PULMONARY FUNCTION TESTS*

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IN the past decade, a large number of physiological tests have been developed for the quantitative and qualitative evaluation of pulmonary function, these are as important to the practice of medicine as are tests of renal, hepatic or neuromuscular function They don't supplant other diagnostic procedures and cannot take the place of an anatomic, bacteriological, and pathological diagnosis, but they indicate how disease has altered function. After all, this is the important matter for anaesthesists

The primary function of the lung is the uptake of an adequate amount of oxygen and the elimination of the excess of carbon dioxide. This involves, however, a number of processes which we have classified for the sake of simplicity in four categories

I Ventilation.
II. Diffusion.
III. Capillary blood flow in the alveoli.
IV. Compliance and elasticity or the mechanics of breathing

TABLE I

BASIC TESTS OF CARDIO-PULMONARY FUNCTION

Ventilation	Diffusion	Capillary Blood Flow	Mechanics of Breathing
Lung volume	Breathing	Right	Recording of
Total volume	1, Room air	heart	intra-oesophageal
Vital capacity	2, Low or high	catheterization	pressures
Functional residual cap	O_2 mixtures	for pulmonary	simultaneously
Expiratory reserve	Min Vent /L	artery pressure	with air flow
Residual cap/total vol %	O_2 cons/cc/min	at rest and	to determine
Mixing index	CO_2 prod/cc	at exercise	the stiffness
Maximum breathing cap	R Q	with	or the
Minute ventilation	*Arterial Blood*	determination	distensibility
O_2 consumption	CO_2 Content	of the cardiac	of the lungs
CO_2 produced	O_2 content	output	and thorax,
Respiratory quotient	O_2 cap	according to	i e the
Dyspnoea index	Hb O_2 % Sat	the Fick	mechanical
Single breath test	pCO_2	principle	compliance
Bronchospirometry	pO_2		
Right % Left %	pH		
Min. vent	Alv pO_2		
O_2 prod	A A Grad		
CO_2 prod	% Venous admixture		
R Q.	Capacity of diffusion		
V C.	% Dead space		

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A great number of tests have been developed in each category because no single one yields all the information desired, in any single patient. As a matter of fact, dyspnoea may result from ventilation insufficiency as judged by the first series with or without involvement of the other processes. On the other hand, the ventilatory reserves may be normal, but the patient cyanotic and much dyspnoeic if the diffusing capacity for oxygen is considerably reduced by an increased thickness of the alveolar membrane, as will be illustrated by the last case.

I. The *ventilation* must be adequate to maintain an alveolar pO_2 around 100 mm.Hg and an alveolar pCO_2 around 40 mm Hg. What counts, then, is the alveolar ventilation. It is dependent on a great number of factors of which the most important are: (1) The tidal air, (2) The frequency of breathing; (3) The dead space; (4) The functional residual air. As a matter of fact the alveolar ventilation per minute is obtained by subtracting the dead space from the tidal air and multiplying by the respiratory rate.

$$\text{Alv Vent.} = (\text{T.A.} - \text{D.S.}) \times \text{R.R.}$$

At rest, the tidal air is around 500 cc. and the dead space 150 cc. for normal men. The dead space should not make more than 30 per cent of the tidal air. For a given minute ventilation, when the D.S is greater, the alveolar ventilation is smaller.

On the other hand, the dead space and the minute ventilation being constant, the respiratory rate is faster and the alveolar ventilation lower. Let us assume a tidal air of 150 cc. equal to the D.S. with a respiratory rate of 50/min. The total ventilation would be 7.5 l. per min , but the alveolar ventilation almost nil.

There will be a rapid drop of pO_2 and rise of pCO_2 with respiratory acidosis. On the other hand a tidal air of 1500 cc with a respiratory rate of only 5 per minute, and the same dead space, will maintain the same total ventilation, but increase the alveolar ventilation to 6.75 l. per min , that is, more than sufficient to adequately ventilate the alveoli. The effect of the dead space in this case will be diminished to 10 per cent.

From the ventilatory point of view it is better to increase the depth of the respiration instead of the rate especially when the dead space is enlarged.

An increase in the functional residual capacity minimizes the effect of the tidal air and increases the time required to wash the alveoli. The tidal air being equal, it may take 12 instead of 6 breaths to renew the alveolar air.

The dead space is increased in absolute figures when the ventilation/capillary blood flow ratio is increased. In other words when the air goes in non-circulated alveoli, this air does not take part in exchanges and increases the total dead space.

It is evident that an increased dead space is a cause of hyperventilation.

On the other hand the minute ventilation and the maximum breathing capacity have a great influence on the apparition of dyspnoea because the shortness of breath usually starts when the ventilation reaches 30 per cent of the maximum breathing capacity.

II. In certain cases, the alveolar ventilation is adequate but the blood leaving the pulmonary capillaries may be only partly oxygenated if there is an impairment of the diffusion of O_2 through the alveolar membrane. We call this trouble a pulmonary capillary block and its importance is measured by the diminution of the diffusing capacity. This may be estimated by the CO method, the fastest and simplest, or by oxygen mixture breathing—the method that we actually use. It involves a complete study of alveolar ventilation and arterial blood.

The causes of diminution of the diffusing capacity may be classified as follows: (1) Diminution of surface area for diffusion by a sufficient decrease in the number of potent capillaries. (2) Distance for diffusion—any disease of the cardiopulmonary system which leads to intra-alveolar or interstitial pulmonary oedema or a thickening of the alveolar capillary membranes as found in pulmonary fibrosis, in vascular sclerosis, or in pulmonary oedema may decrease the diffusing capacity. (3) The qualities of the tissues. A fibrotic tissue may create more impairment of the O_2 diffusion than an oedematous tissue of the same thickness.

III. On the other hand, the normal pulmonary capillary blood flow is around five litres a minute at rest with a mean pulmonary artery pressure of 13 to 15 mm.Hg. that should not rise at exercise despite a great increase of the cardiac output. In many chronic pulmonary diseases, the pulmonary artery pressure is normal or higher than normal at rest and increases significantly at exercise.

The main causes of pulmonary hypertension are· (1) anoxia, (2) The consequent polycythemia and hypervolemia; (3) Increased cardiac output, (4) Reduction in capacity of pulmonary vascular bed

The right heart catheterization gives us an objective idea of the pulmonary haemodynamics and is absolutely necessary in certain cases to fully appreciate the circulatory disturbances due to cardiopulmonary diseases.

IV. Finally the work of arterializing the venous blood should be achieved with a minimal expenditure of energy by the respiratory and circulatory systems.

The work of ventilating the lungs is done normally by the muscles of respiration. This muscular action is opposed by certain forces which may be divided in three components. (1) The force to overcome elastic resistance; (2) The force to move non-elastic tissues; (3) The force to overcome resistance to airflow through the tracheobronchial tree. Numerous tests are now available which measure the specific resistances to breathing and the forces required to overcome them It would be too long to discuss these methods here, but we can say that they aid in more precise diagnosis and often in more specific therapy.

Now I would like to illustrate how advanced obstructive pulmonary emphysema, anaesthesia during thoracic surgery and pulmonary fibrosis, altered the respiratory function

Table II illustrates the great diminution of the maximum breathing capacity and vital capacity with a considerable increase of the residual capacity in 25 emphysematous patients in the IVth stage. Table III includes the arterial blood studies on the same patients. The CO_2 content and pCO_2 are increased, the pH is down towards acidosis, and the saturation is around 80 instead of 95 per cent. The dead space is greatly enlarged and the diffusing capacity markedly reduced

TABLE II

PULMONARY EMPHYSEMA IV
(25 cases, Mean age 47)

	MAXIMUM BREATHING CAPACITY		
	Determined	After vapo	Predicted
	30 L.	40 L.	141 L.
	LUNG VOLUME		
	Determined		Predicted
Total capacity	5 95 L		4.79 L.
Vital capacity available	2 14 L		3.65 L.
Functional residual capacity	4 56 L		1 79 L.
Expiratory reserve	75 L		65 L.
Residual capacity	3 81 L		1.14 L.
Resid cap /Total cap %	64 %		23 8 %
Mixing Index	7 1 %		1 to 2 5%

TABLE III

ARTERIAL BLOOD STUDIES AT REST

PULMONARY EMPHYSEMA	Determined	Predicted
Minute/ventilation in L /m^2 of body area	5 23	4
O^2 consumed in cc /min/m^2 of body area	144	130
R.Q	78	80
CO^2 content vol %	57 9	50 0
Saturation %	85	95
Arterial pO_2 mm Hg	57	88
Arterial pCO_2 mm Hg	54	40
Arterial pH	7 38	7 40
Alveolar pO_2 mm Hg	82	100
A A gradient in mm Hg	26	< 12
D S in c c	228	160
D S in percentage of tidal air	45	< 30%
Venous admixture in percentage of cardiac output	30	< 6%
Mean capillary pressure for O_2	25	< 20
Oxygen diffusion capacity/m^2 of body area	5.71	> 8

at rest. It is evident that none of the subjects included in this study could afford a major thoracic operation.

Figure 1 summarizes the ventilation and acid base equilibrium studies of 23 non-emphysematous patients during major thoracic surgery. There was a significant diminution of alveolar ventilation with disturbances of ventilation/blood flow ratio, and a subsequent increase in pCO_2 content and respiratory acidosis. The breathing of enriched O_2 mixtures kept their blood fully saturated. As they were not emphysematous, they were able to hyperventilate and eliminate the excess of CO_2 in the first hours of recovery.

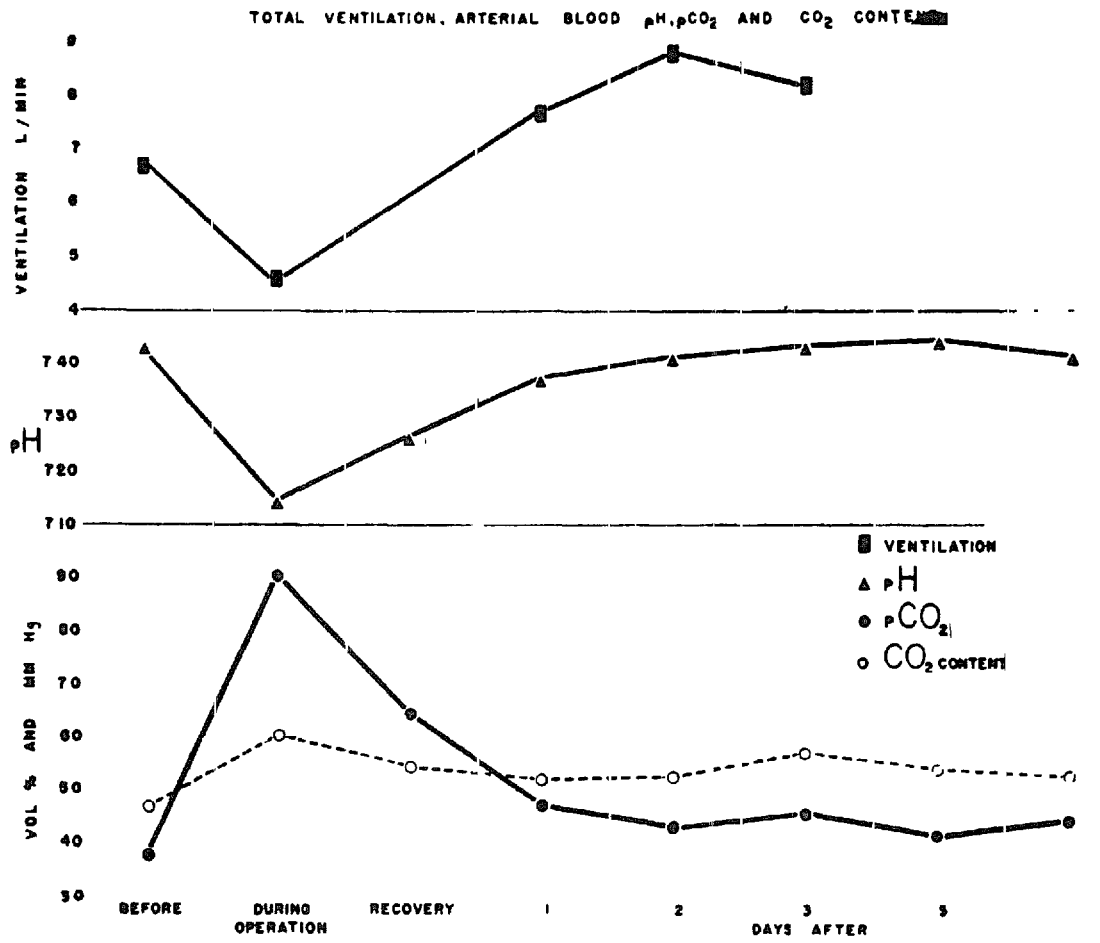

FIGURE 1 Acid base equilibrium during anesthesia

FIGURE 2 Inspiratory and expiratory X-ray (J-G A)

Figure 2 represents the inspiratory and expiratory X-ray of a patient (Mr. J.-G A.) 26 years old sent to us for cough and exertional dyspnoea. This man worked for many years in a dusty place and had suffered from chronic bronchitis since his childhood. For the last year he complained of dyspnoea at work. The X-rays show hypertransparence and trapping of air on the right side. The maximum breathing capacity is greatly diminished but the lung volume is not significantly altered, as seen in Table IV. The bronchospirometry is very instructive because it shows a great diminution of the function on the right side with an increased ventilation/blood flow ratio. At rest, the arterial blood is not normally saturated due to poorly ventilated areas in which the circulation is not proportionally diminished. The venous admixture is increased to 28 instead of 6 per cent. The diffusing capacity at rest is still in normal limits.

It is evident that this patient could be operated on in the right lung, but in case of a left lung tumor, the low function on the right would prevent a left pneumonectomy. It is very rare to find unilateral pulmonary emphysema and this patient is a good example of it.

The next patient (Mr. H.R.) is a 53-year-old male, referred to us for diagnosis and treatment of pulmonary fibrosis. The patient had noticed an increasing dyspnoea since 1947. He coughed and raised much whitish material but did not have fever and did not lose weight. He appeared hyperpnoeic, cyanotic and

TABLE IV

A Case of Unilateral Emphysema (Mr J.-G A.)

	Determined	Predicted
MAXIMUM BREATHING CAPACITY		
	66.55 L.	181 L.
LUNG VOLUME		
Total capacity	5 94 L	5.79 L
Vital capacity available	4 14 L	4 64 L
Functional residual capacity	2 95 L	2 09 L
Expiratory reserve	1 15 L	95 L
Residual capacity	1 80 L	1.15 L.
Resid cap /Total cap %	30 3 %	19 9 %

BRONCHOSPIROMETRY	Right Lung	Left Lung
Ventilation %	28 0	72 0
O_2 consumed %	21 7	78 3
CO_2 produced %	18 7	81 3
R Q	63	76
Vital capacity	34 9	65 1

TABLE V

A Case of Unilateral Emphysema (Mr. J-G A.)

Arterial Blood Studies	At rest 20.93% O_2	At rest 15.26% O_2	At exercise
Minute/ventilation in L	9 06	8 80	29 1
O_2 consumed in c c per min	267.	220.	966
CO_2 produced in c.c per min.	218	210	808.
R.Q	.81	.96	.84
Dyspnea Index	33.9	40	30 1
CO_2 content vol. %	48 67	48 43	46 61
O_2 content vol %	16.71	15 95	17.94
O_2 capacity vol %	18 22	18 29	19.16
Saturation %	91 7	87.2	93 6
Arterial pO_2 mm Hg	66	57.	69.
Arterial pCO_2 mm.Hg.	46.	43.	40.
Arterial pH	7.36	7.39	7.42
Alveolar pO_2 in mm Hg	91.	63	100.
A.A. Gradient in mm Hg	25.	6.	31.
D. S./T. A. %	42.9	39 5	36.6
Diffusion capacity		9 3	
Venous admixture %		28.	

had clubbing of the fingers. There were numerous dry rales at the bottom of both lungs. As can be seen from Table VI, the maximum breathing capacity is normal. This again emphasizes the impossibility of using it as single test. The lung volume is much smaller than normal and all the subdivisions are diminished. The arterial blood is slightly unsaturated at rest with an increased A.A. gradient and percentage of dead space, a diminution of the diffusing capacity at rest and

TABLE VI

A Case of Pulmonary Adenomatosis (Mr. H. R.)

Maximum breathing capacity	Determined	Predicted
	140.5	132 L.

Lung volume	Determined	Predicted
Total capacity	3.75 L.	5.50 L.
Vital capacity available	3.11 L.	4 16 L.
Functional residual capacity	1.38 L.	2.08 L.
Expiratory reserve	.74 L.	.73 L.
Residual capacity	.64 L.	1.34 L.
Resid. cap./Total cap. %	17.1 %	24.4 %

TABLE VII

A Case of Pulmonary Adenomatosis (Mr. H R.)

Arterial Blood Studies at Rest	20.93% O_2	13.16% O_2
Minute/ventilation in L.	9.39	18.1
O_2 consumed in cc per min.	260.	303.
CO_2 produced in cc. per min.	186.	303.
R Q.	.715	1.
Dyspnoea index	36.	59 7
CO_2 content vol %	52.65	46.26
O_2 content vol. %	15 77	12.55
O_2 capacity vol. %	18 36	20.95
Saturation %	85.9	60.
Arterial pO_2 mm Hg.	54.	31.
Arterial pCO_2 mm Hg.	37.	36.
Arterial pH	7 47	7.45
Alveolar pO_2 in mm Hg	97.	58.
A.A gradient in mm Hg.	43.	27.
D. S /T. A. %	46.	52.
Diffusion capacity		6 87
Venous admixture %		49.

a terrific increase of the venous admixture. Figure 3 provides an X-ray result for this patient.

A lung biopsy was done and there proved to be pulmonary adenomatosis. In this case an alveolar capillary block was responsible for the arterial unsaturation and the marked dyspnoea.

A general ideal of cardiopulmonary tests has been given. The importance of

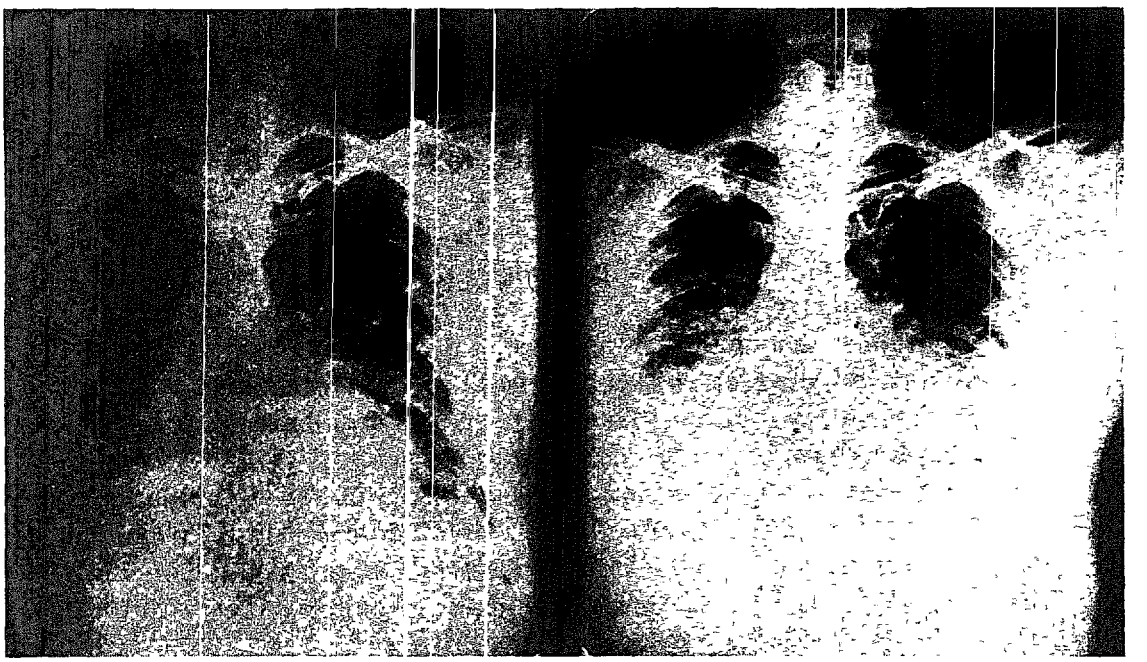

Figure 3. Inspiratory and expiratory X-ray (H. R.)

a physiological study has been stressed, for patients who suffer from a chronic cardiopulmonary condition, and who need major operation, especially on the thorax.

The data on different patients at rest during exercise and during anaesthesia have been presented to illustrate a few theoretical points of interest.

Conclusion

Comroe and his associates in their very instructive and concise book on clinical physiology, entitled *The Lung*, said: "Tests of pulmonary function have proved of definite value both in diagnosis and in guiding therapy of patients with cardiopulmonary disorders." I am sure that a larger utilization of these tests before operating on cardiopulmonary patients would give anaesthetists a better knowledge of the limitations and of the specific function that has been impaired and would help in the patient's pre- and postoperative care as much as in the choice of the anaesthetic.

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Résumé

Durant la dernière décade, un grand nombre de tests physiologiques ont été développés pour l'appréciation qualitative et quantitative de la fonction pulmonaire, et sont demeurés aussi importants pour la pratique de la médecine que les tests de fonction rénale, hépatique ou neuro-vasculaire. On peut diviser la fonction respiratoire en quatre:

I. La ventilation
II. La diffusion
III. Le débit sanguin capillaire pulmonaire.
IV. La mécanique respiratoire

Un certain nombre de tests sont affectés à chaque catégorie et aident à évaluer le degré d'atteinte fonctionnelle et à préciser sa pathogénie. Il ne faut pas oublier en effet que la dyspnée peut aussi bien apparaitre chez un individu dont la capacité respiratoire maximale est normale, s'il y a des troubles de diffusion, que chez un individu aux réserves ventilatoires fortement diminuées.

L'effet de l'espace mort ou nuisible doit être réduit au minimum en augmentant la profondeur plutôt que le nombre de respirations.

Il doit y avoir un équilibre parfait entre la ventilation et la circulation alvéolaire. Le rapport ventilation alvéolaire sur circulation est de 8. S'il augmente c'est signe que certaines alvéoles sont hyperventilées, et s'il diminue c'est qu'il y a hypoventilation. Dans le premier cas, l'espace mort est augmenté, dans le second, c'est la contamination veineuse qui s'accroît.

Le travail d'oxygénation du sang veineux doit se faire avec un minimum de dépenses d'énergie par les systèmes circulatoires et ventilatoires Le cathétérisme

cardiaque au repos et à l'effort apportera des précisions sur le débit cardiaque, les pressions dans l'artère pulmonaire et le travail du ventricule droit.

D'autre part des tests d'élasticité pulmonaire et de changement de volume par variation de pression, renseigneront sur le mécanique ventilatoire.

Pour terminer, quelques données physiologiques concernant des emphysémateux au dernier stade, des patients sous anesthésie et un cas d'adénomatose pulmonaire, ont été présentées afin d'illustrer quelques points importants.

Une plus grande utilisation de ces tests par les anesthésistes fournirait une meilleure connaissance du patient avant l'opération et une meilleure thérapeutique tant préventive que curative.