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UNILATERAL LUNG TRANSRADIANCY: A PHYSIOLOGICAL STUDY

BY

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This paper describes three patients who presented with the clinical and radiographic picture of increased transradiancy of one lung. Each case bore a close resemblance to those studied by Macleod (1954). A satisfactory explanation of the phenomenon has not yet been made. Microscopical examination of the abnormal lung has been limited to descriptions by Swyer and James (1953) and Dornhorst, Heaf, and Semple (1957). Observations on the functional impairment of the abnormal lung are few (Dornhorst *et al.*, 1957), so an attempt has been made in this group to investigate this aspect by physiological methods applicable to pulmonary disease in man.

CASE REPORTS

CASE 1.—A machinist, aged 34 years, was referred for investigation after recovering from an acute respiratory infection. A chest radiograph taken by the Sheffield M.M.R. Unit had revealed abnormal transradiancy of the right lung.

At the age of 5 years he suffered from pneumonia and since had been prone to recurrent attacks of bronchitis. He usually had a morning cough and a little mucoid sputum. Physical examination revealed a well-developed man in whom abnormal findings were confined to the chest. Diminished expansion, hyper-resonance, weak breath sounds, and fine inspiratory rales were found over the whole of the right lung. Radiography showed mild reduction in size but increased transradiancy of the right lung, which appeared to be oligaemic. Fluoroscopy showed normal ventilation of the left lung, whereas aeration and de-aeration of the right lung was negligible. The inspiratory phase was associated with movement of the heart towards the right, while during expiration the mediastinum was displaced towards the left. A radiograph taken in full expiration showed air-trapping, affecting the whole of the right lung. The right diaphragm showed restricted movement but was not paralysed.

Bronchoscopy showed that the main bronchi were normal. Bronchography revealed slight distortion of the bronchial pattern of the right lung. The small peripheral branches of the right middle and lower lobes did not fill completely compared with the normal distribution and pattern of the left bronchial tree. There was no bronchiectasis (Fig. 1).

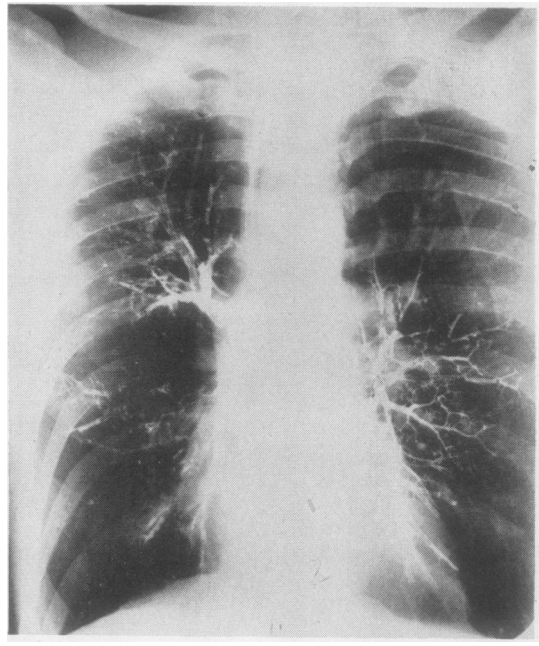

FIG. 1.—Case 1: Postero-anterior bronchogram showing some distortion and poor filling of some of the peripheral branches of the right middle and lower lobes.

Pulmonary angiography showed that the right pulmonary artery was of normal size, but its major branches to the three lobes were reduced in calibre and the number of the small branches throughout the whole of the right lung was much diminished (Fig. 2).

CASE 2.—A crane driver, aged 48 years, had no significant symptoms until 18 months before he was referred to hospital in April, 1957. He had a severe attack of influenza when he was 17 and from then onwards had a mild winter cough. Towards the end of 1955 he noticed breathlessness following a series of colds. He also complained of a dull aching pain across the front of the left side of the chest.

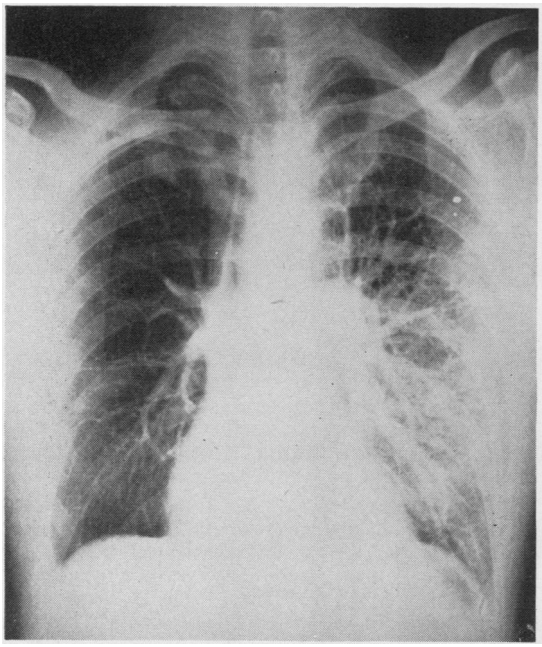

FIG. 2.—Case 1: Angiogram 2.5 seconds after injection showing the contrast between the under-filled right pulmonary artery and the over-filled left side.

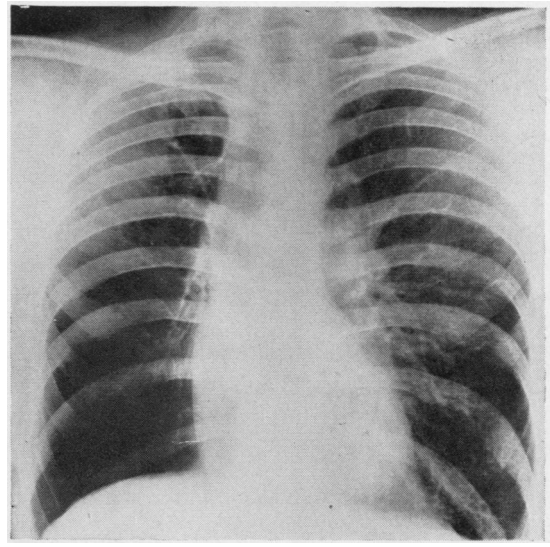

FIG. 3.—Case 2: Postero-anterior radiograph showing the reduced size and relative transradiancy of the right lung. The right hilar shadow is smaller and the left larger than normal.

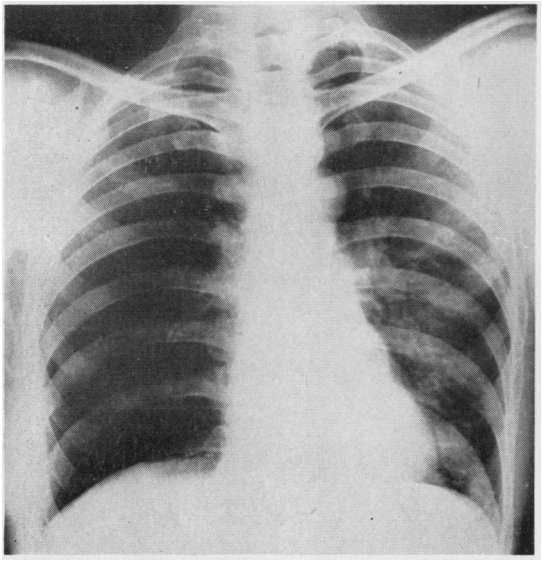

FIG. 4.—Case 2: In full expiration the mediastinum is displaced to the left and air is trapped in the right lung.

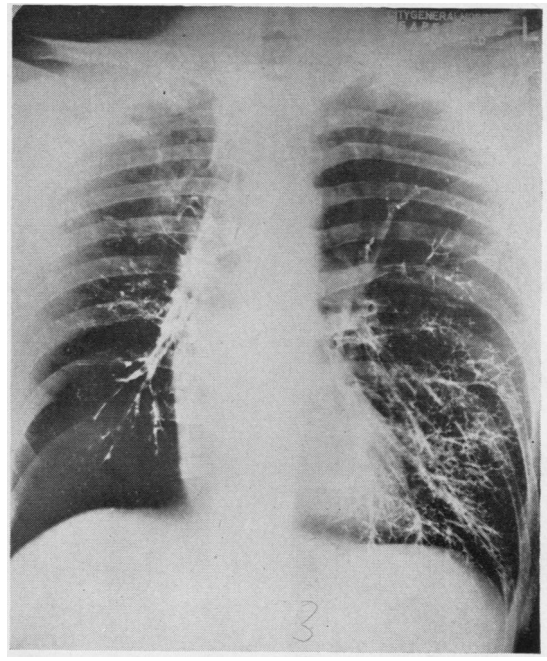

FIG. 5.—Case 2: Postero-anterior bronchogram showing poor filling of the peripheral branches of the right bronchial tree.

On examination there were diminished expansion, hyper-resonance, faint breath sounds, and inspiratory rales over the right side of the chest. There were no rhonchi and the left lung appeared normal. Other systems were normal.

A chest radiograph showed a small right lung with marked transradiancy except at the apex. With the exception of the upper lobe, the vessels appeared small and unusually straight (Fig. 3). Apart from rather large pulmonary vessels the left lung was

normal. Fluoroscopy and a radiograph taken on deep expiration showed air being trapped in the right lung, associated with marked mediastinal displacement towards the left (Fig. 4). The left lung filled and emptied normally. The right diaphragm moved poorly and this appeared related to the limited ventilation of the right lung.

Bronchoscopy showed normal main bronchi. Bronchography indicated some distortion of the right bronchial tree with elevation of the upper lobe bronchi from mild contraction of this lobe. Filling of the peripheral branches was incomplete in contrast to the normal pattern of the left lung. There was no bronchiectasis (Fig 5).

Pulmonary angiography confirmed an overall reduction in perfusion of the right lung. The right branch of the pulmonary artery was not well visualized, but the calibre of its large and small branches to the right middle and lower lobes was diminished. The vascular pattern of the left lung was normal.

CASE 3.—A housewife, aged 24 years, was referred to hospital for investigation in January, 1958, after an attack of right upper lobe pneumonia which had responded to antibiotic therapy. She gave a history of pneumonia in childhood which was followed by mild bronchitis until the age of 14 years. In 1952 and 1954 she had a dry left pleurisy. A chest radiograph in August, 1952, had revealed increased transradiancy of the left lung.

On examination there was diminished expansion of the left side of the chest over which breath sounds were weak and associated with persistent fine inspiratory rales confined to the left lower lobe and the lingula.

A chest radiograph showed that the left lung was rather small and abnormally transradiant as though from relative oligaemia (Fig. 6). On fluoroscopy the right lung filled and emptied freely and to a normal extent whereas the left lung failed to empty even in forced expiration. At this phase the mediastinum was displaced greatly towards the right, but there was little upward movement of the left diaphragm (Fig. 7).

On bronchoscopy, the major bronchi appeared normal. Bronchography revealed a normal distribution of the left bronchial tree and scattered, fusiform dilatation of some of the smaller branches of the left lower lobe and the lingula. The usual peripheral filling was not apparent. The right bronchial tree was normal (Figs. 8 and 9).

Pulmonary angiography showed marked reduction in calibre of the vessels of the left lung. The change affected the lobar arteries as well as the small peripheral branches. The oligaemia contrasted forcibly with the over-filled appearance of the right lung (Figs. 10 and 11). A radiograph taken at a later phase of circulation did not show an enlarged or abnormal bronchial artery supply to the left lung.

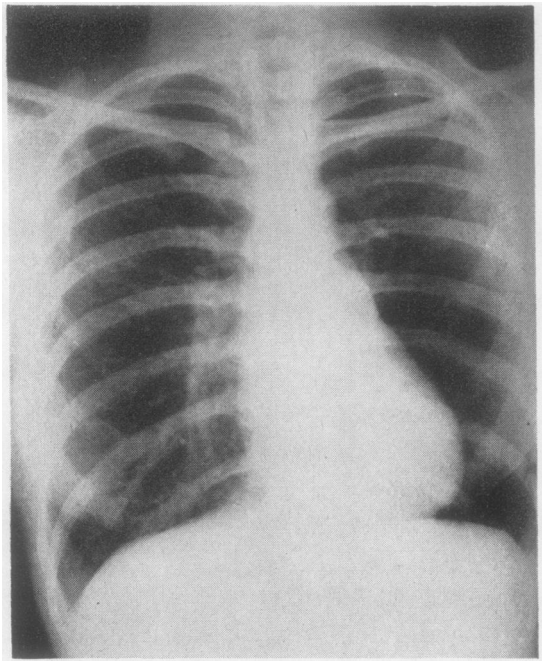

FIG. 6.—Case 3: Postero-anterior radiograph showing diminished volume and relative transradiancy of the left lung. Note the contrast between the oligaemic appearance of the left lung and the normal vascular pattern of the right side.

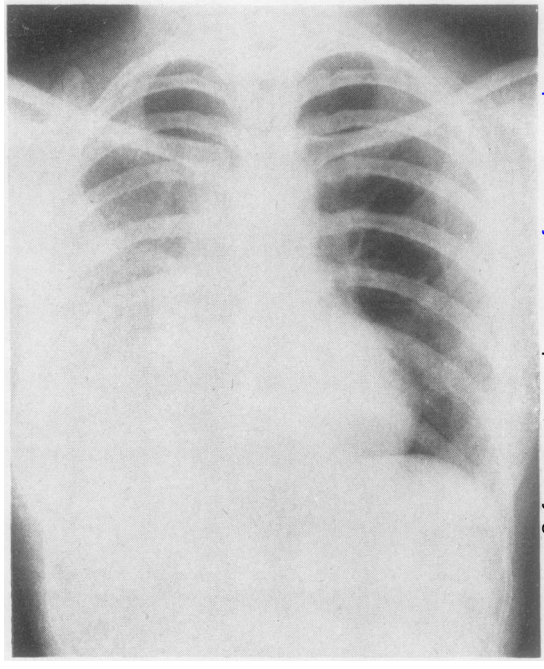

FIG. 7.—Case 3: In full expiration the left lung remains relatively transradiant.

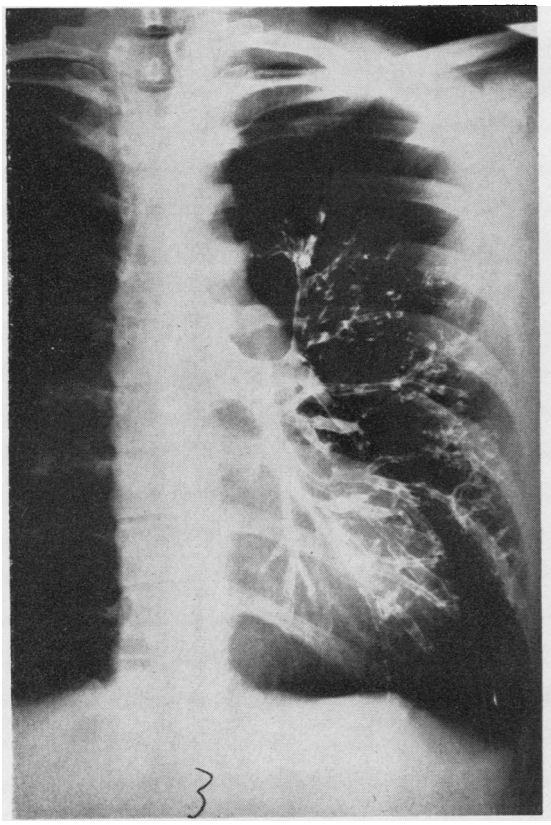

FIG. 8

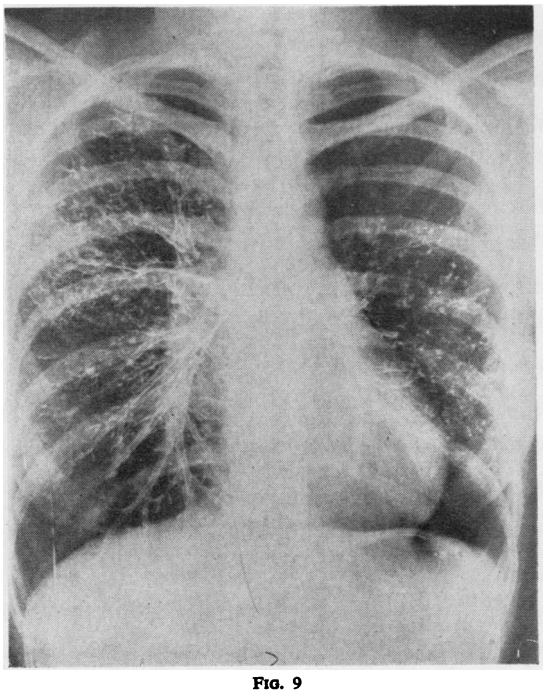

FIG. 9

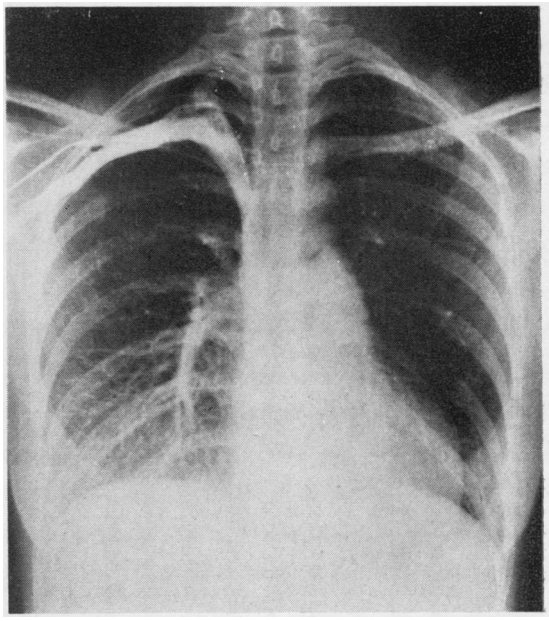

FIG. 10

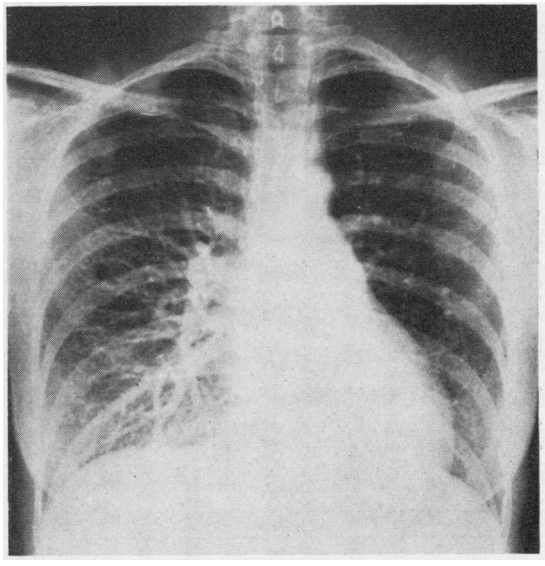

FIG. 11

FIG. 8.—Case 3: Antero-posterior bronchogram of the left lung showing normal distribution with mild dilatation of some of the branches of the left lower lobe and lingula.

FIG. 9.—Case 3: Postero-anterior bronchogram showing a normal appearance of the right bronchial tree.

FIG. 10.—Case 3: Angiogram 2.5 seconds after injection showing the contrast between the normal size, distribution, and filling of the right pulmonary artery with the small left pulmonary artery and branches.

FIG. 11.—Case 3: Angiogram 5 seconds after injection showing the contrast between the well-filled venous channels of the right lung with the oligaemic appearance of the left side.

Physiological Studies

The physiological investigation can be conveniently divided into four parts: (1) An assessment of combined function of the two lungs; (2) a study of the differential function of the two lungs; (3) an investigation of air flow into and out of each lung under general anaesthesia with muscular relaxation; (4) blood gas analysis.

(1) Combined Function of the Two Lungs.—Studies of overall function of both lungs were made on each patient. The lung volumes and mixing efficiency were estimated by the closed circuit method using helium in air as the mixing gas (Bates and Christie, 1950). Ventilatory function was estimated using a Gaensler vitalometer by measuring the percentage of forced vital capacity expelled in one second from which the indirect maximum breathing capacity (M.B.C.) can be predicted (Gandevia and Hugh-Jones, 1957). Lung compliance was studied from tracings of flow rate, oesophageal pressure, and tidal volume. These were recorded simultaneously on a Cambridge multichannel unit. Measurements of oesophageal pressure were made using an air-filled balloon 0.6 cm. in diameter (Mead, McIlroy, Selverstone, and Kriete, 1955) attached by a catheter of 1 mm. bore to a water-filled manometer. The flow rate was obtained from measurements of the changes in pressure across the diaphragm of a Lilly pneumotachygraph. The outlet of the pneumotachygraph was attached to a Kennedy light-weight spirometer from which recordings of tidal volume were taken during periods of quiet respiration. Construction of pressure volume loops gave a measurement of lung compliance.

The results of these procedures are given in Table I. Ventilatory function and mixing qualities showed mild impairment although the vital capacity was not reduced markedly.

Table I
COMBINED FUNCTION OF THE TWO LUNGS

	Case 1	Case 2	Case 3
F.V.C.% of predicted	95·0	89·0	88·0
Indirect M.B.C.% of predicted	50·7	50·4	54·1
R.A./T.L.V. (%)	44·5	49·3	49·0
Mixing	Fair	Fair	Poor
Compliance (l./cm. H_2O)		9·12	

(2) Differential Function of the Two Lungs.—The individual function of each lung was determined using a Carlens catheter. The pharynx, larynx, and trachea and main bronchi were anaesthetized with about 8 ml. of 4% "lignocaine." A Carlens catheter was then introduced. This permitted assessment of lung volumes, oxygen uptake, minute ventilation, and mixing efficiency to be made on each lung. The results are given in Table II. In the abnormal lung minute volume, oxygen uptake and vital capacity were greatly diminished. The residual air was relatively increased and mixing was grossly impaired. During this part of the investigation it was noted that occlusion of the airway from the normal lung was immediately followed by considerable distress, violent ventilatory efforts, and a marked increase in oesophageal pressure. Occlusion of the airway from the abnormal lung was tolerated without measurable change in ventilation or oesophageal pressure.

Table II
DIFFERENTIAL FUNCTION OF THE TWO LUNGS

	Case 1		Case 2		Case 3	
	R.	L.	R.	L.	R.	L.
Bronchospirometry:						
Minute volume (% of total)	31·7	68·3	26·6	73·4	81·0	19·0
Oxygen uptake (% of total)	25·8	74·2	6·5	93·5	79·0	21·0
Vital capacity (% of total)	35·6	64·4	24·1	75·9	71·0	29·0
Helium mixing:						
R.A./T.L.V. (%)	53·5	35·0	80·0	55·8		
Mixing	Poor	Fair	Very poor	Fair		

(3) Air Flow Studies.—With the patient under general anaesthesia and with complete muscular relaxation the inflation of each lung by air under pressure was studied. General anaesthesia was induced with about 250 mg. of thiopentone. Complete muscular relaxation was obtained with succinylcholine (50–100 mg.). The Carlens catheter and the oesophageal balloon introduced during part 2 of the investigation were left *in situ*. The lungs were ventilated with oxygen from a Boyle's machine through the Carlens catheter after the onset of respiratory paralysis. Passive ventilation was discontinued to permit a series of airflow studies and an injection of succinylcholine preceded each of these.

The air flow studies were carried out as follows. A 14 lb. (6.4 kg.) weight was placed on the top of the spirometer bell producing a pressure of 14 cm. of water within the bell. The occluded spirometer outlet tube was attached to one side of the Carlens catheter. As soon as a satisfactory connexion was established the occlusion of the spirometer tube was abruptly released and a recording of the fall of the spirometer and the changes in oesophageal pressure was made. A number of readings were taken and the procedure

was repeated under similar conditions on the other lung. The Carlens catheter used had previously been tested for any difference in resistance to air flow between the right and left channels. None had been revealed. The results are given in Table III.

TABLE III
STUDIES ON INFLATION OF EACH LUNG BY WEIGHTED SPIROMETER BELL

	Case 2		Case 3	
	R.	L.	R.	L.
Time for inflation of each lung by the same volume:				
a. Other lung occluded, chest initially compressed			0·78 sec.	4·3 sec.
b. Other lung not occluded, chest not initially compressed	15·2 sec.	1·5 sec.		
Change in oesophageal pressure (cm. H_2O)				
a. As above			None	8 cm.
b. ,, ,,	None	+7 cm.		

In Case 2 the chest was not compressed before inflation and the lung not connected to the spirometer had a free airway. It was found that the time required to inflate the abnormal lung with a given volume of air was about 10 times that required when inflating the normal side with the same volume of air. No change in oesophageal pressure was noted on inflation of the abnormal lung, but a rise in oesophageal pressure of 7 cm. of water developed on inflating the normal lung. The flow rate of air out of the abnormal right lung was presumably insufficient to prevent a rise in oesophageal pressure when inflating the normal lung. This suggests an obstruction to egress of air from the abnormal lung. In addition the low rate of air entering when inflating the abnormal lung implies that an obstruction to air entering is also present in that lung.

In Case 3 the chest was compressed manually immediately before the weighted spirometer was attached to one side of the catheter. At the same time the airway to the other lung was occluded. The compression of the chest wall was relaxed at the same time as the occluded outlet tube of the weighted spirometer was released. The abnormal lung took about six times longer to receive a given volume of air than the normal lung. The oesophageal pressure became negative when inflating the abnormal lung: no similar negative pressure developed when inflating the normal lung. After manual compression of the chest recoil of the chest wall tends to produce a negative intrathoracic pressure. When the normal lung was connected to the spirometer air passed rapidly into the lung and counteracted this tendency to produce a negative pressure. When the abnormal lung was connected to the spirometer, air flow into the lung was too slow to counteract the recoil of the chest wall.

The investigation into air flow carried out in Cases 2 and 3 suggests that there is a high-grade obstruction to air entering and leaving in the abnormal lung. The procedure in Case 3 is important because the findings appear to exclude the concept of valvular air trapping within the abnormal lung. If a valvular obstruction with air trapping distal to the obstruction was present, the inflow of air from the weighted spirometer would be expected to prevent the development of a negative oesophageal pressure when the abnormal lung was inflated after release of chest compression.

(4) BLOOD GAS ANALYSIS.—Arterial blood samples were analysed. These were taken at rest and on moderate exercise. In Case 2 the arterial oxygen saturation was normal, but showed a mild reduction on exercise. The carbon dioxide tension was normal. The blood became acid on exercise, but this was not due to retention of carbon dioxide. The oxygen saturations were slightly lower in Case 1, but showed the same trend (Table IV).

TABLE IV
STUDIES OF BLOOD GASES

	Case 1		Case 2	
	At Rest	Exercise	At Rest	Exercise
*p*H	7·39	7·13	7·35	7·16
CO_2 content	40·95	27·19	45·0	38·5
O_2 saturation	90·5	84·2	97·0	91·5

DISCUSSION

The physical signs that were found in these three patients were such as have been noted in all descriptions of this disorder. There was diminished expansion and absent or weak breath sounds on the affected side. Fine rales were audible over the abnormal lung. There was no evidence of neuromuscular disorder, disease of the joints, or deformity of the thoracic cage. The radiographic features consisted essentially of relative oligaemia, marked ventilatory impairment, and mild reduction in size of one lung.

Studies of the ventilatory function of both lungs together indicated some impairment as illustrated by the reduction of the vital capacity and indirect M.B.C. The impaired mixing efficiency suggested some form of resistance to air flow preventing

normal ventilation of the lungs. Bronchospirometry confirmed that the impairment was unilateral, a decrease in ventilation being coupled with the diminished oxygen uptake.

Study of the lung volumes of the abnormal lung showed a reduced tidal volume and vital capacity but an increased residual volume in relation to the total volume of that lung. It was concluded that the loss of ventilatory function was not entirely, nor indeed mainly, due to the mild reduction in total volume of the abnormal lung but to obstruction to air flow. The grossly impaired mixing efficiency, insofar as it can be assessed by bronchospirometric technique, added further support to this conclusion.

From the studies on the anaesthetized patient it may be deduced that a high-grade obstruction to air entry and egress exists on the abnormal side and that it is responsible for the reduction in the overall ventilatory function. Gross changes in the pleura and thoracic wall could hardly account for such a severe impediment to air flow and indeed are excluded by the clinical and radiographic findings of normality in these structures. The obstruction to inflation and deflation cannot be placed in the major bronchi or segmental divisions because of the absence of narrowing on bronchoscopy and bronchography. There is no evidence to support the notion of a valvular obstruction since the physiological studies suggest that there was obstruction to air flow both into and out of the abnormal lung.

The only alveolar abnormality which might possibly account for the abnormal physiology would be rigidity of the alveolar walls. This would imply gross thickening and consequent inadequate blood aeration. The known histological findings and the virtually normal arterial blood gas studies at rest and on exertion indicated that this hypothesis was most unlikely.

It is probable that the site of obstruction is at the level of the smaller air passages, and on physiological grounds it could be due to: (1) a diminution in calibre or number or both calibre and number of such passages; (2) an increase in their length; and (3) an alteration in the type of division of the smaller air passages associated with increased turbulence of air flow. It is common to find poor filling of the peripheral air passages on bronchoscopy of the abnormal side and this may be of importance.

The reason for the diminished vascularity of the lung is not clear. However, it is remarkable that perfusion by air and blood in the affected lung is reduced by the same degree. This raises the question of a compensatory reduction in perfusion by blood secondary to the poor ventilation. Since there is no significant shunt of poorly aerated blood and the condition is virtually symptomless, surgical removal of the affected lung is not indicated.

Various hypotheses have been put forward to explain the condition of unilateral lung transradiancy. One ascribes it to a congenital abnormality with poor development of the arterial supply and a defective condition of the smaller bronchioles. Another postulates that the primary abnormality is an obstructive condition of the peripheral air passages with an associated reduced blood perfusion. The physiological findings are compatible with these hypotheses. A further suggestion is that there is loss of rigidity of the peripheral air passages, which tend to collapse in expiration, thus producing a valvular obstruction: the evidence from studies of air movement into and out of the affected lung whose respiratory musculature had been paralysed would appear to exclude this.

Dr. N. Oswald has kindly given us permission to describe the following details of a patient with unilateral lung transradiancy under his care at St. Bartholomew's Hospital, London.

The patient, having had a cough since birth, presented at the age of 18 months in June, 1936, with pneumonic consolidation affecting the whole of the left lung. This took at least 45 days to resolve. Four months later the child was found to have reduced movement, impaired percussion note, and medium rales all over the left side of the chest; the Mantoux test was negative. A chest radiograph taken one year later showed the left lung to be more transradiant than the right. This radiographic finding persisted, and diminished movement and reduced air entry over the left chest were noted clinically. A bronchogram in 1942 showed normal appearances of the major bronchi but poor filling of the peripheral bronchi on the abnormal side. The Mantoux test was still negative.

The age of 3 years, when the condition was first recognized, is noteworthy in this patient. It may be that the severe and prolonged pneumonia was causative. Two of the patients on whom we carried out physiological assessments gave a history of pneumonia in childhood (Cases 1 and 3). Case 2 had a severe attack of influenza at the age of 17 years. All this seems to constitute suggestive evidence for such pneumonic episodes being causative. The condition of unilateral lung transradiancy does not appear to predispose to

attacks of pneumonia on the affected side. Indeed, one patient (Case 3) was first seen in the convalescent period after pneumonia affecting the normal lung. It seems possible that the smaller air passages of a child could be damaged by a severe pneumonia and subsequently fail to develop normally. If this were the case the reduced perfusion by blood in the affected lung could be associated with the decreased ventilation and physiological importance of that lung. However, a congenital basis for the condition cannot be definitely excluded.

Summary

Physiological investigations in three patients with the clinical and radiographic features of increased transradiancy of one lung have been described. A review of the current hypotheses concerning the condition has been made in the light of the physiological findings. This inclines us to believe that there is a non-valvular obstruction of the peripheral air passages in the affected lung. It seems that pneumonic episodes in childhood may affect the developing lung, unilateral lung transradiancy ensuing thereafter.

Our thanks are due to Dr. N. Oswald for permission to describe the details of a case under his care, to Dr. R. R. W. Hill for his skill in anaesthesia, and to Dr. K. Abbott for the radiographic investigations.

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