Dr. Maudsley

Spalding Thores

1861

Very good

Theodore Rooke
"How was his labor act"
Inaugural View

on the

Mechanism

of

Ventricular Emphysema

Session 1860-61.

Gresham Halling.
May 1, 1861.
We are indebted to Laennec for the first accurate description of the condition of the lungs in the different forms of pulmonary Emphysema. He was the first to point out the physical signs by means of which we could discover the condition in the living subject; and also, the first to give a theory as to the mode of its production.

Laennec was anticipated somewhat as to the actual condition of the lungs. He shows some inaccuracy when relating what had been previously observed; for, he says, the Emphysema of which he has given a description was with our unknown. Then, he proceeds to
may, that though no description exists, some authors have given examples. Fontana and Morgagni had given examples of enlarged lungs filled with air. Van Hateren and Strock had noted cases where air was discovered under the pleura. Sir Geo. Floyer in 1698 discovered the same condition in a broken winded mare. Again, he quotes Playfair and Valadier to show that they had discovered the detachment or dilatation of the air-cells.

He, also, refers to Dr. Baillie, who had a tolerably correct idea of the condition of the lungs in emphysema. Dr. Baillie had pointed out that the lungs were enlarged and distended with air, that the air-cells were dilated, and that collections of air occurred under the pleura. He (Dr. Baillie) said, *Mortis Anatomic.*
also, described the post mortem appearances; and shown that the lungs did not collapse when the thorax was opened, that the cells appeared full of air, and that white vesicles were seen on the surface of the lung under the pleura. Further on, Dr. Baille describes the state of nature and condition of persons during life with the morbid state found after death, and adds, that no symptoms were known of means of which the condition could be discovered during life.

The above is the substance of all that was known before Jernsine, and with him came physical diagnostic, cause to reduce Chor to 30.

Jernsine held that vascular depolymer in nearly every case occurred consequent to chronic bronchitis ("Key Calculus"). He
was of opinion, that inspiration was a more powerful act than expiration. To quote from Sir John 
Floyer's translation: "For since 
The muscles of inspiration are con-
tracuent and powerful, while expir-
ation, on the other hand, is produced
chiefly by the elasticity of the parts
and by the fibrous contraction of the
intercostal muscles, it must frequent-
y happen that the air which dur-
ing inspiration had overcome the re-

distance of the constrictor state of the
bronchial membrane and the
esophagus, is unable to force the ob-
struents during expiration, and re-

dains therefore imprisoned in the
air cells. If a mechanism some-
what similar to the valve of an
air gun."

He believed that the more violent
of the subsequent inspirations mainth
Introduction of fresh supplies of air to the nerve cells and occasional dilatation of them, and that when the accumulation was unable to obtain exit, so long as the obstruction remained, expanded, in consequence of becoming heated, it had "some effect" in producing dilatation and causing delirium. He mentions a few occasional causes, such as cases where the breath is long retained, as in the case with players on wind instruments; but this, he adds, is more likely to cause interlobular emphysema. He adds fumoms, aromatic etc. as being very rare causes.

Expiration is a much more powerful act than inspiration. That such is the case, we have ample proof. Professor Bennett has shown that the fact is sufficiently demonstrated in the person of...
Dr. G. on whom the omen was in advance. It was only by means of aspiration that the lungs bulged through the disease.

Professor Burnett has also pointed out, that in a patient who was under his care in the Clinical wards, on whom Mr. Syme performed the operation of pneumothorax, the fluid escaped in a more copious stream during each act of expiration.

Mr. Dobson and Mr. Hutchinson have given ample evidence, in the results of their numerous experiments, which were conducted by the gentlemen with the most of keen care and accuracy, to prove beyond the possibility of a doubt, that such is the fact. The comparative power of the inspiratory and expiratory acts as determined by Mr. Hutchinson from 1500 experiments, is shown in the

following Table:

<table>
<thead>
<tr>
<th>Power of Expansory Muscles</th>
<th>Power of Inspiratory Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5 in this Weak</td>
<td>2 in cher</td>
</tr>
<tr>
<td>2</td>
<td>Ordinary</td>
</tr>
<tr>
<td>2.5</td>
<td>Strong</td>
</tr>
<tr>
<td>3.5</td>
<td>Very Strong</td>
</tr>
<tr>
<td>4.5</td>
<td>Remarkable</td>
</tr>
<tr>
<td>5.5</td>
<td>Very Remarkable</td>
</tr>
<tr>
<td>6</td>
<td>Extraordinary</td>
</tr>
<tr>
<td>7</td>
<td>Very Extraordinary</td>
</tr>
</tbody>
</table>

I have noticed after passing a catheter in order to evacuate the contents of the bladder, that the jet of urine was always increased in size during expiration.

As the expiratory theory is so well supported, we must reject that of the dilatation of the air cells, which, being due to the expansion of heated air.

x Edw. Chev. Trans. 1846.
Dr. C. J. P. Williams says—"When there is partial or complete obstruction in any of the bronchial tubes or cells the inspired air cannot pass with the usual force beyond the obstruction; but it will press with more than usual force into the adjoining tubes and cells, to which its access is quite free and these latter may thus become distended and in time permanently dilated."

The arguments I shall advance, further on, in favour of an expiratory theory, render it unnecessary for me here to enter into a consideration of the merits of the above as a mode of production of the disease in question. I shall also allude to it when giving an exposition of Dr. W. T. Gairdner's theory, in which the mechanism is much the same though the con-
Dr. Williams adds another condition which he considers to be a very frequent cause of the dilatation of the air cells, viz., an inelastic or rigid condition of the longitudinal bronchial fibers, recurring as a result of chronic bronchitis, preventing their elongation. I think this as a cause is more powerful than real. The longitudinal extension of the bronchial fibers can at best take place only to a very limited degree, and when not taking place, the increased amount of expansive power to the interior of the air-cells must be very trifling.

He appeals largely to mortals annulling in order to show what lesions are most frequently found to co-exist in the thorax with emphysema. He details the result of post mortem examination in forty cases, and points out that the condition denominated "collapsed" occurs in connection with the emphysema in the great majority of instances—in 27 of his cases or 67.5 per cent.

I shall enter into a consideration of the value of Dr. Go. theory of the mode of production of pulmonary collapsed, as it is my purpose merely to examine carefully his theory of the mechanism of emphysema. However, I may state shortly, that it appears to me extremely probable, that collapse is very frequently due to other causes besides the one described by Dr. Go. emphysemous in the form of
Tightly, clinging, tenacious, elastic. Parent material, sometimes resembling raw white of egg. (P&G) is very unlikely to perform with a mechanical duty as that of the office of a "full-value." And, though it were insulated to a much greater extent, it would still be unable to act as a value, for at the spot where it was annullated, it would act like a foreign or source of irritation and excite an excitation from the humerus membrane, of means of which it would quickly become insoluble and detached down, and it would also excite the thoracic fibres to effect its extinction. Upon the whole, his theory appears rather to mechanical, but, in the less, I do not deny that it may sometimes operate as a cause. They are not have conditions, sufficient to explain the production of collapse in these cases—
stances, such as, defective innervation, exhausted contractility of the bronchial muscular fibers, general exhaustion of the patient, producing inability to perform the act of inspiration with sufficient energy to effect dilatation of every part of the lung? Expiration is much more readily performed under all circumstances, as it is a much more powerful act than inspiration, and is effected in a great measure by elastic material requiring no effort on the part of the patient, and hence the result, that the air is easily expelled. What is the cause of atelectasis, or pulmonary collapse in newly born children or infants? It only occurs in weakly children. It is a good proof that this is the true cause, that the collapse disappears in a
short time after birth when the child becomes stronger and is able to take more powerful inspirations. Accordingly, is it not rational to suppose that collapse may occasionally occur in the adult in consequence of extreme exhaustion producing inability to expand the air-vessels in the more distant parts of the lungs? Dr. Gairdner’s tables show that many of his examples died in a state of great exhaustion, and that in some instances the state had existed for a length of time before death.

To leave the depression, and proceed to consider Dr. Gairdner’s theory (or, rather, his modification of Lavoisier’s theory) of the mechanism of muscular DEATHS. Lavoisier’s theory had been improved and modified by Dr. Williams before Dr. Gairdner wrote—

Dr. G. states—“But the most serious objection to the expiratory
Theory of this disease is, that the ex-

pect is mechanically incapable

of producing distention of the lung,
or of any part of it. The act

of expiration tends entirely towards

emptying the air vessels by the

uniform pressure of the external

parties of the thorax upon the

whole pulmonary surface; and

even when the air vessels are

maintained at their maximum

or normal state of fullness by

a closed glottis, any further di-

tension of them is out of the que-

tion as would be the further dis-

tension of a bladder blown up

and tied at the neck, by hydro-

static or equalised pressure ap-

plied to its entire external sur-

face. The air vessels can

sustain no obliterating pressure

from the column of air within

the tubes, so that air only con-

comes compressed in virtue of
a force acting on the exterior of
the lung which opposes exactly
to much resistance without an
it creates within. It is un-
usual that a theory so radically
assumed (?) and devoid of di-
cert proof, as this of the produc-
tion of consciousness by ex-
piration, should have been
allowed to maintain a place
in medical literature.

First - expiration is
capable of producing distension of
certain portions of the lung.
Any one who observes an ameliorated
patient during a violent par-
oxysm of coughing will at
once perceive the insufficiency
of the statement contained in
the above quotation. The in-
flating of the distended lung may

x Eden? month. med. journal.
Units effect of injury pressure and distortion of lung on the large thoracic heart.
to see and feel, above the chine, between the cartilages of the larynx, and between the ribs themselves.

Second. — The pressure applied to the surface of the lungs during forced expiration is not "uniform" nor "equalized", because some parts of the walls of the thorax are incapable of exerting so much pressure on the lung as others, and some portions of the thoracic parietes do yield more readily than others to expansive power, applied from without. The apex of the lung is covered with soft, yielding tissue and cannot be subjected to pressure from without.

W. Hutchinson in describing the respiratory movements in the healthy chest says: "The closure of the chest yields in a much less degree than the anterior; all that part co-
As pertaining to the ample of the 
life, situated on either side of the 
spine, cannot be expected to in- 
crease its borders like the anterior 
extension.* The same author 
speaking of extreme expiration, says 
that it is produced by a general 
compensation, approximation and 
contraction of the ribs, and a receding or flattening of the whole anterior part 
of the body, and the ascent of the 
diaphragm.

The moving force (if I may be al- 
lowed to use the expression) thought 
to bear upon the surface of the 
tongue by means of the trachea and 
costal cartilages during expiration, 
does not originate in them, ex- 
cept to a very slight extent, 
for they have very little connect- 
ion with any of the moving agents, 
but is supplied indirectly through 
the ribs. Now, suppose a full inspiration has been taken; the glottis is closed; and all the moving forces of forced expiration are acting. From this, we have all the conditions necessary for a violent effort of coughing. What phenomena are presented before and after the opening of the glottis? At the commencement of the expiratory act, the movements are such as those above quoted from Dr. Hutchinson; but when the maximum force is excited, the glottis still being closed, a change takes place—the lower part of the sternum with the sternal ends of some of the lower costal cartilages advances, the apex of the lung bulges above the clavicle, and bulging also takes place in the intercostal spaces at the anterior part of the thorax; and, lastly, when the glottis is suddenly open-
ed, the lower part of the sternal
veers a little, but quickly
advances again and carries with
that portion of it, the sternal
ends of the costal cartilages.
It is not easy to determine the
exact movements of the upper part
of the sternum during this proc-
I can say with considerable con-

defidence—from personal, though
imperfect experiment—that the
lower part of the sternum advan-
ces during a violent expiratory
effort with a closed glottis.

To quote from Mr. Lister's obser-
vations on the respiratory movements
in Emphysema, and as his ob-
servations are deduced from the
results of numerous carefully con-
ducted experiments, his state-
mants are extremely trustworthy,
and may be considered the simp-
le expression of facts. While,
During inspiration the lower part
of the chest first advances recedes and then advances, during expirations that part first advances and then recedes: But sometimes the advance of expiration is much greater than the falling back of inspiration. The advance of the lower end of the sternum during expiration is due to the quick ascent of the diaphragm, which pushes the lungs suddenly upwards. As the air in the lungs can only escape with difficulty, their lateral diameter is increased and the lower end of the sternum is driven forwards during expiration as much as, or even more than, it falls back during inspiration.

So much for "uniform" or "equalised" pressure on the surface of the lungs during forced
expiration, and the irregularity character of the thoracic painless.

Third. — The expirating theory "absolutely invalid" and "void of proof." It is a mere assertion, and is totally unsupported by fact or reason.

Professor Bumett in one of his clinical lectures this winter, related the case of one of his patients — a gentleman who wished to have the injection of a solution of nitrate of silver tried. The gentleman's air passages were extremely irritable, but being exceedingly sensuous that the operation should be performed, he made an effort to sustain the injection, but his lungs were packed into the trachea. He suddenly experienced a sharp pain in the chest, which was followed by untold dyspnoea, which,
In obstetric practice examples occasionally occur of rupture of air vesicles during very powerful acts of expiration; or, which is the same thing, during the violent expulsive efforts attending the second stage of parturition, when all the auxiliary forces of expiration are called into play, general or subcutaneous emphysema of the neck and face is sometimes produced. Rupture of the air vesicles and production of general emphysema is sometimes the result of a paroxysm of coughing in Hooping-Cough, now, if the forces, which
are, I think, without doubt. The agents in the production of general circulatory states in the above mentioned examples, were operating with less power, acting frequently and extending over a considerable length of time, it is reasonable to suppose—

Taking into consideration, that certain portions of the thoracic passages are capable of yielding during forced expiration—that dilatation of the air-cells would be the result.

The conclusions deducible from the arguments brought forward under these three heads are,—that the inspiratory theory is not radically incorrect, but quite the reverse; and that the pressure exerted on the surface of the lungs is not always equal, that pressure applied from within is capable
of producing distention of portions of the lung, and that the expansion of these portions of the lungs is prevented, e.g. the yielding of certain parts of the thoracic walls, to take place during a violent inspiration, to a greater extent than is normal.

Dr. Gaudin states: — "I am prepared, then, to maintain that employment of the lung cavity, in all cases which I have witnessed, be satisfactorily accounted for by considering it a secondary mechanical lesion, dependent on some condition of the respiratory apparatus leading to partly diminished bulk of the pulmonary tissue, and consequently disturbing the balance of air in inspiration."

In "diminished bulk" of the lung, from whatever cause (the cavity of the pleura being normal), it is not possible for
the elastic properties of the thorax to adapt themselves in a great measure to the altered circumstances?

Mr. Selson shows that retraction of the lower end of the diaphragm, or any set of ribs, occurs on one side in which there exists pulmonary condensation (Collapse), while on the other side of the thorax the respiratory movements are exaggerated. To quote from Mr. Selson: "In the extreme cases, in which no air can enter the lung during the inspiratory efforts, the diaphragm descends with power, and drags down the yielding, spongy lung. The lung is considerably lengthened, and, as no air can get into it, it necessarily collapses at the side and its front, owing to atmospheric pressure. Under these circumstances, the walls of the chest are forced back.
wards. The force of the muscular expansion of the chest is compen-
sated by the superior force of atmospheric pressure. According
to the degree of diaphragmatic descent and of closure or narrow-
ing of the air passages, is the falling back of the thoracic walls
partial or universal."

Dr. Reid states: "The passage
of the air into and from the lung
has an important effect upon
the muscular respirating move-
ments. When a lung, or a con-
siderable portion of it, is prevented
from expanding by disease or
any other cause, the pressure
of the air on the inner surface
of that portion of the chest con-
cerning the inextensible lung is
not new exercised during its
dilatation; in other words, this for-

one of the chest, in expanding, must do so in opposition to the whole of the atmospheric pressure on its outer surface, amounting to 15 lbs. on the square inch. This pressure appears to be too much for the muscles of inspiration acting upon that part of the chest, to overcome, for the ribs are then motionless, or nearly so, and, if the lung is in a state of collapse, the walls of the trachea crossing it fall in.

To quote again from Mr. Lidsey's memoir. — The falling in of the lower end of the trachea, and of the contiguous fifth costal cartilages, is, in these cases, almost invariable, unless, as in old age, the costal cartilages be ossified, then the lower end of the trachea 

*End of Anatomy & Physiology*
of the vacuum may be pretended by the upward and forward movement of the ribs, but in this case there is usually falling in of the little and eighth ribs to the side."

Dr. Gärtner entirely overlooks the fact, that flattening of a portion of the chest is an almost invariable concomitant of phthisis. Deformity of the chest is a frequent consequence of some pleurisy, or of empyema. The lung compressed by pleuritic fluid, or bound down by adhesions, cannot expand again in the act of inspiration, and the side of the chest falls in to accommodate itself to the crippled lung.

Simple uncomplicated empyema is seldom attended with much danger. When it proves fatal, it is so in consequence of the supposition of some other disease. In fact, it affords the patient a pretty
the chance of a long life.

Now, the collapse which caused the lesion (according to Gairdner) must, I suppose, exist in the chest during the remaining portion of the patient's life. In cases of extensive employment, an excessively resonant note is elicited on percussion, on the anterior aspect of the thorax. In these cases (according to B.G.) there would occur accumulations of serous and tramatic collapse in the lower posterior parts of the lungs.

Now, if the collapse were communicated with the extent of employment, we should expect to find dulness on percussion over these portions. I think, in the great majority of cases we might look for it in the brain. Of course, when the patient became exhausted (if exhaustion is allowed, too, to act as a cause) and too
weak to expire with sufficient force
to expand the lungs: the condition
might very readily occur. The
Demonstration of the existing
states in the post-mortem room
would hardly warrant us in coming
to the conclusion that they
stood in the relation of cause
and effect—that the collapse
preceded and was the cause of
the emphysema.
I will venture to state that Em-
physema frequently exists, even
to a considerable extent, in cases
where there is no diminution in
vitality of any part of the lung.
May not extensive Emphy-
seea occasionally press upon
the surrounding pulmonary
tissue and cause collapse?
An Emphysema at the lung becomes
much more voluminous than a
healthy one; and in some in-
stances, where one was more
affected than the other, it has been known to press upon and displace the heart.

Mr. Mainly attributes the production of emphysema to a process of "fatty degeneration" of the "pulmonary membrane" which surrounds the air-cells and forms a support for the capillary pleura.

It is possible that this degeneration may occur subsequent to and be a consequence of the emphysema. That is, the lung tissue which is compressed and unable to fill to perform its function may be removed in this manner.

We have now considered several theories of emphysema.
And each in its turn, on careful examination has been found, either unsupported by fact or insufficient to explain all the requirements of the case. One only remains to be briefly discussed, viz., the Explication theory.

It is, in my humble estimation, worthy of universal adoption by the profession. It is susceptible of proof. The requirements obtained from the laws of Mechanics, and is based on extensive observation and the results of accurate experiments.

Dr Jenner has promulgated and ably supported the Explication in his admirable monograph published in the Med Chirur. Trans. for 1857.

It is unnecessary for me here to do more than simply enumerate the theory laid down by Dr Jenner, as the
arguments in favour of it have already been produced in order to oppose other theories.

Dr. J. says—How it is manifest that if there be parts of the thoracic walls which are more yielding, or which already powerfully expiratory efforts with a closed glottis contract less than others, that the air immediately before the opening of the glottis will be driven from the compressed portions of the lungs into the air-vessels of the lung situated under each part of the walls with a force proportionate to the general muscular and other powers in play, to the local want of compression and to the degree of yielding of the walls at these particular spots.

That thus are such parts, and that they are exactly those
which are most frequently the seat of vesicular emphysema, and the sole seat of extreme dilatation is demonstrable.

It has been stated in the Essay, that the peculiarities of the thorax are the after, the anterior border, and the anterior margin of the base of the lung yield readily, steadily, during a forced expiratory act with a closed glottis. It is equally true, that when the same efforts are made with a partially closed glottis, the same effect will be produced.

Vested Anatomy establishes the fact, that the portions of the lung above enumerated are the seats of vesicular emphysema. Dr. Jenner examined five horses the lungs of which were

*Med. Chir. Trans., vol. 40*
When there is some hindrance to the exit of air from the lungs.
empysematous. In each case he found, that the seat of emphysema was under those portions of the thoracic cavity which are capable of yielding closure expiratory efforts.

Dr. Jenner adds, there was no other affection of the lungs; not, so far as he could judge, any “trace of old or recent collapse.”

What is the direct determining cause of emphysema? Any condition of the lungs which excites frequent and severe paroxysms of coughing; or, any circumstance which produces repeated extreme efforts of expiration. The glottis may be entirely closed or only partially so in either case. We have a good example of the former in chronic bronchitis; of the latter in
draught horses when drawing heavy loads.

To quote again from Dr. Jenner:

"Vesicular emphysema is the invariable and necessary consequence of whatever impedes the free exit of air from the lungs, and at the same time excites powerful expiratory efforts; because by such efforts the air is driven from the more powerfully compressed parts of the lungs into those parts which are less powerfully compressed, and the walls over which yield more or less readily to pressure."

The election of the borders of the lungs as the seat of emphysema, may be determined in some measure by the following:—

W. Rayney Long, the air-vesicles are much larger in the parts most distant from
the centre of the lung. In fact, so large that sometimes normal lung may be mistaken for emphysematous. If this is the case, the air in the lungs, when compressed, must necessarily have a much greater expanding power in these portions, than if the air-cells were smaller.

He further remarks, that not only are the air-cells smaller in the central parts of the lungs than at the periphery but the network of capillaries is much closer and denser.
Summary

I. Eupnea is a much more powerful act than inspiration.
II. Forced expiration is capable of producing distension and occasionally rupture of the air-seccles.
III. Certain portions of the faculties of the thorax yield readily to the pressure of the lung during a forced expiratory act with a closed glotti. This being the case, the air-cells of the subjacent pulmonary texture are liable to distension and dilatation.
IV. Some portions of the thoracic walls perform the expiratory movements with greater force than others.
V. "The air is driven from the more powerfully compressed parts of the lungs into those parts which are less powerfully compressed and the walls of which yield more or less readily to pressure."
VI. We have all the conditions
necessary for the production of emphysema in chronic bronchitis, viz., frequently repeated, violent efforts of expiration with either a closed or partially closed glottis.

VII. Emphysema occurs in those portions of the lungs where the thoracic wall presents least resistance to expansion during expiratory effort.

VIII. The expanding power applied to the interior of the unaffected air cells in a lung partially collapsed, during inspiration, can only be augmented in a very slight degree.

IX. Distention in both of the pulmonary tissue is not necessarily present when emphysema occurs.

G. F. Spalding,
May 1, 1861.