Lung Perfusion Scanning in Coal Workers Pneumoconiosis[1,2]

ANTHONY SEATON, N. LEROY LAPP, and C. H. JOSEPH CHANG

SUMMARY

Thirty-seven miners and ex-miners were studied clinically, physiologically, and by lung perfusion scanning. All 14 with complicated pneumoconiosis had avascular zones in relation to conglomerate masses and bullae. A fissure sign related to nodules and emphysematous bullae was present in one. There were several reasons for assuming that avascular zones were due to vascular obliteration rather than intrapulmonary shunting. Among 21 miners with simple pneumoconiosis, only 2 had avascular zones related to nodules rather than to other causes of hypoperfusion. The results of these studies accord well with previous pathologic studies of the pulmonary vasculature in pneumoconiosis.

Introduction

The initial lesion in coal workers pneumoconiosis is an accumulation of coal dust around the respiratory bronchioles (1, 2) with the production of focal emphysema. At autopsy relatively few subjects with simple pneumoconiosis show significant changes in the vascular bed of the lung; however, obliteration of pulmonary vessels is common in complicated pneumoconiosis (3), and some workers have suggested that the pulmonary capillary bed is reduced in some subjects with simple pneumoconiosis (4). The present study was carried out to determine whether avascular areas could be detected in the lungs of coal miners by perfusion lung scans.

(Received in revised form October 13, 1970)

1 From the Appalachian Laboratory for Occupational Respiratory Diseases, Department of Health, Education, and Welfare, U. S. Public Health Service, Bureau of Occupational Safety and Health; and the Departments of Medicine and Radiology, West Virginia University Medical Center, Morgantown, West Virginia 26506.

2 Mention of commercial concerns or products does not constitute endorsement by the Public Health Service.

Materials and Methods

Thirty-seven volunteer miners and ex-miners from the mining population of West Virginia and Pennsylvania, many in the process of seeking industrial injury compensation, were studied. All but 5 had symptoms of chronic bronchitis, and all but 2 had radiographic evidence of pneumoconiosis. Fourteen had complicated pneumoconiosis (progressive massive fibrosis), and the other 21 had simple pneumoconiosis. Clinical and physiologic data are recorded in table 1.

After preparation with Lugol iodine, all subjects were intravenously injected with 300 μc of a commercially available suspension of aggregated albumin-^{131}I while breathing quietly in the seated position. Three photoscans, one each in the anteroposterior and right and left lateral positions were obtained using a Pho-Dot scanner[3] with a 19-hole collimator focused to 6.8 cm for 100 per cent resolution. In addition standard six-foot posteroanterior and lateral radiographs of the chest

3 Model #1735, Nuclear Chicago Corp., Des Plaines, Illinois.

TABLE 1

CLINICAL, PHYSIOLOGIC, RADIOGRAPHIC, AND LUNG SCAN DATA FOR 37 COAL MINERS AND EX-MINERS WITH PNEUMOCONIOSIS

Patient No.	Age (years)	Years Worked Underground	Cigarette Smoking (pack years)	FEV_1/FVC (%)	RV/TLC (%)	DL_{CO} (% normal)	Qs/Q (%)	VD/VT (%) Rest	VD/VT (%) Exercise	PAP (mm Hg)	Radiographic Category	Other Abnormalities	Additional Findings on Scan
1	57	37	60	68	65		8.4	52			B 2n	Bullae	
2	42	16	0	70	44	69	8.4	51	44	20	C 3m		Peripheral underperfusion
3	66	36	80	62	44	58	7.5	45		27	B 2p		
4	62	32	15	24	57	33	5.8	38	37		A 1m	Bullae, probable silicosis	
5	57	22	21	54	40	54	3.2	53		11	B 2p	Bullae, bilateral UL fibrosis	LL underperfusion
6	77	24	64	53	70	52	7.9	61			B 1m	Bullae	Fissures
7	68	33	0	60	39	35	10.1	56		28	B 2p		Underperfused R lung
8	59	23	72	69	37	59			28		B 2m		
9	64	38	104	27	37	40	7.9	58			B 2t	Bullae	
10	58	10	42	52	80		7.2	58	48		C 3p		
11	64	16	70	46	47	61	6.5	53	32		B 1n	Silicosis, UL fibrosis	
12	63	50	3	74	63	56	6.4	40	37		A 2m		
13	58	34	33	69	62	37	5.0	57	50	30	C 2n	Silicosis, pleural thickening	
14	54	15	58	51	56	31	6.3	63	41		C 2p	Bullae	
15	57	32	0	80	41	73	8.4	47		12	2m	Old RUL TB	(a) RUL
16	58	25	0	84	55	84	7.8	53		14	3n	Early conglomerate RUL	(a) RUL
17	61	0*	41	73	46	73	10.0	57	33		1m	Old LUL TB, pleural thickening R apex	(a) Bilateral UL
18	69	43	0	78	40	52	6.0	10	37		2m	Old RUL TB	(a) RUL
19	55	33	17	75	36	54	6.7	55	38		1m	Old RUL TB	(a) RUL
20	71	44	0	85	52	45	6.7	30	36		2p		
21	49	25	0	83	28	76	6.2	47	28		1m	Pulmonary infarct, R base	(b) R base
22	47	23	0	92	46	66	8.6	17	20	17	1m		Basal underperfusion
23	64	47	37	72		63					2n		
24	59	19	3	76	25	63					1t		
25	52	24	0	85	40	93					3n		(a) Apices
26	46	26	0	71	27	72					2m		
27	60	40	0	81	26	65	3.3	56	42	13	1p		
28	66	29	0	60	76					22	1p	Kerley lines	
29	51	33	4	61	63	35	6.0	52	25		1p		
30	64	42	0	81	46	47	4.0	42	37		1p		
31	57	36	24	82	54	81	6.2		36		1p		
32	60	38	66	79	28	67				18	1m		
33	52	43	3	88	34	86	6.2	50	30		1m		
34	60	44	42	67	67	67	5.6	47	30		O		
35	59	24	33	53	59	36					O	PAH	(c) Bilateral, even flow
36	59	18	54	40		143	22.0	67		65	1t	PAH; PVH	Basal underperfusion
37	43	12	10	65	62	100	6.1	47		20	1t		

*Surface tippleman.

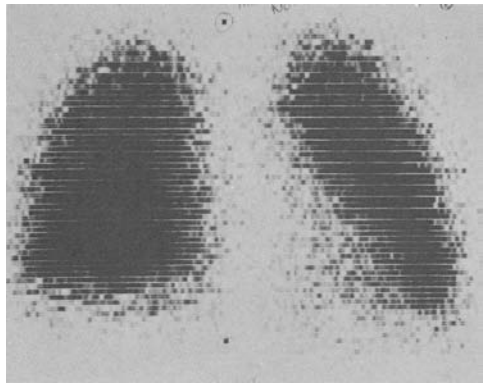
Key: FEV_1 = forced expiratory volume in one second. FVC = forced vital capacity. RV = residual volume. TLC = total lung capacity. DL_{CO} = diffusing capacity of the lung for carbon monoxide. Qs/Q = ratio of venous admixture or venoarterial shunt to total pulmonary blood flow. VD/VT = ratio of dead space to tidal volume. PAP = mean pulmonary arterial pressure at rest. UL = upper lobe. LL = lower lobe. (a) = area of diminished activity surrounded by areas of higher activity. (b) = peripheral crescents. (c) = fissure sign. PAH = pulmonary arterial hypertension. PVH = pulmonary venous hypertension.

were obtained and categorized according to the UICC[4]/Cincinnati Classification of the Radiographic Appearances of Pneumoconioses (5). The pulmonary function tests performed included spirometry and measurement of lung volumes in the constant volume body plethysmograph (6), arterial blood gases, and physiologic dead space. The venous admixture was determined while the patients breathed 100 per cent oxygen. Pulmonary diffusing capacity was estimated by the steady state method (7), normal values for which were those of Connor (8). Thirteen patients underwent right heart catheterization.

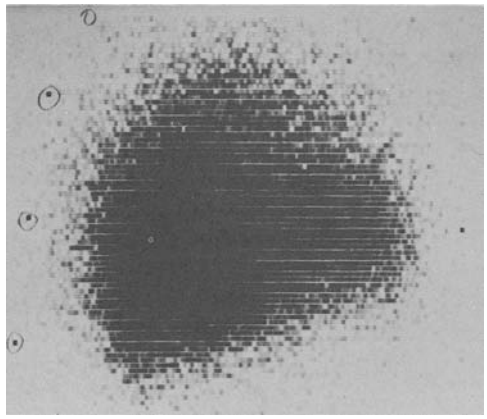
For every patient, detailed medical and occupational histories were taken. Duration of dust exposure and cigarette consumption are recorded in table 1. Evidence of cardiac disease was found in only one patient (*Patient 35*). Bronchitis, as determined by the responses to a modified questionnaire developed by the Medical Research Council of Great Britain (9), was present in all but 5 subjects (*Patients 3, 8, 15, 27,* and *33*).

Scans were initially classified by two observers without knowledge of the chest radiograph as abnormal, equivocal, or normal. The abnormalities sought were: (*1*) areas of diminished activity surrounded by areas of higher activity; (*2*) localized peripheral indentations of diminished activity (crescents) (10); (*3*) fissure sign (11); and (*4*) absence of the normal gradual decrease in activity from lower to upper zones (12). The scans were then compared with the radiographs, and those previously equivocal were reclassified as normal or abnormal. Any abnormality appearing in

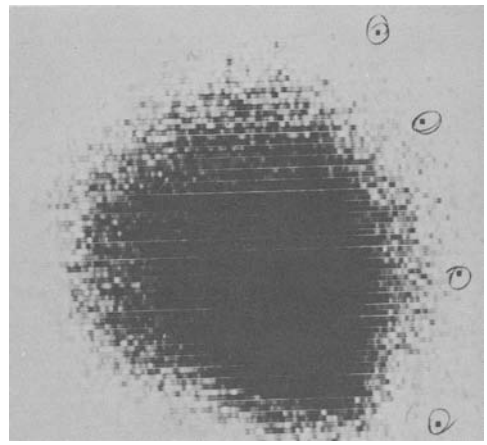
[4] Union International Contra Cancre.



A



B



C

Fig. 1. Typical normal pulmonary perfusion scans. A. Anteroposterior. B. Right lateral. C. Left lateral.

either the scan or the radiograph but not in both was recorded (table 1). A typical normal scan is shown in figure 1.

Results

In all 14 subjects with complicated pneumoconiosis the conglomerate masses in the chest films had been previously detected as perfusion defects in the scans (figure 2), even for *Patient 12,* who had an upper lobe lesion 11 mm in diameter (figure 3). In addition, 6 of the subjects had underperfused areas on the scans that represented bullae visible in the radiographs. In 4 subjects (*2, 5, 6* and *7*), however, zones of underperfusion that appeared relatively normal in the radiograph occurred in lungs containing large conglomerate lesions and probably represented "compensatory" emphysema (figure 4).

Ten of the subjects had physiologic evidence of airway obstruction, and all but one of the 13 in whom it was measured had an increased physiologic shunt. None of the 5 patients in whom catheterization was performed had a resting mean pulmonary arterial pressure greater than 30 mm Hg.

The 2 patients with category 3 simple coal workers pneumoconiosis had abnormal scans. One showed peripheral defects in the upper zones and a central perfusion defect in the right upper lobe. This patient had worked mainly as a motorman and probably had silicosis, although this diagnosis could not be made from the roentgenogram. Possibly he had early conglomeration in the right upper lobe. The other patient, who had a heavy nodular infiltrate in the upper zones, showed some underperfusion of the apexes (figure 5).

Three of the patients with category 2 pneumoconiosis had normal scans. The other 2 had zones of underperfusion in the right lung related to old fibroid tuberculosis. The perfusion defects appeared larger than the radiographic opacities. Neither patient had airway obstruction, but both had an increased physiologic shunt.

Of 14 patients with category 1 pneumoconiosis, 9 had completely normal scans, 2 in the presence of airway obstruction. Of the

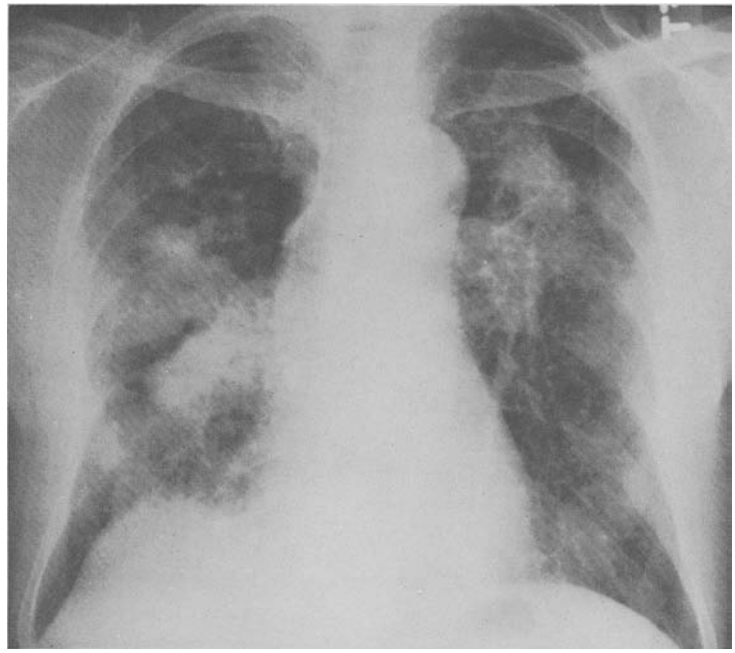
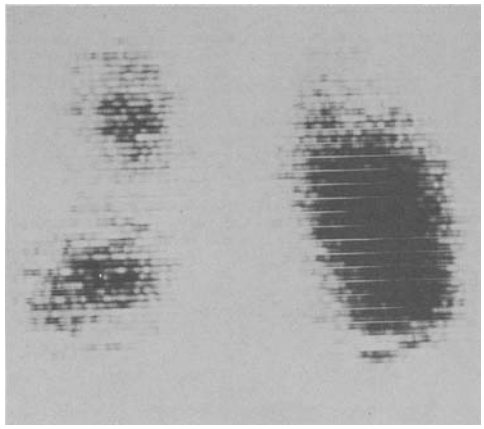
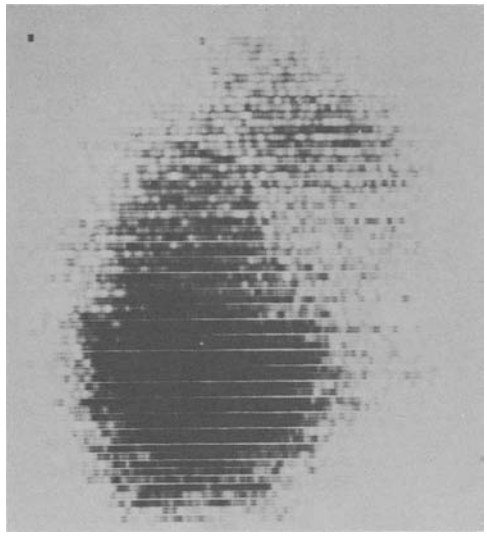


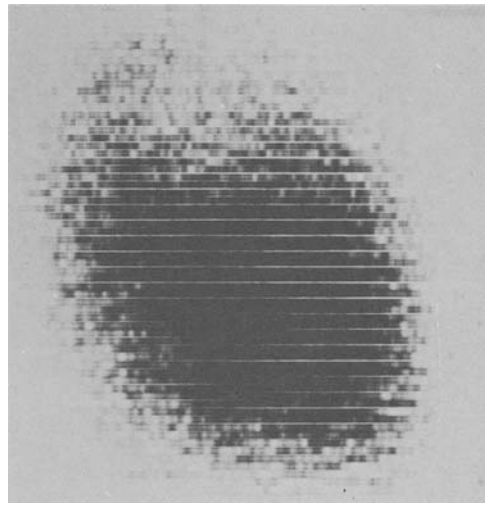
Fig. 2A. *Patient 3.* Posteroanterior radiograph showing category B complicated pneumoconiosis.



B



C



D

Fig. 2B, C, D. *Patient 3.* Anteroposterior (B), right lateral (C), and left lateral (D) scans showing avascular zones corresponding to areas of conglomeration.

remaining 5 patients, 2 had upper lobe fibrosis probably due to old tuberculosis, one had a radiographic shadow that appeared to be a pulmonary infarct, one had severe pulmonary arterial and venous hypertension with a reversal of the normal base-to-apex decrease in perfusion (13), and one had bilateral basal underperfusion in the scan related to no discernible radiographic abnormality.

Two remaining patients had no pneumoconiosis. One had a history of tuberculosis and severe chronic bronchitis; his scan showed bilateral fissure signs and an even distribution of perfusion, suggesting pulmonary arterial hypertension (11). The other subject had a normal scan.

Discussion

The characteristic pathologic lesion in coal workers pneumoconiosis is an accumulation of coal dust around the respiratory bronchioles associated with some surrounding fibrosis (1). Dilatation of the respiratory bronchiole and atrophy of the bronchiolar smooth muscle result in focal emphysema. In simple pneumoconiosis, however, obliterative changes are not usually found in the pulmonary vessels at autopsy unless there has been complicating chronic bronchitis or tuberculosis (2). By contrast, in complicated pneumoconiosis, the conglomerate nodules cause complete obliteration of pulmonary arteries and arterioles, both by invasion and erosion from without and by endarteritis within (3). Conglomerate masses have been shown to be surrounded by plexuses of wide-diameter capillaries (50 μ to 150 μ) constituting a potential shunt that would allow the passage of the macroaggregates used in this study (14). In lungs with complicated pneumoconiosis, both bullous and traction cysts and also compensatory emphysema are often present. These lesions also cause obliteration of the vascular bed and

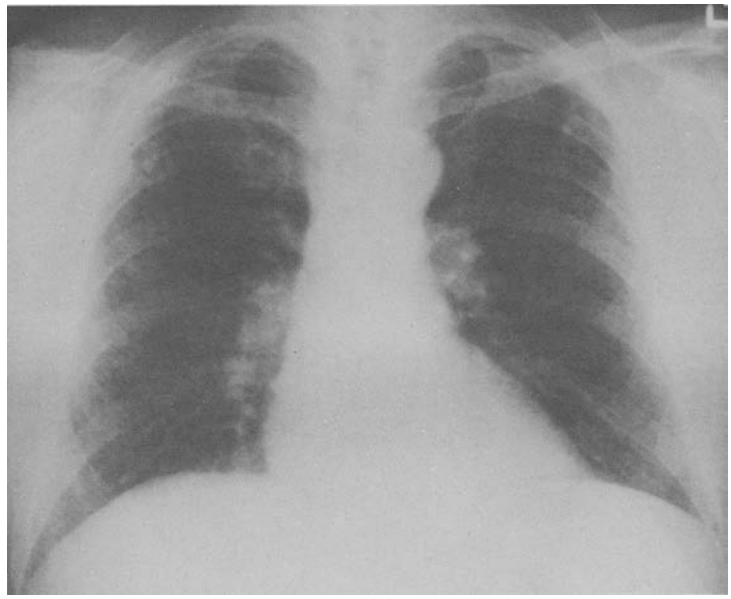
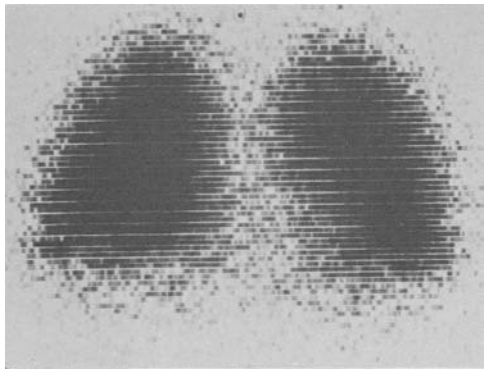
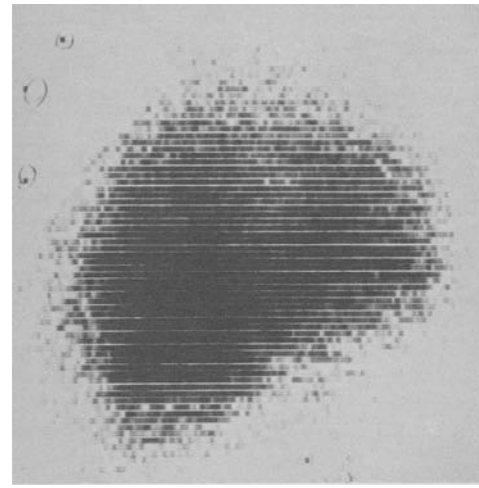


Fig. 3A. *Patient 12.* Posteroanterior radiograph showing early conglomeration in the right upper zone.



B



C

Fig. 3B, C. *Patient 12.* Anteroposterior (B) and right lateral (C) scans showing zone of underperfusion in the right mid-upper zone.

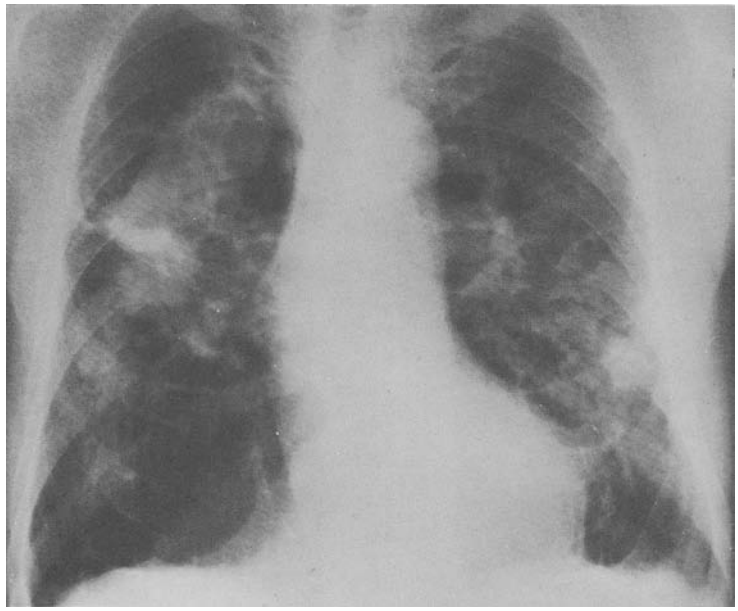
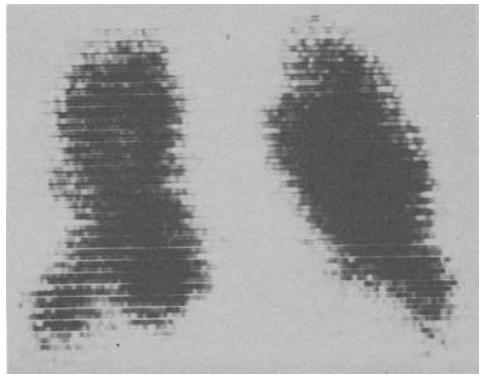
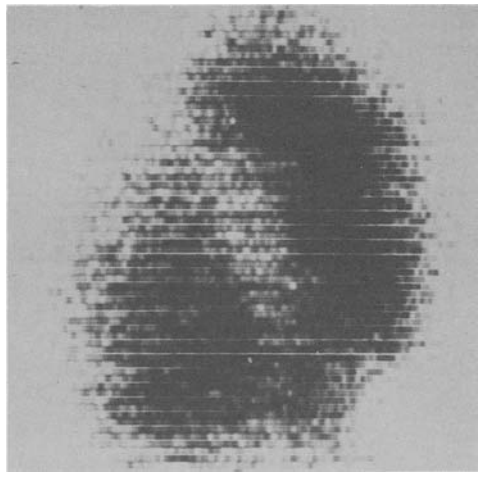


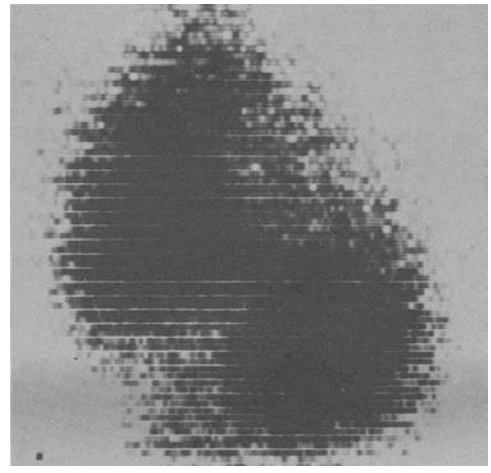
Fig. 4A. *Patient 6.* Posteroanterior radiograph showing category B complicated pneumoconiosis and basal bullae.



B



C



D

Fig. 4B, C, D. *Patient 6.* Anteroposterior (B), right lateral (C), and left lateral (D) scans showing avascular zones corresponding to conglomeration, bullae, and bilateral fissure signs.

contribute to the development of pulmonary hypertension in some subjects. If tuberculosis is present, there is also likely to be a zone surrounding the lesion where there is obliteration of arterioles by endarteritis (3).

Despite these pathologic data, it has been suggested on the basis of physiologic tests that simple pneumoconiosis can produce obliterative disease of the small vessels, which might be responsible for some of the symptoms (4). It is conceivable that arterial or arteriolar narrowing might be present in life when none can be demonstrated at autopsy. Such zones of underperfusion have been demonstrated in acute episodes of asthma by means of perfusion lung scanning (15). With this in mind, the present study was planned to investigate whether significant small vessel disease exists in coal workers with pneumoconiosis, using a combined radiographic and physiologic approach as advocated by Quinn (16).

The subjects studied were not a random sample, having been obtained from a population of hospital outpatients and claimants who either had appeared or were to appear before industrial injury compensation boards. They did, however, represent all grades of severity of both simple and complicated coal workers pneumoconiosis.

Three broad conclusions can be drawn from the study. First, in only 2 patients with simple pneumoconiosis was there an abnormality of perfusion that was not explicable on the basis of some other condition. It is, nevertheless, significant that abnormalities were present in one patient with very early conglomeration, indicating that vascular obliteration occurs early in the stage of conglomeration, and in another who had a heavy, nodular infiltrate involving especially the upper zones. This latter patient might have been similar to the subjects described by Caplan (17, 18) in whom nodular opacities were associated with the rheumatoid diathesis and in whom arteritis was observed in the pulmonary lesions (19). Neither of the 2 patients had obstructive

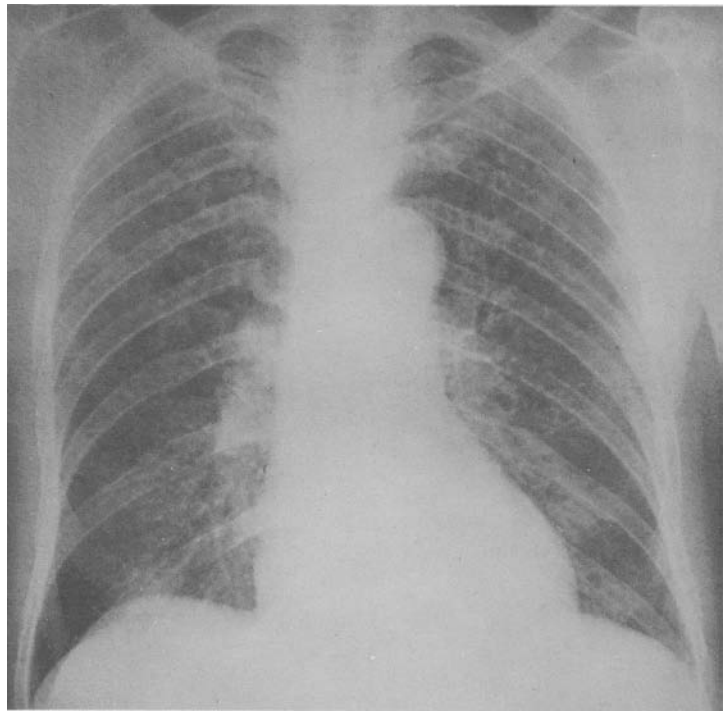


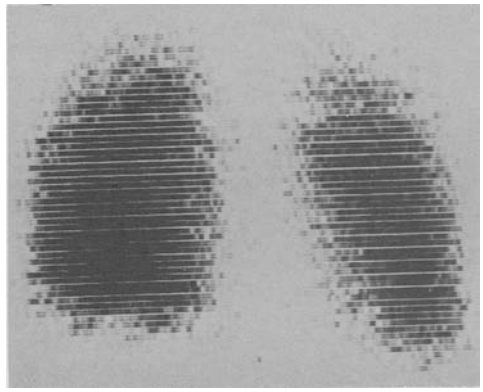
Fig. 5A. *Patient 25*. Posteroanterior radiograph showing category 3, or simple, pneumoconiosis.

airway disease or any other condition that might have caused the abnormality. The first patient, however, might have been exposed to silica in his work as a motorman, and the nodules of silicosis are known to encroach on blood vessels (20). It appears, therefore, that zones of hypoperfusion can occur occasionally in simple pneumoconiosis and might herald the onset of conglomeration.

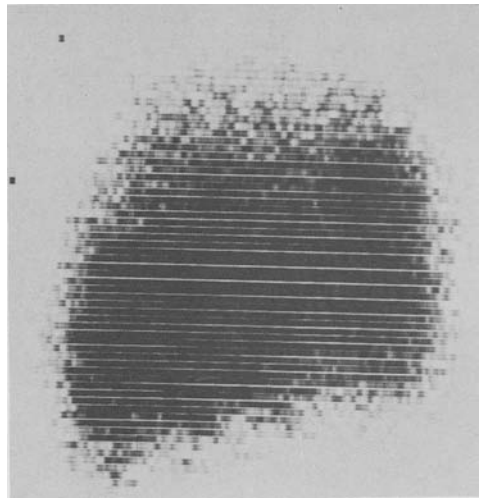
Second, in complicated pneumoconiosis avascular areas always appear at the site of nodules, their size being approximately the same by the two techniques. Even in the one very early case of conglomeration, an avascular area was present at the site of conglomeration. In theory, these avascular areas might be due to vascular obliteration, intrapulmonary shunting, or both. Although all but one of the patients had an increased physiologic shunt, in none was it of a size consistent with that of the avascular zone on the scan. These findings are compatible with arterial obliteration as the principal cause of the perfusion abnormality, as would have been expected from pathologic studies. The absence of radioactivity in the liver and spleen also support the absence of significant shunting through the dilated capillaries (14).

Third, bullae and compensatory emphysema, common in complicated pneumoconiosis, are often easier to detect in a scan than in a radiograph. If bullous emphysema is distributed on the lung surface, a fissure sign can be produced in the absence of pulmonary hypertension, as in *Patient 6* (figure 4). In the presence of a large fibrotic mass, "compensatory" emphysema of the rest of the lung can produce underperfusion of the whole lung, as in *Patients 4* and *7*.

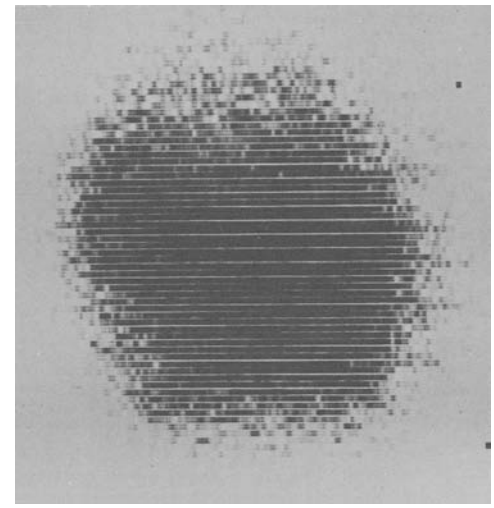
It should be pointed out that the scars of old tuberculosis can be associated with disproportionately large areas of underperfusion (21); this is consistent with what is known of the pathologic features of such lesions (3).



B



C



D

Fig. 5B, C, D. *Patient 25.* Anteroposterior (B), right lateral (C), and left lateral (D) scans showing apical underperfusion.

This study demonstrates the value of a combined clinical, physiologic, and radiographic approach in investigating the perfusion patterns of the lung in disease, because complete knowledge of the presence or absence of such lesions as airway obstruction, cardiac disease, or large intrapulmonary shunts assists greatly in the interpretation of avascular areas on the scan. The results indicate that the vascular bed of the lung in life is normal in most patients with simple pneumoconiosis and becomes abnormal in the presence of conglomeration and bullous disease. These findings are strikingly similar to those from detailed pathologic studies (3).

RESUMEN

Escrudiñamiento de la perfusion pulmonar en la neumoconiosis de oberos de carbon

Treinta y siete mineros y ex mineros fueron estudiados por medio del escrudiño de la perfusión pulmonar y una combinación de aproche clínico y fisiológico. Todos los catorce con neumoconiosis complicada tenían zonas avasculares en relación al conglomerado de masas y bullae. En uno de los pacientes se encontró un signo de fisura relacionado con los nódulos y con bullae enfisematosa. Se discuten las razones por las cuales se asume que las zonas avasculares fueron debidas a la obliteración vascular más que a la desviación intrapulmonar. Entre veintiún mineros con neumoconiosis simple, solamente en dos estuvieron presentes las zonas avasculares relacionadas con los nódulos más que con otras causas de hipoperfusión. Los resultados de estos estudios en mineros vivos están bien de acuerdo con los estudios patológicos previos de la vasculatura pulmonar en la neumoconiosis.

RESUME

Scanning de la perfusion pulmonaire dans des cas de pneumoconiose du mineur

Trente-sept mineurs et anciens mineurs ont été étudiés au moyen d'une méthode de scanning de la perfusion pulmonaire, et en ayant recours à un approche clinique physiologique combiné. Les 14 mineurs souffrant de pneumoconiose compliquée présentaient des zones non-vascularisées qui étaient en relation avec des masses agglomérées et avec des bulles. Chez un sujet, un signe de fissure était en relation avec des nodules et avec des bulles d'emphysème. On discute des raisons qui permettent de penser que les zones non-vascularisées sont produites par l'oblitération des vaisseaux plutôt que par un court circuitage intra-pulmonaire. Parmi les 21 mineurs souffrant de pneumoconiose simple, ce n'est que chez deux d'entre eux que les zones non vascularisées qui ont été observées pouvaient être mises en relation avec des nodules plutôt qu'avec d'autres causes connues d'hypoperfusion. Les résultats obtenus au cours de ces études chez des mineurs en vie concordent bien avec les études pathologiques antérieures qui ont été menées sur la vasculature pulmonaire dans la pneumoconiose.

References

1. Gough, J.: Pneumoconiosis in South Wales coal mines, Lancet, 1946, *1,* 462.
2. Heppleston, A. G.: The pathological anatomy of simple pneumoconiosis in coal workers, J. Path. Bact., 1953, *66,* 235.
3. Wells, A. L.: Pulmonary vascular changes in coal workers' pneumoconiosis, J. Path. Bact., 1954, *68,* 573.
4. Rasmussen, D. L., Laqeur, W. A., Futterman, P., Warren, H. D., and Nelson, C. W.: Pulmonary impairment in southern West Virginia coal miners, Amer. Rev. Resp. Dis., 1968, *98,* 658.
5. UICC/Cincinnati classification of the radiographic appearances of pneumoconioses; A cooperative study by the UICC Committee, Chest, 1970, *58,* 57.
6. DuBois, A. B., Botelho, S. Y., Bedell, G. N., Marshall, R., and Comroe, J. H.: A new method for measuring airway resistance in man using a body plethysmograph, J. Clin. Invest., 1956, *35,* 322.
7. Bates, D. V., Boucot, N. G., and Dormer, A. E.: The pulmonary diffusing capacity in normal subjects, J. Physiol. (London), 1955, *129,* 236.
8. Connor, P. J.: The measurement of the carbon monoxide diffusing capacity of the lung, Thesis, Graduate School of the University of Minnesota, August, 1960.
9. Medical Research Council: Definition and classification of chronic bronchitis for clinical and epidemiological purposes, Lancet, 1965, *1,* 775.
10. Wagner, H. N., Sabiston, D. C., McAfee, J. G., Tow, D., and Stern, D. S.: Diagnosis of massive pulmonary embolism in man by radioisotope scanning, New Eng. J. Med., 1964, *271,* 377.
11. Eaton, S. B., James, A. E., Greene, R. E., Lyons, J. H., Potsaid, M .S., and Fleishner, P. G.: New patterns of abnormal pulmonary perfusion associated with pulmonary emboli, J. Nucl. Med., 1969, *10,* 571.
12. West, J. B., and Dollery, C. T.: Distribution of blood flow and ventilation-perfusion ratio in the lung measured with radioactive CO_2, J. Appl. Physiol., 1960, *15,* 405.
13. Friedman, W. F., and Braunwald, E.: Alterations in regional pulmonary blood flow in mitral valve disease studied by radioisotope scanning, Circulation, 1966, *34,* 363.
14. Thomas, A. J., and James, W. R. L.: The right ventricle and small pulmonary arteries in coal workers, Brit. Heart J., 1958, *20,* 403.
15. Mishkin, F. S., Wagner, H. N., and Tow, D. E.: Regional distribution of pulmonary arterial blood flow in acute asthma, J. A. M. A., 1968, *203,* 1,019.
16. Quinn, J. L.: The lung: The challenge of nuclear medicine, Amer. J. Roentgen., 1969, *105,* 251.
17. Caplan, A.: Certain unusual radiological appearances in the chest of coal miners suffering from rheumatoid arthritis, Thorax, 1953, *8,* 29.
18. Caplan, A., Payne, R. B., and Withey, J. L.: A broader concept of Caplan's syndrome related to rheumatoid factors, Thorax, 1962, *17,* 205.
19. Gough, J., Rivers, D., and Seal, R. M. E.: Pathological studies of modified pneumoconiosis in coal miners with rheumatoid arthritis (Caplan's syndrome), Thorax, 1955, *10,* 9.

20. Spencer, H.: Pathology of the Lung, Pergamon Press, New York, 1962, p. 307.
21. Poulousa, K., Rebe, R. C., and Wagner, H. N.: Characterization of the shape and location of perfusion defects in certain pulmonary diseases, New Eng. J. Med., 1968, *279,* 1020.