A CLINICAL APPRAISAL OF A PULMONARY-FUNCTION UNIT

A STUDY OF 100 CONSECUTIVE PATIENTS

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The place of pulmonary-function investigation in clinical medicine, as seen from the clinical point of view, has received scant attention in the literature, and only one recent study[1] and reference to another[2] could be found. On the other hand, several studies[3-5] illustrate the information to be obtained from a pulmonary physiology laboratory, and case reports and individual tests have been considered mainly from the physiological point of view. This probably accounts for the state of affairs which led Bertrand and Williams[6] to state that 'in many instances the availability of these laboratory facilities has not been matched by appropriate clinical use'.

We analysed the records of 100 consecutive patients referred to the pulmonary physiology laboratory and considered each case in relation to the problem as stated on the request form and the physiological data as presented to the clinician; we also evaluated the use made of such data from the clinical summary or progress notes.

Methods in Use in the Laboratory

The spirometer in use is a Godart pulmotest double spirometer calibrated for volume and kymograph speed. The functional residual capacity is determined by the constant-volume, closed-circuit method,[7] using helium as tracer gas. This also allows assessment of the gas-mixing in the lungs.

As an index of ventilating function the maximal mid-expiratory flow rate[8] and maximal breathing capacity are estimated on the spirometer.

Diffusion studies are performed by a simple method[9] and used for exercise studies by a modification previously described in this *Journal*[10] by one of us (H.P.W.).

Analysis of arterial blood for oxygen saturation is performed by the method of Peters and van Slyke[11] or on a Brinkman haemoreflector[12] calibrated against the van Slyke procedure.

Arterial pH, PCO_2 and bicarbonate were measured at 37·5°C. according to the method of Astrup.[13]

A series of normal subjects had values corresponding to the normal values of the prediction formulae used,[14] with the exception of the maximal breathing capacity, which corresponds better to the data of Needham *et al.*[15]

Results

A survey of the request forms shows that 83 patients were referred for a quantitative appraisal of pulmonary dysfunction, diagnosed with confidence on clinical evidence. In only 17 the problem required an evaluation of the physiological disturbance to assist in reaching a definitive diagnosis. In several patients from both groups an evaluation of the effect of therapy on pre-operative assessment was required.

The analysis can thus be considered under 3 arbitrary headings: (1) The material studied, (2) the diagnostic value, and (3) the evaluative use of the pulmonary-function unit.

MATERIAL STUDIED

The final diagnosis recorded on the clinical record of the patient falls into one of the 9 groups in Table I. The groups 'emphysema' and 'fibrosis' contain some cases showing a combination of the conditions, but since the final diagnosis often does not mention the lesser of the two defects, such cases are included under the dominant abnormality.

TABLE I. SUMMARY OF GROUPS

1. Emphysema	45
2. Chronic bronchitis and asthma	16
3. Fibrosis	13
4. Bronchiectasis	6
5. Carcinoma of the lung	3
6. Pulmonary surgery (lobectomy 1, pneumonectomy 1)	2
7. Skeletal deformities	2
8. Functional dyspnoea	1
9. Miscellaneous conditions	12
Total	100

Miscellaneous conditions are considered under 'diagnostic use' below.

It is evident from Table I that conditions 1 - 8 permit of clinical diagnosis. It is also evident that 61 patients referred for study had an obstructive ventilatory defect, while a restrictive defect from fibrosis was present in 13.

Either 'emphysema' or 'fibrosis' was the preliminary diagnosis in 62 patients referred to the laboratory for a quantitative expression of their abnormality — these are analysed further in Table II.

TABLE II. FURTHER ANALYSES OF 62 CASES

Patients referred with 'emphysema'		42
Results of physiological tests		
Diagnosis confirmed	36	
Modified to emphysema and fibrosis	4	
Restrictive abnormality	2	
Patients referred with 'fibrosis'		20
Results of physiological tests		
Diagnosis confirmed	8	
Modified to fibrosis and emphysema	4	
Obstructive abnormality	8	

(The final clinical diagnosis in some of these cases fits into categories 2 and 4 - 8 in Table I.)

In the patients referred, it would appear that emphysema (or an obstructive ventilatory defect) is diagnosed accurately on clinical and radiological evidence. Though not one of the various clinical symptoms and signs or radio-

logical patterns found in emphysema is diagnostic by itself, the clinician, by considering them together, reaches an accurate diagnosis. This does not signify that emphysema cannot at times be overlooked, and in these cases the physiological tests would reveal this ventilatory defect,[4 (case 6)] but such cases were not found in this series.

The diagnosis of fibrosis seems to be made on radiological evidence alone and it would appear that the finding of a diffuse stippling and fibrotic change on roentgenograms is often of decisive importance to the clinician. The correlation between radiological appearance and physiological findings, however, as in the case of emphysema, does not appear to be very good.

Case 1

The patient, a 66-year-old European female, complained of dyspnoea on exertion for several years, and this had increased in severity during the preceding months.

On examination no signs of cardiac failure could be detected. A few coarse basal crepitations were found bilaterally. The electrocardiogram was within normal limits and a roentgenogram of the chest revealed fine peribronchial stippling and fibrosis. Some pleural thickening was seen.

The patient was referred for quantitative assessment of the degree of fibrosis present.

Pulmonary-function data

Lung volume	*Litre BTPS*	*Percentage of predicted normal*
Vital capacity (VC)	1·998	62%
Inspiratory reserve volume (IRV)	1·321	44%
Expiratory reserve volume (ERV)	0·451	37%
Functional residual capacity (FRC)	4·48	145%
Residual volume (RV)	4·03	192%
Total lung capacity (TLC)	6·028	116%
FRC/TLC%	74·3%	(Pred. normal = 59%)
RV/TLC%	66·8%	(Pred. normal = 39%)

Mechanical tests of ventilation		
Maximal breathing capacity	36·09 l./min.	46%
Maximal mid-expiratory flow	1·69 l./sec.	(More than 2 l./sec.)

Blood gas analysis	0_2 saturation 81·5%
Spirogram	Air-trapping
Gas-mixing	Markedly delayed

The lung volume reveals a normal total capacity, but the residual volume and functional residual capacity occupies an abnormally large part of the total lung capacity. There is evidence of prolonged expiration, air-trapping and unequal gas-mixing in the lungs. The maximal breathing capacity is limited. The pattern is that of an obstructive ventilatory defect, and no indication of a restrictive abnormality is apparent.

In a discussion of this case with the physician, he conceded that emphysema was probably also present, but he thought that the fibrosis shown on the roentgenogram was significant. The patient was started on steroid therapy, and a follow-up study after 5 months revealed no significant change from the initial study. Analysis of arterial blood at this date showed arterial hypoxia (90% saturation) and a respiratory acidosis with normal arterial pH from a secondary rise in bicarbonate:

		Normal values
Art. O_2 saturation	90%	95%
Art. pH	7·44	7·36 – 7·44
PCO_2	75 mm.Hg	36 – 44 mm.Hg
Standard bicarbonate	42 mEq./l.	21 – 27 mEq./l.

The finding of a raised PCO_2 with arterial hypoxia always signifies a ventilation defect. Distribution or diffusion defects have a normal or low PCO_2 in association with the arterial hypoxia.

DIAGNOSTIC USE

The 12 patients included in Table I under the heading of 'Miscellaneous conditions' were all referred to obtain aid in the diagnosis. Table III lists the diagnosis in these cases:

TABLE III. DIAGNOSES

Cardio-pulmonary dyspnoea	3
Sarcoidosis	2
Polycythaemia	2
Arteriovenous fistula of the lung	1
Exfoliative dermatitis	1
Fracture (C.5) and paraplegia	1
Rheumatic pneumonia	1
Pulmonary oedema	1
	12

Cardio-pulmonary Dyspnoea

Three patients presenting with a pulmonary condition as well as a cardiac condition, each capable of producing dyspnoea in its own right, fell within this group. The respective conditions were thought to be: mitral stenosis and emphysema, Bantu cardiomyopathy and silicosis, and a mitral lesion with healed fibrotic tuberculosis lesions.

As shown in these cases there appear to be 2 types of restrictive abnormality which we have also encountered on several occasions in other patients studied.

Case 2

A Bantu male, 39 years old, was admitted in cardiac failure after a recent acute attack of rheumatic fever.

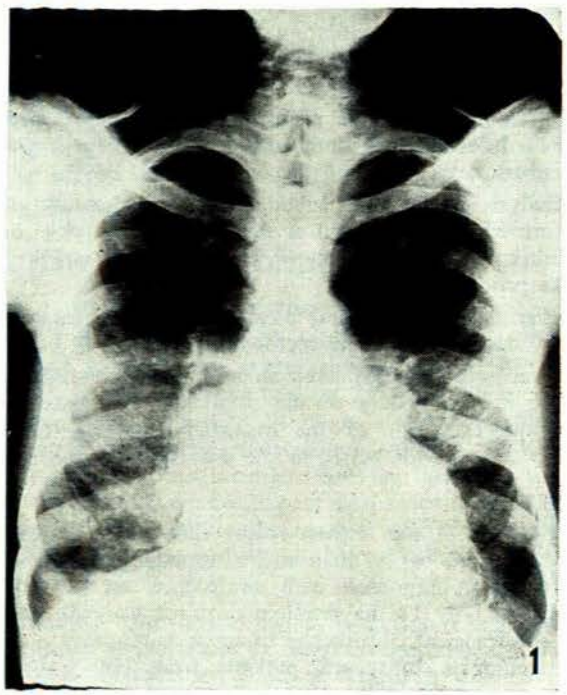

Fig. 1. Mitral stenosis and fibrotic changes in lungs with some congestion — case 2.

The physical examination revealed a tight mitral stenosis and insignificant aortic incompetence. A radiograph of the chest (Fig. 1) showed cardiac enlargement and a mitral configuration of the heart with changes in the lung fields consistent with a fibrotic condition and some congestion.

He was referred for pulmonary-function studies, when his cardiac failure was thought to be controlled by appropriate treatment with digitalis and diuretics.

Pulmonary-function data

Lung volume	*Litres BTPS*	*Percentage of predicted normal*
Inspiratory reserve volume	1·212	
Expiratory reserve volume	1·244	
Vital capacity	3·126	77%
Functional residual capacity	1·428	47%
Residual volume	0·184	11·2%
Total lung capacity	3·310	57%
FRC/TLC%	43·14%	(Pred. normal = 58%)
RV/TLC%	5·56%	(Pred. normal = 30%)
Maximal mid-expiratory flow	2·17 l./sec.	More than 2 l./sec.
Maximal breathing capacity	92·25 l./min.	81·6%

Comment. A restrictive abnormality was present as shown by a decrease in all the subdivisions of lung volume, but this was especially marked in the residual volume. The maximal breathing capacity was slightly decreased. The RV/TLC% was markedly decreased.

At mitral valvotomy a biopsy of the left auricle and of the lung was taken. The histological examination (Prof. H. W. Weber) revealed:

(i) *Left auricle.* Myocardial hypertrophy and chronic non-specific interstitial myocarditis.

(ii) *Lung tissue.* Chronic congestion with moderate fibrosis of pulmonary tissue.

Case 3

A Bantu male, aged 36 years.

The patient received treatment in 2 other hospitals previously for cardiac enlargement and a fibrotic pulmonary condition ascribed to silicosis in 1957 and 1959. He worked underground in a mine for 2 years and has been employed in a stone-quarry for 5 years.

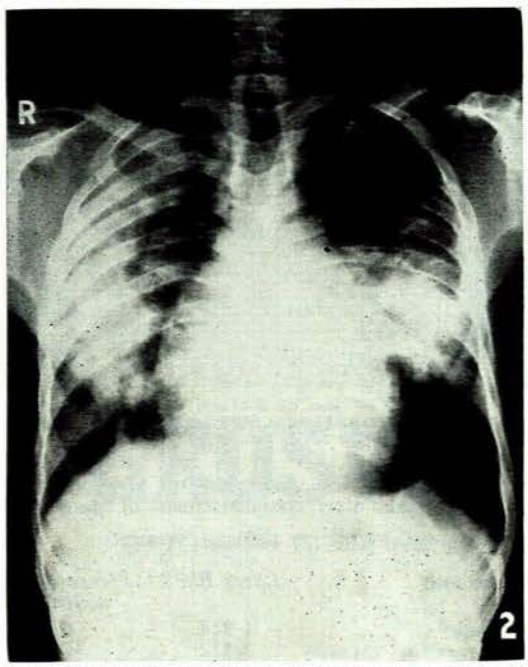

Fig. 2. Cardiomegaly with ill-defined infiltration in both middle lung fields — case 3.

Pulmonary-function tests performed elsewhere were interpreted as showing a restrictive abnormality, but this was not considered to be an important contributory factor to his dyspnoea, the final diagnosis (1959) being 'beri-beri heart disease with chronic non-interfering silicosis'.

Physical examination revealed the signs of congestive cardiac failure. The blood pressure was 110/70 mm.Hg. A systolic murmur of tricuspid incompetence and a triple rhythm were found. The breath sounds were diminished, and dullness on percussion and crepitations were found at both lung bases.

The electrocardiogram showed left ventricular hypertrophy, while the chest X-ray showed dense, ill-defined infiltration in both middle lung fields with cardiomegaly (Fig. 2). A bronchogram revealed deformed upper lobes, lingula and middle lobe on the right, with compensatory emphysema of both lower lobes.

The cardiac failure was brought under control by digitalis, bed rest and diuretics, and the patient was referred for pulmonary-function tests. Cardiac catheterization and lung biopsy were refused.

Pulmonary-function data

Lung volume	*Litres BTPS*	*Percentage predicted normal*
Inspiratory reserve volume	0·936	
Expiratory reserve volume	0·810	
Vital capacity	2·1060	52%
Functional residual capacity	1·6500	54·2%
Total lung capacity	2·9464	52·5%
FRC/TLC%	56·0%	(Pred. normal = 59%)
RV/TLC%	28·5%	(Pred. normal = 29%)
Maximal mid-expiratory flow	1·98 l./sec.	More than 2 l./sec.
Maximal breathing capacity	60·39 l./min.	80%

Comment. In contrast to the position in case 2, the subdivisions of lung volume are proportionately decreased as shown in the normal FRC/TLC and RV/TLC ratios.

A decrease in residual volume may be found in patients with diffuse disease occluding or destroying alveoli in many regions of the lung. This may be progressive in pulmonary granulomatosis and diffuse fibrosis and has also been found in a case with pulmonary alveolar proteinosis.[16] The disproportionate decrease of RV, which we often encountered in patients with congestive cardiac failure (shown to be present in case 2 by the histological findings), suggests that the residual volume is encroached on by congestion, oedema of the alveolar walls or oedema-fluid within the alveoli. This congestion, causing a regional increase in the stiffness of the lung, probably allows the upper part to remain compliant, thus causing less involvement of the FRC. In congestive cardiac failure it has been reported that the RV/TLC increased as a result of a restriction of other lung compartments while the residual volume stayed constant.[16] This was found in some cases, but in our experience more often the opposite was encountered. It is possible that this might result from congestion in lungs with pre-existent fibrosis.

Case 3 shows a restrictive abnormality which we consider significant, all lung volume subdivisions being reduced to about one half of normal. The maximal breathing capacity is impaired. Actual figures for the previous pulmonary-function tests, performed elsewhere, were not available, and arterial blood-gas examination was not performed in this case. Comparison of the previous lung-volume findings would have allowed a decision as to the progression of the pulmonary lesion, and blood-gas studies would have revealed whether ventilation was adequate relative to blood flow.

Sarcoidosis

Although other patients thought to have sarcoidosis are included in this series (e.g. case 5), only the 2 proved by biopsy are discussed here. Pulmonary function may be normal, as found in one of these cases. Obstructive ventilatory defects have been described[17] as well as restrictive abnormalities.[18] The occurrence of diffusion defects in some

cases has received considerable attention[19] and the effect of steroid therapy has been evaluated by pulmonary-function studies.[20]

The following case is interesting because the initial finding was that of a marked obstructive defect superimposed on a restrictive abnormality, and because a clear-cut improvement was noted after steroid therapy. It has been found that the radiological improvement after steroid therapy is usually much more impressive than the physiological changes.[20]

Case 4

A Coloured female, aged 19 years. The patient had a normal pregnancy and delivery 6 months previously and shortly thereafter developed a chronic cough, productive of some mucoid sputum at times. There was a history of loss of weight and progressive dyspnoea.

On physical examination clubbing of the fingers was present, very little chest movement occurred on respiration, and scattered fine crepitations were heard through both lung fields. X-ray of the chest showed a diffuse ground-glass appearance of both lung fields (Fig. 3).

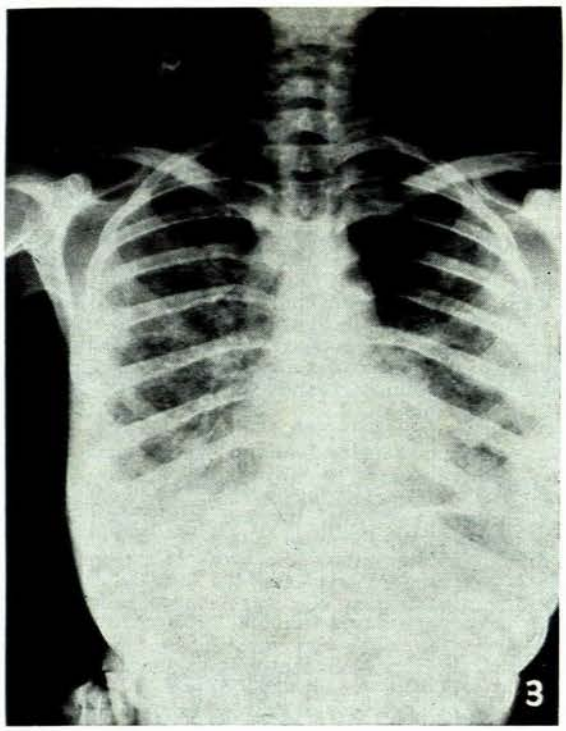

Fig. 3. Diffuse ground-glass appearance of both lung fields. X-ray of chest 3 weeks before lung biopsy — case 4.

Pulmonary-function tests (18 January 1961)

Lung volume	*Litres BTPS*	*Percentage of predicted normal*
Vital capacity	0·955	33%
Functional residual capacity	2·391	101%
Residual volume	2·072	220·4%
Total lung capacity	3·028	76%
FRC/TLC%	79%	(Normal = 62%)
RV/TLC%	68%	(Normal = 27%)
Ventilatory tests		
Maximal mid-expiratory flow	0·828 l./sec.	(More than 2 l./sec.)
Maximal breathing capacity	28·5 l./min.	36%

Diffusion studies

CO removal	15·4%	(Normal=50–60%)
Art. O_2 saturation	79%	
Art. PCO_2	52 mm.Hg	
Art. pH	7·50	
Standard bicarbonate	36 mEq./l.	

Comment. A restrictive abnormality is present (TLC=76%), but the RV and FRC are increased relative to the total lung capacity. There is a greatly reduced maximal mid-expiratory airflow and maximal breathing capacity.

The impairment of diffusion correlates with the patient's grade IV dyspnoea, and could be the result of the obstructive ventilatory defect present. It does not necessarily indicate impaired membrane diffusion. The arterial hypoxia (which escaped clinical observation) cannot, however, be explained solely by defective ventilation since there is but relatively little CO_2 retention. There is also a metabolic alkalaemia which could not be explained.

On the basis of these findings we thought that the patient was a bad operative risk, but the physician felt that a lung biopsy would be justified to aid further management. During the biopsy procedure the lung was observed to be of nearly solid appearance and the histology was that of sarcoidosis. The patient had a very stormy postoperative course, but recovered and subsequently responded well to steroid therapy. Clinically the improvement was striking and this was associated with marked radiological improvement. The chest X-ray (Fig. 4)

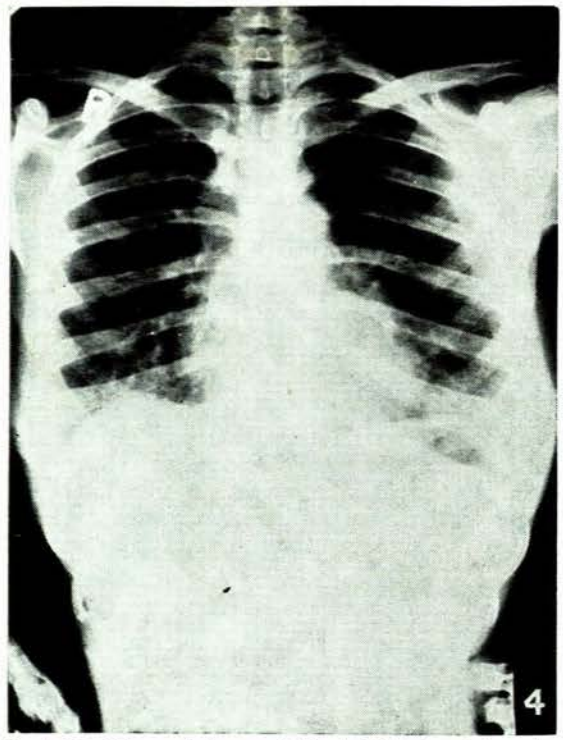

Fig. 4. Final X-ray of chest on 2 March 1961 taken 3 days after the follow-up pulmonary-function study and 7 weeks after commencement of steroid therapy — case 4.

was taken 3 days after the follow-up pulmonary-function studies and 7 weeks after commencement of steroid therapy.

Pulmonary-function data (27 February 1961)

Lung volume	*Litres BSPS*	*Percentage of predicted normal*
Vital capacity	1·814	62%
Functional residual capacity	2·132	91%
Residual volume	1·287	137%
Total lung capacity	3·101	78%

	Litres BSPS	*Percentage of predicted normal*
FRC/TLC%	69%	(Normal = 62%)
RV/TLC%	42%	(Normal = 27%)
Maximal mid-expiratory flow	3·13 l./sec.	
Maximal breathing capacity	75·14 l./min.	94%
CO extraction	30·3%	
Art. O_2	88%	
PCO_2	26·5 mm.Hg	
pH	7·41	
Standard bicarbonate	18·5 mEq./l.	

The data, presented above, reveal a marked improvement from a decrease in the obstructive element. The total lung capacity is still approximately the same, but the expiratory flow rate and breathing capacity are normal. The diffusion is still impaired and the blood-gas analysis now confirms a diffusion defect in a patient with physiological and clinical evidence of diffuse fibrosis.

The striking functional improvement appears to be due to relief of bronchospasm or other reversible cause of obstructive ventilatory defect.

Other Conditions

Polycythaemia. The 2 cases studied presented the problem of differential diagnosis between a polycythaemia vera and secondary polycythaemia. From both clinical and physiological points of view these cases present interesting problems. We are at present studying this condition and will not consider these cases further.

Arteriovenous fistula of the lung. This case has been described in full elsewhere by one of us (A.J.B.).[21]

Pulmonary-function studies can confirm the presence of shunting and distinguish it from other causes of cyanosis.[4] Ear oxymetry and/or blood-gas analysis before and after breathing 100% oxygen will demonstrate failure to attain arterial levels of oxygenation in the presence of a shunt. In this specific instance the patient also had a fibrotic pulmonary lesion and emphysema, and pulmonary-function tests including bronchospirometry were used to assess the operative risk.[21]

Fracture, C.5. This patient was injured in a fall sustained in a mountaineering expedition and was paralysed from the neck downwards. The orthopaedic surgeons considered doing a laminectomy and the anaesthetist requested pulmonary-function tests, asking specifically for an assessment of the patient's ability to produce an effective cough. The lung volume showed some increase in residual volume, the vital capacity was impaired (40%), and the maximal breathing capacity was reduced to 35% of normal. This is not a true reflection of lung volume, however, as the patient was studied in a supine position.

The problem here was to assess the effectiveness of his coughing ability. We considered the effectiveness of a cough to be proportionate to the ability to produce a high instantaneous flow-rate, and thought that the peak flow-meter of Wright and McKerrow[22] might be of especial use in this case. We are not aware of any mention of its use in this respect in the literature. The best reading of 5 attempts was 190 l./min., and from prediction tables[23] a flow of about 620 l./min. would be expected. This would in our opinion reflect a severe impairment of the potential coughing ability.

The patient with exfoliative dermatitis was studied at the request of the dermatology section to exclude emphysema. Laboratory tests revealed approximately normal pulmonary function.

The patient with rheumatic pneumonia was studied after recovery, and an indication for these studies, at that time, was not clear. Apart from a slightly impaired maximal breathing capacity, pulmonary function was found to be normal.

The patient with pulmonary oedema was studied when radiological signs in the lungs failed to improve with clinical improvement in a patient with hypertensive cardiac failure. The findings were compatible with early emphysema, and some restrictive abnormality.

EVALUATIVE FUNCTION

The Assessment of Drug Therapy

In many cases a bronchodilator was used in an endeavour to differentiate a reversible obstruction (bronchospasm) from the irreversible change of emphysema. In 5 patients the physician requested a study of the effect of bronchodilators. In 3 patients with emphysema a considerable improvement was demonstrable following the use of a bronchodilator.

The Effect of Steroid Therapy

The value of steroid therapy could be judged in a case of sarcoidosis (*vide supra*). Four other patients with radiological signs of a diffuse fibrosis were referred especially to study the effect of steroid therapy. One of these proved to have emphysema rather than fibrosis (case 1). Two patients were on steroid therapy for some time before studies were performed and, although subjectively improved, they showed little evidence of physiological improvement.

In one patient studied before initiation of therapy and

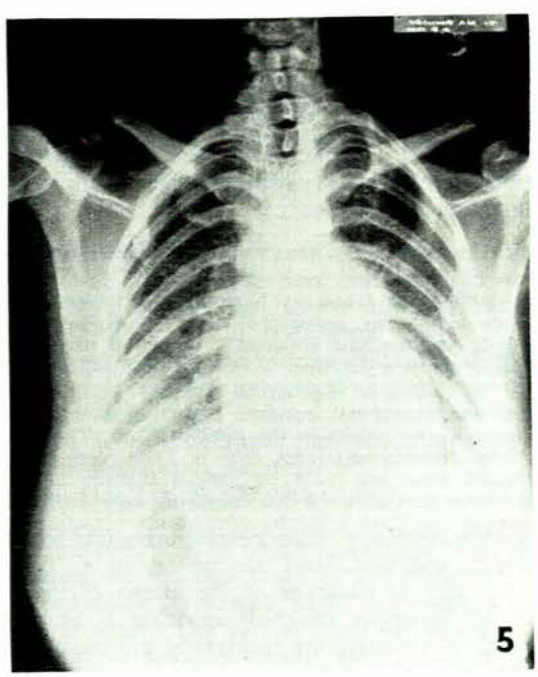

Fig. 5. Diffuse mottling of lower lung fields, and left hilar mass, which appeared to be enlarged glands on tomography — case 5.

again at intervals of 3 months and 1 year after the start of therapy a great improvement was noted in the first 3 months, but practically no change thereafter.

Case 5

A European female, 31 years old, complained of dyspnoea for the past 4 years since the birth of a child. This improved slightly, but again became worse with a subsequent pregnancy. Owing to dyspnoea on exertion she was unable to perform ordinary household duties. At the age of 14 she had a deep venous thrombosis after an appendicectomy.

On examination, she appeared plethoric and had a peripheral cyanosis. There was telangiectasis on the face. Diffuse bilateral crepitations were heard in the chest. The heart was found to be normal.

A roentgenogram of the chest (Fig. 5) showed a diffuse mottling of the lower lung fields and a mass in the left hilar region, which appeared to be enlarged glands on tomography.

The normal sedimentation rate (2 mm. Westergren) was thought to preclude disseminated lupus erythematosus. A negative tuberculin test (1/1000) with normal sedimentation rate, raised gamma globulin and enlarged hilar lymph-nodes, was thought to favour the diagnosis of sarcoidosis. The patient improved on steroid therapy, and although histological evidence was not sought for, this was the accepted diagnosis.

The pulmonary-function data as at August and November 1959 and September 1961 are set out in Fig. 6. The subsequent

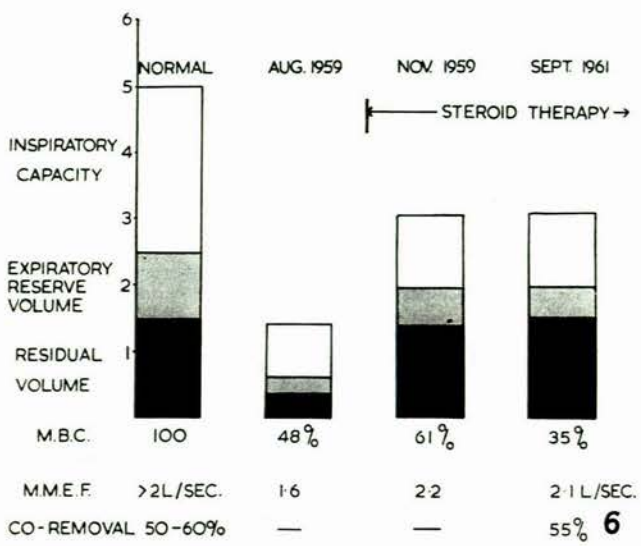

Fig. 6. Pulmonary-function data: lung volume in litre BTPS.

chest radiographs taken in November 1959 and September 1961 are similar in appearance (Fig. 7). Radiologically the total lung volume had apparently increased while the lower lung fields show a decrease in the diffuse mottling.

The lung volumes showed an increase mainly in residual volume and functional residual capacity, though there was also some improvement in the vital capacity. The increase in total lung capacity was from 27% to 57%. Very little change was noted after one year on steroid therapy although the patient maintains that she has improved considerably.

Bronchospirometry. Bronchospirometry was requested in 8 patients and performed successfully in 4. The failure in 1 was due to blockage of the lumen of the tube by tenacious secretions coughed up from a bronchiectatic lung and necessitating the removal of the tube. In another patient adequate anaesthesia of the pharynx and larynx could not be obtained even after they were sprayed with 40 ml. of 4% 'xylotox'. The 2 other failures were due to an inability to pass the Carlens tube.

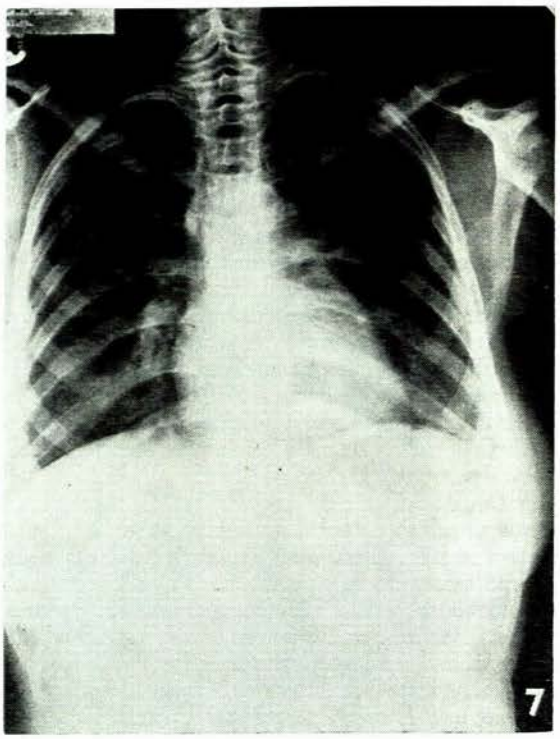

Fig. 7. Follow-up X-ray of chest after steroid therapy — case 5. (Note the radiologically apparent increase in total volume as compared to Fig. 5.)

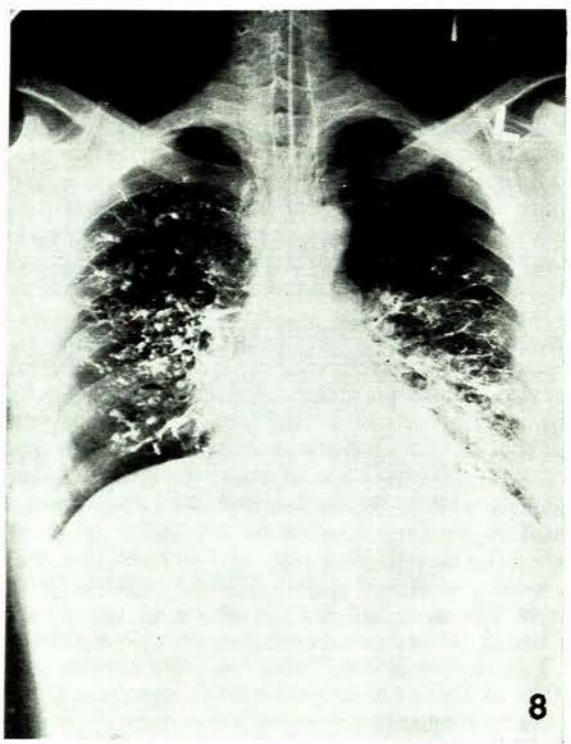

Fig. 8. Moderate, diffuse bronchiectasis of right lung — bronchogram, case 5.

Case 6

A Coloured male, 31 years old, complained of haemoptyses for 5 days, occurring 10 - 15 times per day. Crepitations were found at the right base, but no further signs were found on physical examination. Bronchoscopy showed a blood clot in the right main bronchus, and the left bronchial tree was normal. No cause for the haemoptysis was found.

Bronchogram (Fig. 8) showed a moderate diffuse bronchiectasis of the right lung.

Diagnosis: mild bronchiectasis.

Pulmonary-function data	*Percentage of normal*
Lung volume	
Vital capacity	61%
FRC	86%
RV	109%
TLC	73%
Maximal breathing capacity	90%
CO removal	34·1%

Diffusion study: CL, at rest, 4·74 ml./min./mm.Hg; on exercise, 4·116 ml./min./mm.Hg.

The lung volumes show some restrictive abnormality but no gas-trapping, and an approximately normal maximal breathing capacity. The impaired diffusion, and especially the fact that the conductance decreased on exercise, pointed to a serious physiological defect.

Differential function	*Ratio*
Tests	*right : left lung*
IRV	54 : 45
ERV	60 : 40
Vital capacity	56 : 44
O_2 uptake	10 : 91·24 ml./min.

The maximal breathing capacity revealed that the right lung accounted for about 60% of the ventilation, but the oxygen uptake from that lung was about 10% of the total O_2 uptake.

The blood-gas studies showed arterial hypoxia with a normal PCO_2 and pH. This gave further support to the finding of a limited diffusion, which fell on exercise. Marked disturbance in ventilation-perfusion ratio could also explain these findings.

Art. O_2 saturation	87%
Art. PCO_2	43 mm.Hg
Art. pH	7·49
Standard bicarbonate	24 mEq./l.

On consultation with a thoracic surgeon it was felt that since operation would entail a pneumonectomy it was preferable to follow-up the patient for a further year. The physiological findings were at variance with what was considered clinically to be a mild diffuse bronchiectasis.

In retrospect, this patient's clinical picture and physiological data conform to the findings reported in cases of unilateral non-functioning lung.[28]

A review of these 100 cases suggests that although bronchospirometry was requested in only 8 cases it might have been of value in a further 8 conditions where the pulmonary pathology had been mainly unilateral in distribution.

DISCUSSION

This series of 100 patients is very similar to the series reported from the Mayo Clinic[1] — even the final diagnoses being very similar. It is also evident that most of the patients were referred for a quantitative assessment of an abnormality already known to be present. This was found to be the case in 83% in our series and in 77% in the series of Divertie.[1] It would appear that at present the clinician requires a quantitative expression of ventilatory abnormality in most of the patients referred; but it is evident that in many in whom a complete battery of tests were done, the quantitative expression could be obtained more economically as to time and labour if simple screening tests (e.g. the peak flow-meter for ventilation, and a blood-gas analysis for PCO_2 and O_2 saturation (haemoreflector) were performed.

Patients referred with 'emphysema' appear to be diagnosed fairly accurately on clinical grounds, but the presence of a restrictive abnormality is difficult to find clinically. This was also the experience of Divertie *et al.*,[1] but it is contrary to the impression prevalent in the literature. It might be valuable to refer all patients with fibrosis for physiological study before concluding that the radiological picture truly reflects the physiological disturbance in such a case.

Bronchospirometry should probably be performed more often, since valuable information may occasionally be obtained.

If simple and quickly performed tests are performed where 'quantitation' is the main clinical requirement, more intensive study could be offered to those patients (17% of this series) in whom diagnostic aid is really required. It does not appear to be generally appreciated that performing pulmonary-function tests and calculating the data require considerable time and labour. Bates[5] considered that one full-time research worker and one part-time worker with 2 fully trained technicians could handle about 12 patients a week. During the past 3 years we studied on an average 72 patients *per annum*, apart from studies on normal subjects and some experimental studies. This figure appears to bear out the experience of Bates.[5]

We attempted to assess the use made of data provided by the laboratory by analysing the final clinical summary by the registrar in charge of the case. This analysis excludes all outpatients and those cases where either the pulmonary-function report or the clinical summary was not available in the patients' clinical record at the time of study.

Clinical records complying with these requirements	58
Laboratory report not mentioned	27
Laboratory report mentioned	31
Data presented without discussion	9
Data presented and discussed with reference to clinical findings	22

The cases where the laboratory report was not mentioned are those in which the studies were performed to obtain quantitative information and where such information agreed closely to the clinical assessment.

Where data were presented without discussion they very often supported the clinical impression. Those cases where unexpected findings were reported or where diagnostic aid was found in 'difficult' cases, form the group in which such data were adequately discussed.

To illustrate the fact that blood-gas analysis is probably the most valuable single physiological test, we present 2 patients with emphysema in whose cases there is no diagnostic problem. In the first (case 7) the problem of an operative procedure presented itself, but was decided against before study. The second (case 8) illustrates the development of an acute respiratory failure and its 'compensation' by the secondary response. The spirometric investigation confirmed the presence of emphysema.

Case 7

A European male, 47 years old. He had a history of a chronic cough, dating back 22 years following the inhalation of petrol. During the winter the cough was occasionally found to be productive of sputum. The cough was generally worse during the winter. About 9 months before the present consultation he had been severely ill with a pulmonary infection for a few weeks and since then had never been the same. He became dyspnoeic on exertion and his family noticed cyanosis on exertion.

Physical examination. Peripheral and central cyanosis was present. No signs of cardiac failure were seen. Blood pressure 140/80 mm.Hg.

Chest. Scattered rhonchi, emphysematous chest and prolonged expiration.

Erythrocyte sedimentation rate = 2 mm., *haemoglobin* = 16 G. per 100 ml.

Fluoroscopy. There was very little movement of the diaphragm, which was low in position. The chest moved little with respiration.

X-ray chest (Fig. 9). Several large emphysematous bullae occupied about one half of the right hemithorax, the appearance suggesting a vanishing lung. Similar bullae were present on the left side.

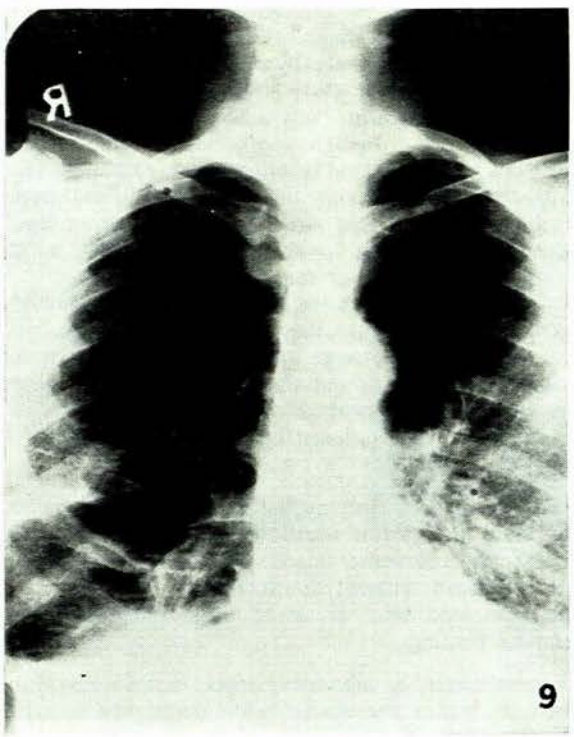

Fig. 9. Bullous emphysema. Several large bullae, one occupying about one half of the right hemithorax.

Pulmonary-function data

Lung volume	*Litres BTPS*	*Percentage predicted normal*
VC	2·073	43%
FRC	5·057	210%
RV	4·085	173%
TLC	6·288	84%
RV/TLC%	64%	(Normal = 33%)
FRC/TLC%	80%	(Normal = 50%)
MEF	0·36 l./sec.	(Normal more than 2 l./sec.)
MBC	29 l./min.	19·5%

The lung volume shows a decrease in total lung capacity. Gas-mixing is severely impaired, the residual volume is increased, and air trapping is evident on the spirograms. The mid-expiratory flow is greatly decreased. The maximal breathing capacity is markedly decreased.

Blood-gas analysis

Art. O_2 saturation	82%
pH	7·36
PCO_2	47 mm.Hg (36 - 44 mm.Hg)
Standard bicarbonate	24·5

These studies were carried out at rest, and the marked undersaturation with insignificant increase in CO_2-tension suggest that the ventilatory defect is not the predominant physiological defect in this case. There is markedly uneven gas-mixing, and the mechanism responsible for the hypoxia appears to be rather a maldistribution of blood flow relative to ventilation.

An excellent review of the clinical and physiological aspects of bullous emphysema and pulmonary cysts is presented by Osler Abbott.[24] A full physiological study should include venous catheterization and gas analysis of the contents of such bullae. Pulmonary-function laboratory studies have been performed by Kaltreider and Fray[25] and Baldwin *et al.*[26] In 6 subjects Kaltreider and Fray found the arterial CO_2 content to be high in 3 and normal in 3 cases, with arterial hypoxia in 4 of the 6 patients. Baldwin *et al.* divide their 16 patients into 3 groups, and our patient probably fits their category IIIb where the following findings were recorded:

Some restriction of total lung capacity despite a high residual volume, MBC less than 20% in 3 of the 5 patients, and the usual criteria for the diagnosis of emphysema. All patients showed arterial hypoxia on exercise, and only 2 were normal at rest, but, as in our case, the PCO_2 was normal in 3 out of 4 patients in whom it was investigated.

The problem is summarized by these authors[25] as follows: 'The respective part played in the development of arterial hypoxia by unequal ventilation-perfusion relationship and impaired diffusion of respiratory gases cannot be precisely defined'.

Selection for operation in such cases is extremely difficult, but studies, before and after surgery, have been performed.[24] Extensive pulmonary emphysema with bullae appears to be not specially suited for surgical treatment if the combined defects produce arterial hypoxia and the disability is both a ventilatory and an alveolar respiratory insufficiency,[24] but some satisfying results were obtained in group IIIa of Baldwin *et al.*[26]

Such patients should be referred for exhaustive study of the physiological mechanisms operative in the individual case and should not summarily be considered as beyond operative treatment only on the apparently hopeless clinical findings.

Case 8

A European male, 60 years old, with a history of chronic bronchitis of several years' standing was admitted with an acute respiratory infection. An X-ray of the chest, previously taken from the outpatient department, confirmed the clinical diagnosis of emphysema. Shortly after his admission his blood pressure fell and he became severely cyanosed. On administering oxygen his respiratory rate decreased.

O_2 saturation	58% (28 March 1962)
pH	7·32
PCO_2	51 mm.Hg
Standard bicarbonate	27 mEq./l.

As the hypoxia appeared to be disproportionate to the CO_2 retention and only slight acidaemia was present, it was suggested that oxygen could be administered. Even if the ventilation decreased somewhat the resulting increase in CO_2 would cause a further drop in pH, but there would still be some margin of safety because a pH of above 7·20 and a PCO_2 of below 60 mm. are usually well tolerated. The administration of 100% oxygen resulted in the following findings (29 March 1962):

O_2 saturation	88%
pH	7·21
PCO_2	76 mm.Hg
Standard bicarbonate	27 mEq./l.

Oxygen was then administered intermittently over the next 24 hours and a subsequent analysis (30 March 1962) revealed:

Art. O_2 saturation	79·5%
pH	7·50
PCO_2	61 mm.Hg
Standard bicarbonate	44 mEq./l.

These findings are summarized in Fig. 10, showing the development of an acute respiratory acidaemia and its compensation by an increase in standard bicarbonate (respiratory acidosis).

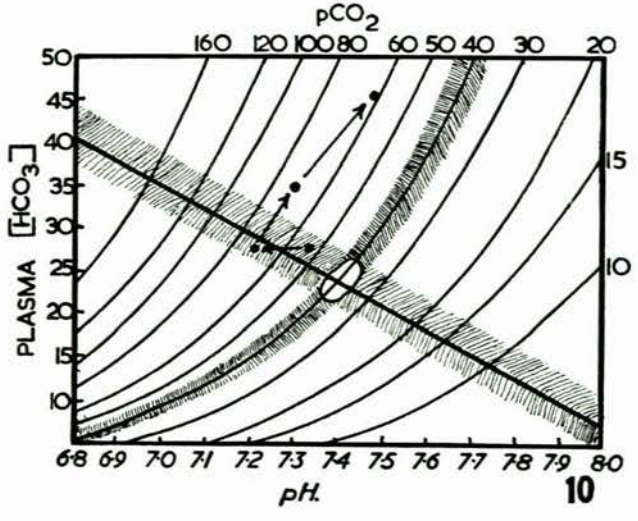

Fig. 10. Standard HCO_3, pH and PCO_2 set out in the diagram of Nunn[29] to illustrate the development of a respiratory acidaemia and the secondary response leading to a respiratory acidosis (compensated acidaemia).

CONCLUDING REMARKS

This analysis shows that at present most cases referred to the pulmonary physiology laboratory are referred to obtain a quantitative evaluation of an abnormality clinically evident in most cases. This results in an uneconomical use of time and labour which could be prevented by the use of a few simple and quickly performed screening tests. The value of arterial blood-gas analysis cannot be overstressed and should probably be included as a screening test. Hypoxia, between 80 and 90% saturation, appears seldom to be diagnosed on clinical grounds. For adequate management of hypoxia the pH, PCO_2 and standard bicarbonate should be known. We agree with Fletcher[27] that 'the clinician has no more justification for the diagnosis and treatment of hypoxaemia without laboratory guidance, than he has for the diagnosis and treatment of anaemia simply on the basis of pallor'.

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