HYPOVENTILATION COMPLICATING CHRONIC BRONCHITIS

CASE REPORT

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INTRODUCTION

In 1965 McNicol and Pride described in detail the case histories and pulmonary function tests of four patients, not grossly obese, with chronic bronchitis whose clinical picture was dominated by hypoventilation, polycythemia and cor pulmonale.[5] The chronic hypoxemia and hypercapnia were not adequately explained by their lung disease and were attributed mainly to unexplained underventilation of the lungs. The possibility that chronic hypoventilation, complicating chronic bronchitis (with its resulting severe blood gas abnormalities), may play a much more important part in determining cor pulmonale than the morphologic chronic bronchitis itself has only recently been emphasised.[1, 2] The present report describes a further case of this interesting group of patients.

CASE REPORT

A.C.T. A 51 year old male Chinese, first presented at this hospital in November 1965 with a history of cough with haemoptysis of 3 days' duration. Clinical examination revealed a B.P.= 130/80, with bilateral crepitations in the lung bases. There was no clubbing of the fingers. A chest radiograph showed "increased lung markings". He was though to be suffering from bronchiectasis and was followed up as an outpatient.

In May 1966 he was noticed to be plethoric and slightly cyanosed. The only significant finding clinically were a few crepitations in the lung bases. His weight was 166 lbs., and his haemoglobin was 16.4 gm. per 100 ml. The packed cell volume was 68% with a normal total white and platelet count. A bronchogram revealed minimal bronchiectatic changes in the right lower lobe.

He was seen again for haemoptysis in September 1967. A fourth heart sound was heard, but there was no evidence of heart failure. An electro-cardiogram showed right ventricular hypertrophy. The blood urea, serum electrolytes and bone marrow examinations were normal. An intravenous pyelogram did not reveal any abnormality.

In view of the uncertain aetiology of his polycythaemia, he was readmitted in July 1968 for further clinical assessment and investigation of his pulmonary function.

The patient was born in China and came to Singapore at the age of 18 to work as a labourer in a biscuit factory. He left his job after 2 years and has been working as a coffee merchant ever since. There is no history of any exposure to an industrial pulmonary hazard, nor is there a history of encephalitis or obesity in the family. He had bronchial asthma when he was 22; the asthmatic attacks subsided after a period of 9 years. Since then he has been free of asthma. He, however, began to develop a cough productive of about 1-2 ozs. of blackish sputum daily over the past 12 years which he attributed to smoking cigarettes (he has been smoking 10 cigarettes a day since he was 26). In 1954 he had a right pleural effusion which resolved. He noticed that his face was flushed over the past 10 years and that his memory was not as good as it used to be. He also had occasional morning headaches and somnolence for the past 3 years.

Except for the haemoptysis he has been well and has remained at work. He has no dyspnoea on exertion and keeps himself fit with daily physical exercises. Since January 1967 he has been having regular venesection for his polycythaemia.

Clinical examination showed a fairly tall (5′ $6\frac{1}{2}$″) well built man weighing 169 lbs. He had a plethoric facies and there was central cyanosis, but no ankle oedema or clubbing. The jugular venous pressure was not raised. The blood pressure was 130/85 mm. Hg. The heart was enlarged with dual rhythm. There were some crepitations over the right lung base. There was no papilloedema and no abnormalities were found in the rest of the examination.

RESULTS OF INVESTIGATIONS

The blood investigations, done in January 1967 showed a haemoglobin of 19.0 gms./100 ml., a packed cell volume of 62%, and normal total

white cell and platelet counts. The red blood cell volume was 39.2 ml./Kg. and the plasma volume was 23 ml./Kg.

The present haemoglobin (after a recent venesection) was 14.4 gm./100 ml. with a packed cell volume of 51%.

The chest radiograph (Fig. 1) revealed a large heart and main pulmonary arteries, with thickening at the right costo-phrenic angle.

The electrocardiogram (Fig. 2) showed a Grade 2 right ventricular hypertrophy, using the criteria of Goodwin and Abdin.[3]

Lumbar puncture showed a normal cerebrospinal fluid. The V.D.R.L. test was negative.

An electroencephalogram revealed no significant abnormality.

PULMONARY FUNCTION TESTS

Methods

The forced expiratory volume (F.E.V.) was recorded on the Collin's 6-litre Vitalometer. The maximum voluntary ventilation (M.V.V.) was then measured (using the same apparatus) over a period of 15 secs., with the patient breathing as hard and as fast as possible. Three inhalations of a bronchodilator aerosol (Medihaler-iso forte) were given and the F.E.V. repeated after a period of 20 mins.

The total lung capacity (T.L.C.) and its subdivisions were measured on the Collin's helium closed circuit residual volume (R.V.) apparatus. The mixing efficiency (M.E.) was calculated from fall in the helium concentration.

With the Cournand needle in situ in the brachial artery for 30 mins., resting ventilation was measured with a 3-min. collection of expired gas. The frequency of respiration was also noted. At the same time arterial blood was sampled over a period of 2 mins. The expired gas was subsequently analysed on the Scholander apparatus.

The patient was then told to hyperventilate for 2 mins. and another blood sample taken.

100% oxygen was then given to the patient to breathe for a period of 30 mins. Resting ventilation was measured using a gasometer over a period of 2 mins. and a blood sample taken.

The patient then inhaled a mixture of 95% O_2 and 5% CO_2 for 20 mins. and another blood sample taken simultaneously as the ventilation was measured over a period of 2 mins.

All blood samples were immediately analysed for arterial oxygen tension (PaO_2) using the Radiometer electrode E.5046 and for pH and carbon-dioxide tension ($PaCO_2$) on the Astrup apparatus, Type A.M.E1.

Physiological dead space was calculated from the Bohr equation, and the alveolar oxygen tension was obtained from the alveolar air equation.

All lung volumes are expressed in B.T.P.S. The predicted normal values for lung volumes and flow rates are from regression equations obtained from tests on 50 normal adult male Chinese in Singapore.[7] The rest of the predicted normal values are taken from the paper by McNicol and Pride.[5]

The increase in ventilation per mm. Hg. rise in the $PaCO_2$ and per 10^{-9} mol./litre rise in H ion concentration, together with the rise in $PaCO_2$ and [H+] required to double the resting ventilation were calculated.[5]

The standardised ventilation using the step test with a work load of 350 KgM./min. was measured.[4]

RESULTS

The results of the lung function tests are presented in Tables I, II, III (a), and (b). The total lung capacity was normal but there was an increase in the residual volume. Ventilatory tests showed a diminution in flow rates with a reduced $F.E.V._1/F.V.C.\%$, indicating airways obstruction. The response to bronchodilator aerosol was fair. Gas distribution was impaired, and resting ventilation was below normal. Alveolar ventilation was below normal and the alveolar arterial oxygen tension difference was increased. There was carbon dioxide retention and hypoxemia at rest. Voluntary hyperventilation resulted in a fall of the $PaCO_2$ from 58 mm. Hg. to 40 mm. Hg. and a rise in the PaO_2 from 58 to 94 mm. Hg. The pH rose from 7.30 to 7.48. The patient was able to complete six minutes of the standardised test without severe dyspnoea. The ventilatory response to CO_2 inhalation was impaired.

DISCUSSION

The patient reported here resembles the previous 4 cases described, although the clinical picture is not so severe. There is chronic alveolar underventilation at rest, with polycythaemia and symptoms of headache and episodic somnolence.

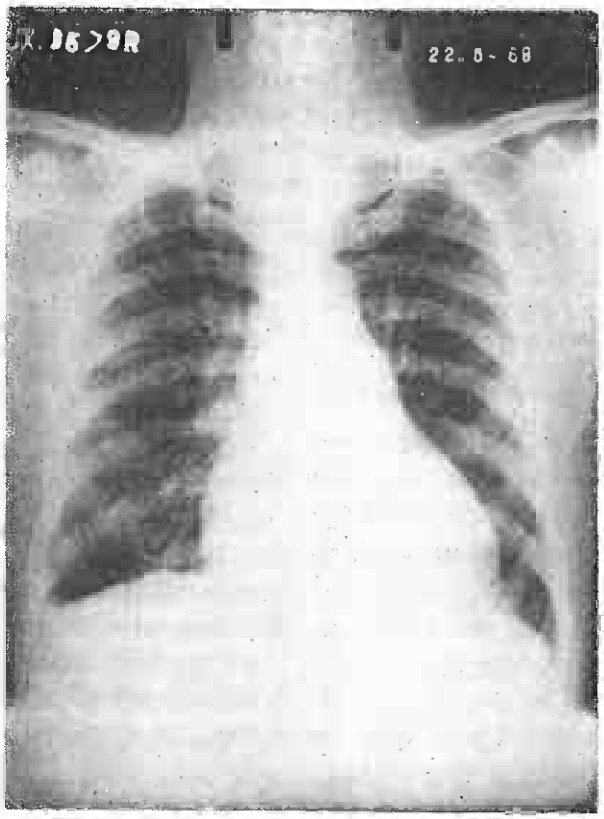

Fig. 1. Chest radiograph showing an enlarged heart and main pulmonary arteries, with obliteration of the right costo-phrenic angle.

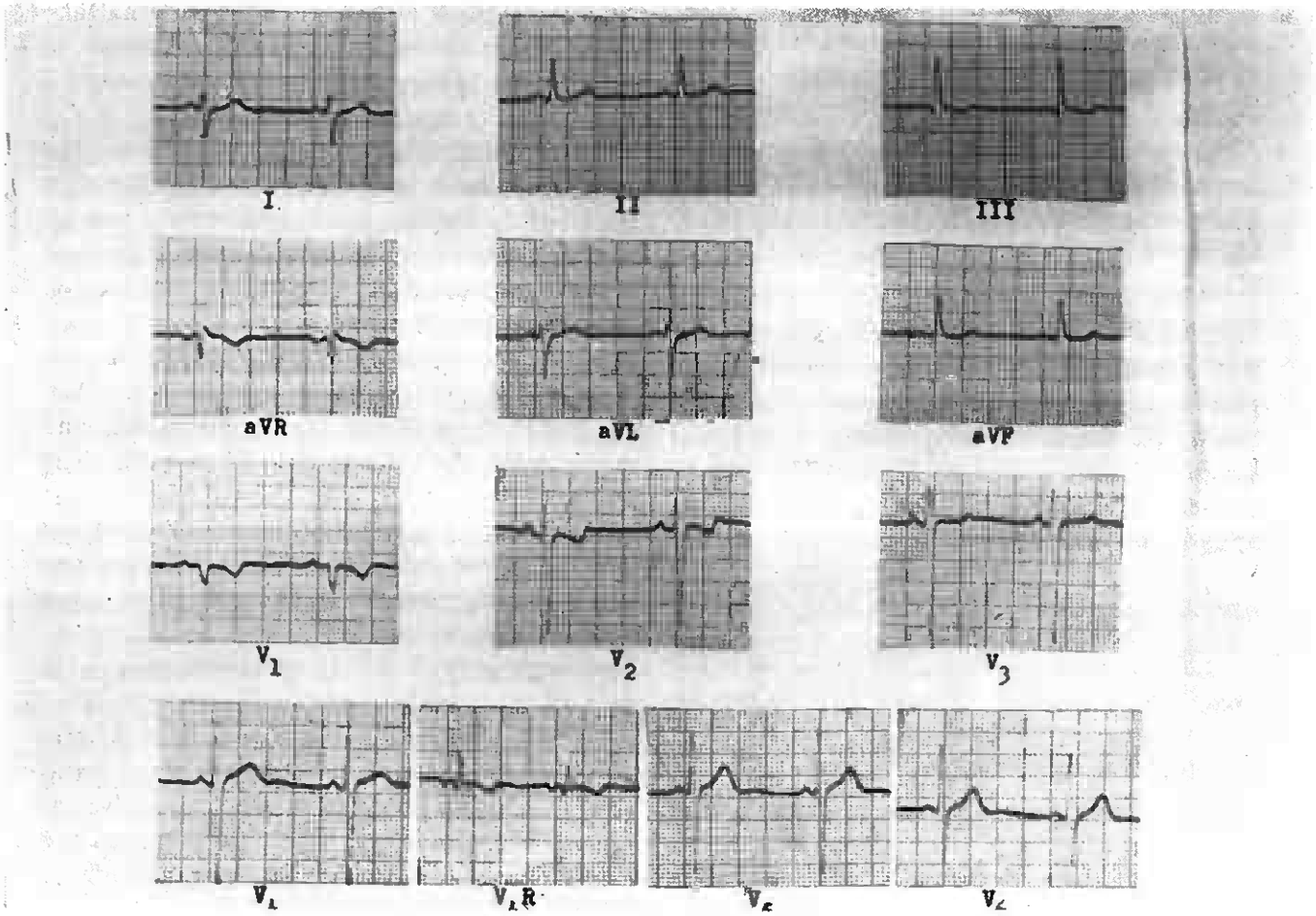

Fig. 2. Electrocardiogram revealing right ventricular hypertrophy.

TABLE I

LUNG FUNCTION TESTS

	V.C. (L)	F.R.C. (L)	R.V. (L)	T.L.C. (L)	R.V./ T.L.C. %	M.E. %	M.M.F.R. (L/sec.)	F.E.V. 75×40 (L/sec.)	M.V.V. (L/min.)	F.E.V. 1.0 (L)	F.E.V.1/ F.V.C. %
Predicted Normal	3.57	2.53	1.65	5.39	31	55	3.3	105	105	3.2	73
A. C. T. 14.7.68	3.10	3.80	2.17	5.27	41	34	1.06 (1.31)	56 (65)	77	1.64 (1.86)	58 (60)

( )—After bronchodilator aerosol.

TABLE II

LUNG FUNCTION TESTS

	Patient A.C.T.	Predicted Normal
$\dot{V}_E$ (l./min.)	6.42	8.2
$\dot{V}_A$ (l./min.)	2.51	4.20-5.40
Resp. Rate (/min.)	12	17.6
V_T (ml.)	535	466
V_D (ml.)	323	<160
V_D/V_T	0.60	< 0.30
pH	7.36	7.40
$PaCO_2$ (mm. Hg.)	58	40
PaO_2 (mm. Hg.)	58	95
D(A-a) PO_2 (mm. Hg.)	25	15
Rise of HbO_2% after 2 mins. hyperventilation	+9%	+3%
Standardised Ventilation (350 Kgm./min.) (l./min.)	31	26

TABLE III(a)

RESTING MINUTE VENTILATION AND BLOOD GAS VALUES BREATHING 100% OXYGEN AND A MIXTURE OF 5% CARBON DIOXIDE AND 95% OXYGEN

	Minute Ventilation (l./min.)		Arterial Blood				
			pH		$PaCO_2$ (mm. Hg.)		SaO_2%
Inspired Gas	100% O_2	5% CO_2 95% O_2	100% O_2	5% CO_2 95% O_2	100% O_2	5% CO_2 95% O_2	100 % O_2
Predicted Normal	8.8	16.3	7.40	7.37	40	45	>100
A.C.T.	7.4	12.7	7.32	7.26	66	79	101

TABLE III(b)

RESPONSE TO CARBON DIOXIDE INHALATION

	A. C. T.	Predicted Normal
Rise in $\dot{V}_E$ per mm. rise in $PaCO_2$	0.41	2.50
Rise in $\dot{V}_E$ per $1 \times 10\text{-}9$ mol./L rise in [H+]	0.75	4.54
Rise in $PaCO_2$ required to double resting ventilation	18.1	3.5
Rise in [H+] required to double resting ventilation	9.9	2.0

The results of ventilatory tests show evidence of airway obstruction consistent with a clinical diagnosis of chronic bronchitis. However, patients with chronic bronchitis and/or emphysema have been shown not to have an elevated $PaCO_2$ unless the $F.E.V._{1.0}$ is 1 litre or less;[5] the patient's blood gas abnormalities would thus appear out of proportion to those that one would expect in patients with a similar degree of airways obstruction.

Ventilatory response to inhalation of 5% CO_2 is poor and this together with the changes in the blood gas tensions after voluntary hyperventilation help to confirm the diagnosis of chronic hypoventilation coexisting with chronic bronchitis.

Exercise tolerance is good, as shown by the patient being able to complete the fairly severe exercise test without undue dyspnoea. The standardised ventilation is only slightly raised. It is interesting to speculate as to why his F.E.V. 75×40 is much lower than his M.V.V. Bates[6] has put forward the intriguing possibility that if the patient is sufficiently hypoxic, this degree of hypoxia will lead to muscle weakness. Performance of a single F.E.V. would be likely to be impaired by muscle weakness, whereas a 15-sec. M.V.V. might, by initial hyperventilation, so increase the PaO_2 that he substantially improves his voluntary effort.

The patient thus resembles physiologically the syndrome of primary alveolar hyperventilation described previously[9] and reviewed recently.[8] Whilst the latter cases usually give a history or show clinical evidence of neurological disturbance, in this patient with the exception of mild bronchitis, the alveolar under-ventilation is the only other isolated finding.

Attempts have been made to correlate blood gas tensions with morphologic changes. It is apparent that such correlations will be made more difficult by cases, such as the one described above, where hypoventilation coexists with chronic bronchitis.

SUMMARY

A case of chronic hypoventilation complicating chronic bronchitis is described. The clinical picture is one dominated by chronic underventilation. Clinically and physiologically except for the presence of mild airways obstruction, this patient resembles cases of primary alveolar hypoventilation.

ACKNOWLEDGEMENT

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