	P.Cy.C.	–	–
103	M	24	1	R1	–	2	–	–	–	1-3	–	–	–	Partial	P.Cy.C.	–	Died 10th Day.
104	M	19	1	R1	–	2	–	–	–	1-3	4-6	7-9	–	–	P.Cy.C.	–	Large Effusion.
105	F	21	1	R1	–	2	–	1350	850	1-3	4-6	7,8	–	Yes	P.Cy.C.	–	–
106	M	34	1	R1	–	1	–	3200	2000	1-3	4-6	7,8	–	Partial	P.Cy.C.	–	–
107	F	24	2	R1	–	3	–	1200	950	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–
108	F	31	1	L1	–	2	Yes	2300	1900	1-3	4,5	6,7	–	Partial	P.Cy.C.	Yes	–
109	F	30	2	La	Ra	2	–	1700	1150	1-3	4-6	7,8	–	Partial	P.Cy.C.	–	–
110	F	36	2	L1	–	2	–	1400	700	1-3	4,5	6,7	–	–	P.Cy.C.	–	–
111	F	26	1	L1	–	2	–	2200	1700	1-3	4,5	6,7	–	Partial	P.Cy.C.	–	Pleura Opened.
112	F	25	3	L1	RH	3	–	1300	800	1-3	4-8	–	2,3	Partial	P.Cy.C.	–	–
113	F	24	1	L1	–	2	–	1500	950	1-3	4-6	7,8	–	Yes	P.Cy.C.	Yes	–
114	F	20	1	L1	–	1	–	1800	1450	1-3	4,5	–	–	Yes	P.Cy.C.	–	–
115	F	37	1	L1	–	1	–	2000	1100	1-3	4-6	–	–	–	P.Cy.C.	–	–
116	F	43	2	L1	–	3	–	1100	1000	1-3	4,5	–	–	–	P.Cy.C.	–	Bronchitic.
117	M	23	1	R1	–	3	–	2750	1700	1-3	4,5	6,7	–	–	P.Cy.C.	–	Spread of Disease.
118	M	34	2	R1,2	La	2	Yes	2700	1800	1-3	4-6	–	–	Partial	P.Cy.C.	–	Pneumoperitoneum.
119	M	29	1	R1,2	–	2	–	2600	900	1-3	4,5	6,7	–	–	P.Cy.C.	–	Spread of Disease.
120	M	21	2	R1,2,3	–	3	–	2500	1000	1-3	4,5	–	–	Partial	P.Cy.C.	–	Spread of Disease.

Cases of thoracoplasty (Contd.)

No.	Sex.	Age.	Risk.	Disease T.S.	Disease O.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Vit. Cap. Post.	Stages and Ribs. 1st	2nd	3rd	Ant.	Apico	Anaes.	Atel.	Remarks.
121	M	31	2	R1,2	–	1	–	1800	950	1-3	4-6	–	–	Yes	P.Cy.C.	–	Pre-op. Effusion.
122	M	22	3	R1	–	3	Yes	1500	1200	1-3	4,5	6,7	–	Yes	P.Cy.C.	Yes	–
123	M	28	1	L1	–	1	–	2500	1900	1-3	4-6	–	–	Yes	P.Cy.C.	–	–
124	M	32	1	R1	–	1	–	2300	1150	1-3	4-6	7,8	–	Partial	P.Cy.C.	–	A.P. Right Side.
125	M	29	1	R1	L1	3	–	2000	1100	1-3	4-6	7,8	–	Yes	P.G.T.C.	–	–
126	F	34	1	LA	–	3	–	1800	1000	1-3	4,5	6,7	–	–	P.Cy.C.	–	Pleura Opened.
127	M	38	2	L1,2	–	3	–	2500	1400	1-3	4,5	6,7	–	Partial	P.Cy.C.	–	–
128	F	24	3	L1,2	RH	3	Yes	1600	1000	1-3	4-6	7-9	–	Yes	P.Cy.C.	Yes	–
129	M	32	3	R1,2	–	3	–	1450	1000	1-3	4,5	6,7	–	Yes	P.Cy.C.	Yes	A.P. Left Side.
130	M	32	1	RA	–	1	–	3050	2600	1-3	4-6	7	–	Yes	P.Cy.C.	–	–
131	F	38	1	R1	–	3	–	1400	1100	1-3	4-6	–	–	Yes	P.Cy.C.	–	Large Effusion.
132	F	20	1	L1	–	3	Yes	1700	1050	1-4	5-7	–	–	Yes	P.Cy.C.	–	Pneumoperitoneum.
133	M	18	2	L1	RH	2	Yes	2600	1500	1-3	4-7	–	–	Yes	P.Cy.C.	–	Pneumoperitoneum. Pleura Opened.
134	M	19	2	R1,2	–	1	–	2850	2100	1-3	4-6	–	–	–	P.Cy.C.	–	–
135	F	34	2	R1,2,3	–	3	Yes	1500	500	1-3	4-6	7,8	–	Yes	P.Cy.C.	–	–
136	F	27	2	RA	LH	3	–	1900	800	1-3	4-7	–	–	Yes	P.Cy.C.	–	Pleura Opened.
137	F	20	1	L1	–	3	–	2350	1500	1-3	4,5	6,7	–	Yes	P.Cy.C.	–	–
138	M	31	2	R1,2	LH	3	–	2000	1400	1-3	4-7	8,9	–	Partial	P.Cy.C.	–	Spread of Disease.
139	M	28	2	L1	R1	3	–	2850	1950	1-3	4,5	6,7	–	Partial	P.Cy.C.	–	–
140	M	28	2	R1,2	–	3	–	2800	1900	1-3	4,5	6,7	2,3	Partial	P.Cy.C.	–	Large Effusion.
141	M	44	3	L1	–	3	–	2250	2000	1-3	4,5	6,7	–	Partial	P.Cy.C.	Yes	Spread of Disease.
142	F	21	1	L1	–	3	–	1600	700	1-3	4,5	6,7	–	Yes	P.Cy.C.	–	Pleura Opened.
143	F	23	1	L1	–	2	–	1100	700	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–
144	F	24	3	L1,2	–	3	–	1700	800	1-3	4,5	6,7	–	Yes	P.Cy.C	Yes	–
145	F	24	2	R1	–	2	–	1500	900	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–
146	F	32	2	R1,2,3	–	3	–	1350	1000	1-3	4,5	6-8	–	Yes	P.Cy.C.	–	–
147	M	39	1	R1,2	LH	1	Yes	1100	1300	1-4	5-7	–	–	Partial	P.Cy.C.	–	–
148	M	28	2	L1	–	2	–	2000	1300	1-3	4,5	6,7	–	Partial	P.Cy.C.	–	–
149	M	39	3	R1	–	3	Yes	1800	950	1-3	4,5	6	–	Yes	P.Cy.F.	–	–
150	F	38	2	L1	RH	2	–	1450	800	1-3	4,5	–	–	Partial	P.Cy.C.	–	–

Cases of thoracoplasty (Contd.)

No.	Sex	Age	Risk	Disease T.S.	Disease Q.S.	Cav.	Ph.C.	Vit. Cap. Pre.	Vit. Cap. Post.	Stages and Ribs. 1st	2nd	3rd	Ant.	Apico.	Anaes.	Atel.	Remarks.
151	F	20	1	R1	–	2	–	2100	800	1-3	4,5	6-8	–	YES	P.Cy.F.	–	–
152	M	25	2	L1	R1	2	–	1400	1200	1-3	4,5	–	–	YES	P.Cy.F.	–	–
153	M	27	3	L1,2	R1	3	–	1700	1700	1-3	4,5	6,7	–	YES	P.Cy.C	YES	RE-ACTIVATION OF DISEASE.
154	M	18	1	R1,2	–	2	–	2300	1100	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
155	M	38	1	L1	–	2	–	2350	1000	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
156	F	25	3	L1,2	–	3	–	1300	800	1-3	4,5	6,7	–	YES	P.Cy.F.	YES	–
157	M	18	1	L1	–	2	–	2300	700	1-3	4,5	6,7	–	YES	P.Cy.F.	–	LARGE EFFUSION.
158	M	18	1	R1	–	2	–	3000	1700	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
159	F	24	3	R1	LA	3	–	1200	750	1-3	4,5	6,7	–	YES	P.Cy.C.	–	–
160	F	24	2	L1,2	R1	3	–	1500	700	1-3	4,5	6,7	–	YES	P.Cy.F.	–	LARGE EFFUSION.
161	M	38	1	R1,2	–	3	–	1950	1500	1-3	4,5	6,7	–	PARTIAL	P.Cy.F.	–	LARGE EFFUSION.
162	M	46	1	R1,2	–	2	–	1700	1400	1-3	4,5	6,7	–	PARTIAL	P.Cy.F.	–	LARGE EFFUSION.
163	M	27	1	L1	RA	3	–	1600	1100	1-3	4,5	6,7	–	PARTIAL	P.Cy.F.	–	LARGE EFFUSION.
164	M	28	1	L1,2	RH	2	–	3000	2200	1-3	4,5	6,7	–	PARTIAL	P.Cy.F.	–	–
165	M	39	1	R1,2	–	3	–	2400	1850	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
166	F	28	1	R1,2	–	3	–	1600	800	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
167	F	21	3	R1,2,3	LH	3	–	1500	700	1-3	4,5	6,7	–	YES	P.Cy.F.	–	–
168	M	25	1	R1,2	–	2	YES	1700	800	1-3	4-6	–	–	YES	P.Cy.F.	–	PLEURA OPENED.
169	F	20	3	L1,2	RH	3	–	–	–	1-3	–	–	–	YES	P.Cy.F.	–	SPREAD OF DISEASE
170	M	22	1	R1	–	2	YES	2500	1700	1-4	–	–	–	YES	P.Cy.F.	–	–
171	M	38	1	R1	–	1	–	2050	2100	1-3	4-6	–	–	YES	P.Cy.F.	–	–
172	M	22	1	R1,2	–	3	–	1700	2100	4-7	–	–	–	YES	P.Cy.F.	–	MODIFIED THORACOPLASTY.
173	M	33	1	R1,2	–	3	YES	2100	1200	1-4	5,6	–	–	YES	P.Cy.F.	–	PNEUMOPERITONEUM.
174	F	22	1	L1	–	1	–	2100	500	1-3	4-6	–	–	YES	P.Cy.F.	YES	–
175	M	41	2	L1,2	–	2	–	2500	1500	1-4	–	–	–	–	P.Cy.F.	–	SPREAD OF DISEASE.

Appendix III.

Details of the 37 cases of atelectasis following

thoracoplasty in relation to:-

(a) onset of atelectasis.

(b) post-operative reaction.

(c) temperature, pulse and respiratory

rates.

Notes on Tabulation:

DAY.   - refers to the day of onset of post-operative

atelectasis and corresponds to the post-operative

day on which the diagnosis of atelectasis was

apparent.

REACT. - refers to post-operative reaction.

+++   denotes a severe post-operative reaction -

a temperature of 101° Fahrenheit or over was

recorded.

++   denotes a moderate post-operative reaction -

the temperature ranged between 99 and 101°

Fahrenheit.

+   denotes a slight post-operative reaction -

the temperature remained below 99° Fahrenheit.

T.P.R. - refers respectively to the maximum sustained

temperature, pulse rate per minute and respiratory

<u>T.P.R.</u>
(Cont.)   - rate per minute. (A rise of temperature which

subsided within twenty-four hours was

attributed to causes other than atelectasis and

was discounted).

A space containing a dash indicates that atelectasis did
not occur following the corresponding stage of thoracoplasty.
A totally blank space indicates that a corresponding stage
of thoracoplasty was not performed.

*   *   *   *   *   *

Cases of post-operative atelectasis.

Case No.	First Stage					Second Stage					Third Stage					Revisionary				
	Day of Onset	React.	T.	P.	R.	Day of Onset	React.	T.	P.	R.	Day of Onset	React.	T.	P.	R.	Day of Onset	React.	T.	P.	R.
2	2nd	+++	103.6	140	38															
7	—	—	—	—	—	1st	++	100	120	30	1st	+	98.8	110	34					
8	1st	++	99.8	113	27	1st	++	99.5	120	27	2nd	+	98	100	25					
9	2nd	+++	103.4	103	28	1st	+	99	100	22						1st	+	99	106	23
10	1st	+++	103	130	30	1st	+++	101	120	30	1st	++	100	100	25					
11	1st	+++	102	128	30	2nd	+	98	90	22	2nd	+	98	90	22					
14	2nd	+++	101.4	120	29	2nd	++	100.7	110	39										
17	2nd	+++	103	108	29	2nd	+	97.6	90	18						1st	+	97.8	90	22
18	1st	+++	103	110	29	2nd	++	100	100	23	1st	+	98.4	95	20					
20	1st	+++	103.8	130	22	2nd	+	98	120	23										
21	2nd	++	99.6	100	23	1st	+	98.8	86	18										
22	1st	+++	101.7	123	23	1st	++	100.8	120	23	1st	++	99.6	110	27					
23	1st	+++	103.2	124	28	1st	+++	102.6	108	26						1st	++	100.6	120	27
28	—	—	—	—	—	2nd	+++	103	126	30										
29	—	—	—	—	—	—	—	—	—	—	1st	++	99.8	120	27					
31	—	—	—	—	—	1st	+++	103.5	140	33	2nd	+	98.6	120	20					
33	1st	++	100	110	23	1st	+++	103	125	28										
39	1st	+++	101.2	100	23	1st	++	100	118	23	1st	++	99.4	94	23					
47	1st	+++	102	120	23	2nd	++	100.5	120	23						1st	++	100	110	25

Cases of post-operative atelectasis (Contd.)

Case No.	FIRST STAGE					SECOND STAGE					THIRD STAGE					REVISIONARY.				
	Day of Onset	React	T.	P.	R.	Day of Onset	React	T.	P.	R.	Day of Onset	React	T.	P.	R.	Day of Onset	React	T.	P.	R.
50	1st	+++	103	158	33															
55	—	—	—	—	—	—	—	—	—	—	1st	+++	103	120	32					
60	2nd	++	100.8	130	23	1st	+	98.7	108	31	1st	+	98	100	23					
69	2nd	+++	103	145	43	1st	++	100	125	38	1st	+	98.4	140	23					
70	2nd	++	101	130	23	2nd	++	99.2	130	22	1st	+++	101	140	32					
71	1st	+++	103	130	29	1st	++	100	120	25	1st	+	98	110	24					
90	1st	+++	104	140	30	1st	+++	103	130	35										
93	1st	+++	103	130	27	1st	++	100.3	125	25	1st	++	101.6	130	29					
108	2nd	+	98	84	20	1st	++	101	110	22	1st	+	98.8	88	20					
113	1st	+++	102	120	23	6th	+	98.2	96	18	1st	+	98	84	20					
122	—	—	—	—	—	—	—	—	—	—	1st	+++	103	156	32					
128	—	—	—	—	—	1st	+	97.8	80	22	1st	+	98	120	22					
129	—	—	—	—	—	1st	+	98.4	112	22	1st	+	98.2	100	22					
141	—	—	—	—	—	2nd	+	98.4	88	20	1st	+	988	94	23					
144	1st	+++	103	130	25	1st	+	98.6	90	23	1st	+	99	100	24					
153	2nd	+	98.8	86	20	1st	+	98.7	100	25	1st	+	99	110	25					
156	1st	+++	102	130	25	1st	+++	102	146	22	1st	++	100.4	130	27					
174	2nd	++	100	120	26	1st	+	99	100	22										

Appendix IV.

Details of the 37 cases of atelectasis following

thoracoplasty in relation to:-

(a) classification of atelectasis.

(b) degree of re-aeration of the

atelectasis.

(c) final state of the lung.

Notes on Tabulation:

ATEL.   - refers to post-operative atelectasis.

    MASSIVE   - denotes atelectasis involving two-thirds or

more of the affected lung.

    PARTIAL   - denotes patchy or scattered areas of

atelectasis throughout the affected lung.

RESULT.   - refers to the degree of re-aeration of the

atelectasis that was effected during the

interval between the stages of thoracoplasty

or within four weeks of completion of

thoracoplasty.

FINAL RESULT. - refers to the state of the atelectatic lung

six months or longer after completion of

thoracoplasty.

A totally blank space indicates that a corresponding stage
of thoracoplasty was not performed.

*   *   *   *   *   *

Cases of post-operative atelectasis.

Case No.	First Stage Atel.	Result	Second Stage Atel.	Result	Third Stage Atel.	Result	Revisionary Atel.	Result	Final Result.
2	Massive	No Re-aeration							Died of unrelieved atelectasis.
7	Nil	—	Massive	Re-aeration	Massive	Partial Re-aeration			Permanent atelectasis of upper lobe. Signs of bronchiectasis.
8	Massive	Re-aeration	Massive	Partial Re-aeration	Massive	No Re-aeration			Permanent massive atelectasis. Signs of bronchiectasis.
9	Massive	Partial Re-aeration	Massive	Partial Re-aeration.			Massive	Partial Re-aeration	Permanent basal atelectasis. Signs of bronchiectasis.
10	Massive	Partial Re-aeration	Massive	Partial Re-aeration	Massive	No Re-aeration			Permanent massive atelectasis. Signs of bronchiectasis.
11	Massive	Re-aeration	Massive	Re-aeration	Massive	Re-aeration			Early complete re-aeration.
14	Partial	Re-aeration	Massive	No Re-aeration					Permanent basal atelectasis. Signs of bronchiectasis.
17	Massive	Re-aeration	Massive	Partial Re-aeration			Massive	No Re-aeration	Permanent massive atelectasis. Signs of bronchiectasis.
18	Massive	Partial Re-aeration	Massive	Partial Re-aeration	Massive	Temporary Re-aeration			Permanent massive atelectasis. Fatal haemoptysis.
20	Massive	Partial Re-aeration	Massive	No Re-aeration					Permanent massive atelectasis. Signs of bronchiectasis.
21	Massive	Partial Re-aeration	Massive	No Re-aeration					Permanent basal atelectasis. Signs of bronchiectasis.
22	Partial	Re-aeration	Massive	Re-aeration	Massive	No Re-aeration			Permanent massive atelectasis. Signs of bronchiectasis.
23	Massive	No Re-aeration	Massive	No Re-aeration			Massive	No Re-aeration	Permanent massive atelectasis. Signs of bronchiectasis.
28	Nil	—	Massive	No Re-aeration					Gradual re-aeration. Signs of bronchiectasis.
29	Nil	—	Nil	—	Massive	Re-aeration			Early complete re-aeration.
31	Nil	—	Massive	Partial Re-aeration	Massive	No Re-aeration			Permanent atelectasis of upper lobe. Signs of bronchiectasis.
33	Massive	Re-aeration	Massive	Re-aeration					Early complete re-aeration.
39	Massive	Re-aeration	Massive	Re-aeration	Massive	Re-aeration			Early complete re-aeration.
47	Massive	Re-aeration	Massive	Partial Re-aeration			Massive	Re-aeration	Early complete re-aeration.

Cases of post-operative atelectasis (Contd.)

Case No.	First Stage Atel.	Result	Second Stage Atel.	Result	Third Stage Atel.	Result	Revisionary Atel.	Result	Final Result
50	Massive	No Re-aeration							Died of unrelieved atelectasis.
55	Nil	—	Nil	—	Massive	Partial Re-aeration			Gradual Re-aeration Signs of Bronchiectasis.
60	Massive	Re-aeration	Massive	Re-aeration	Massive	Partial Re-aeration			Gradual Re-aeration Signs of Bronchiectasis.
69	Massive	Re-aeration	Massive	No Re-aeration	Massive	No Re-aeration			Patient Not Traced.
70	Massive	Re-aeration	Massive	Partial Re-aeration	Massive	No Re-aeration			Irregular Dismissal.
71	Massive	No Re-aeration	Massive	No Re-aeration	Massive	No Re-aeration			Permanent Massive Atelectasis. Signs of Bronchiectasis.
90	Massive	Re-aeration	Massive	Partial Re-aeration					Permanent Basal Atelectasis. Died after Pneumonectomy.
93	Massive	Re-aeration	Massive	Re-aeration	Massive	Re-aeration			Early Complete Re-aeration.
108	Massive	Re-aeration	Massive	Re-aeration	Massive	No Re-aeration			Permanent Massive Atelectasis. Signs of Bronchiectasis.
113	Partial	Re-aeration	Partial	Re-aeration	Massive	Re-aeration			Early Complete Re-aeration.
122	Nil	—	Nil	—	Massive	Re-aeration			Early Complete Re-aeration.
128	Nil	—	Massive	Re-aeration	Massive	Partial Re-aeration			Permanent Basal Atelectasis. Signs of Bronchiectasis.
129	Nil	—	Partial	Re-aeration	Partial	Re-aeration			Early Complete Re-aeration.
141	Nil	—	Partial	Re-aeration	Massive	Re-aeration			Early Complete Re-aeration.
144	Partial	Re-aeration	Partial	Re-aeration	Massive	Partial Re-aeration			Permanent Basal Atelectasis. Signs of Bronchiectasis.
153	Massive	Re-aeration	Massive	Re-aeration	Massive	Re-aeration			Early Complete Re-aeration.
156	Partial	Partial Re-aeration	Massive	Partial Re-aeration	Massive	No Re-aeration			Permanent Massive Atelectasis. Signs of Bronchiectasis.
174	Massive	Re-aeration	Partial	Re-aeration					Early Complete Re-aeration.

Appendix V.

A comparison of the incidence of atelectasis which complicated, (a) the first 30 cases of thoracoplasty.

(Series A).

and (b) the subsequent 145 cases of thoracoplasty.

(Series B).

in respect of the following factors:-

*   *   *   *   *   *   *

NOTE: ATEL. refers to atelectasis.

Table a.

Age in Years	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
15-21	7	4	57.14	37	5	13.51
21-25	5	3	60.00	47	10	21.28
26-30	7	3	42.86	23	3	13.04
31-35	9	5	55.56	18	3	16.67
36-46	2	-	-	20	1	5.00
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table b.

Size of Cavity.	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
1	8	-	-	25	2	8.00
2	3	1	33.33	55	6	10.91
3	19	14	73.68	65	14	21.54
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table c.

Phrenic Crush.	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
Present	8	5	62.50	22	5	22.73
None	22	10	45.46	123	17	13.82
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table d.

Apicolysis	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
Extensive	17	6	35.29	87	10	11.49
Partial	2	1	50.00	40	6	15.00
None	11	5	45.46	18	-	-
Total:	30	12	40.00	145	16	11.03

* * * * * * *

Table e.

Anaesthesia	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
Semi-open	6	3	50.00	30	5	16.67
Closed	24	9	37.50	115	11	9.57
Total:	30	12	40.00	145	16	11.03

* * * * * * *

Table f.

Season	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
Oct.-Mar.	18	9	50.00	74	12	16.22
Apr.-Sep.	12	6	50.00	71	10	14.08
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table g.

Sex	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
Male	11	4	36.37	69	5	7.25
Female	19	11	57.89	76	17	22.37
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table h.

Risk.	Series A.			Series B.		
	CASES	ATEL.	ATEL.%	CASES	ATEL.	ATEL.%
1	11	1	9.09	69	7	10.14
2	8	5	62.50	49	2	4.08
3	11	9	80.91	27	13	48.15
Total:	30	15	50.00	145	22	15.17

* * * * * * *

Table i.

Extent of Disease.	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
RA	6	–	–	10	2	20.00
R1	6	2	33.33	37	1	2.70
R1,2.	–	–	–	22	2	9.09
R1,2,3.	2	1	50.00	10	1	10.00
LA	–	–	–	7	–	–
L1	6	3	50.00	37	6	16.22
L1,2.	10	9	90.00	22	10	45.45
Total:	30	15	50.00	145	22	15.17
Bilateral	6	2	33.33	39	4	10.26

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Table j.

Ribs.	Series A.			Series B.		
	CASES.	ATEL.	ATEL.%	CASES.	ATEL.	ATEL.%
3	13	4	30.77	127	11	8.66
4 +	17	8	47.06	18	5	27.78
Total:	30	12	40.00	145	16	11.03

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