The incidence of lobar pneumonia rose with age, segmental pneumonia was most frequent in young children, and acute bronchitis was most often found in the young and the elderly. Similar facts were observed in the report of the College of General Practitioners (1956). These three clinical conditions also had quite different disability rates at follow-up. The highest disability rate was found in those who had suffered from acute bronchitis (71%), and the lowest in those with segmental pneumonias (16%); that for lobar pneumonia was in between (42%). It was also found that the lower social classes were more likely to suffer from these acute chest infections, whereas only 11% of the practice population belonged to classes IV and V; 44% of the patients with these chest infections were in these two groups.

When the disabled group was analysed in an attempt to answer the questions *who* was disabled and *why*, some pointers emerged. Of those disabled 63% (118 out of 187) were over the age of 40. The disability rate for males (47%) was only a little higher than that for females (40%). It was very difficult to interpret the role of smoking because while 90% of disabled men were smokers (but then more than 76% of men in the London area are smokers) only 40% of the disabled women were smokers (in London 48% of women are smokers). It may well be that men were more susceptible to the effects of smoking and that they smoked more, or that women found it easier to give up once symptoms developed. More studies are obviously necessary to clear up this point.

The disabled group contained 53% in social classes IV and V, whereas only 11% of the practice were in these two classes. It seems fairly definite that the lower social groups are more likely to suffer disability after pneumonias and acute bronchitis. A previous history of chest trouble also had a marked adverse effect on the prognosis. The disability rate in those with such a history was 71%—almost twice that for the whole series, which was 40%.

A picture thus builds up that those *most* likely to develop disabilities from chest infections are men over 40 who smoke, who belong to social classes IV and V, and who have a previous history of chest troubles. There are also other factors which have not been taken into account here, but which must be considered—the environmental effects, such as atmospheric pollution, and occupational risks and genetic and familial influences. The treatment given may also play a part in influencing the ultimate course of the condition. All these facts require further assessment by planned long-term studies before prevention can be attempted.

Summary

424 patients who suffered from pneumonia or acute bronchitis in 1949–54 were followed up for 5 to 10 years and reassessed functionally in 1959.

As many as 43% were considered to be disabled according to the method of grading used, 9% were complete invalids, and 24 had died during the acute illness. This rate of disability was more than twice that present (21%) before the infection 5 to 10 years earlier.

Three distinct clinical conditions were recognized—lobar pneumonia, segmental pneumonia, and acute bronchitis.

Lobar pneumonia accounted for 15% of the whole series. The age incidence rose with age. The sex dis-

tribution was equal. It was more frequent in lower social groups, in male smokers, and in those with a previous history of chest illness. There was a disability rate of 42%, compared with 21% before the episode.

Segmental pneumonias were much the most frequent in young children. There was a disability rate at follow-up of only 16%, and some relations were noted with social classes and smoking; even this rate was much higher than the 6% before the infection.

Acute bronchitis had the unusual age incidence of being frequent in the young and the old. Disability at follow-up had occurred in 71%, and there were close associations with smoking, social classes, and previous chest illnesses. The initial disability rate had been 38%.

The importance of acute chest illnesses is evident from the fact that one million persons are affected each year and that 43% appear to be left with some disability, which is twice the initial figure. Preventive measures must take into account the associations between disability and age, sex, social class, smoking habits, past history, and environmental and genetic influences.

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HYPOXIA AND THORACIC SCOLIOSIS

BY

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It has been recognized for many years that thoracic deformity predisposes to the development of hypoxia and respiratory failure, but the mechanism by which these are brought about is not fully understood. Gray (1956) studied 22 patients with kyphoscoliosis, and concluded that the abnormalities in pulmonary function found in this condition were similar to those occurring in emphysema. This implies that the primary cause for the hypoxia in patients with a chest deformity is uneven distribution of air and blood within the lungs (Bates, Knott, and Christie, 1956). However, after investigating 16 patients, Schaub, Bühlmann, Kälin, and Wegmann (1954) reported that the main factor was alveolar hypoventilation. Fishman, Bergofsky, Turino, Jameson, and Richards (1956) and Hanley, Platts, Clifton, and Morris (1958) reached similar conclusions, and suggested that patients with thoracic deformities underventilated owing to restriction of the chest bellows.

The common association of chronic bronchitis with deformities of the chest tends to obscure the effect of the latter upon pulmonary function. Other difficulties in interpreting the results of pulmonary function studies in this condition are the variety of types of deformity and the lack, until recent years, of a satisfactory technique for measuring the relationship of ventilation to blood-flow within the lungs. The present study was

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undertaken in an attempt to discover mechanisms by which deformity of the chest may predispose to hypoxia. It was confined to patients whose deformity was due to scoliosis of the thoracic spine, but included cases with and without bronchitis.

Material and Methods

Fourteen patients with severe scoliotic deformity of the thoracic spine were studied. In all cases the deformity had been present since adolescence and was obvious clinically. X-ray films of the two least severe cases are here shown (see Fig.). Nine patients had a past history of chronic winter bronchitis. Further details of the patients are given in Table I.

TABLE I.—Clinical Data of Patients with Scoliosis

Case No.	Sex and Age	Wt. (kg.)	Spinal Deformity		Duration of Bronchitis (Years)	Presenting Complaint	Present or Past Cardiac Failure
			Cause	Age at Onset			
1	M 23	57.9	Idiopathic	Childhood	17	Dyspnoea	— *
2	M 50	43.4	"	"	15	Cough	—
3	M 53	81.0	"	"	12	"	+
4	M 34	47.7	Poliomyelitis	"	16	Head-aches	+
5	F 36	31.8	Idiopathic	"	Nil	Oedema	+
6	M 64	49.4	"	"	19	Dyspnoea	+
7	F 38	45.8	"	"	4	Cough	+
8	M 62	40.3	"	Adolescence	11	Dyspnoea	+
9	M 72	43.6	Idiopathic + osteoporosis and collapse	Childhood	Nil	Abdominal distension	—
10	M 44	63.5	Idiopathic	Adolescence	25	Cough	—
11	M 22	52.8	Congenital	Birth	Nil	Back pain	—
12	F 39	50.0	Idiopathic	Childhood	"	Chest pain	—
13	F 47	35.0	"	"	"	M.M.R. pick up	—
14	M 57	45.0	"	Adolescence	40	Cough	+

* Developed one year after.

Arterial Blood Gases.—These were studied in nine patients; in five the arterial samples were taken when the subjects attended the out-patient department, and in four after they had been treated in hospital. In all instances the patients were thought to be at their best; only one had no previous history of bronchitis.

Arterial blood was collected from the brachial artery by percutaneous puncture after the skin had been anaesthetized with 2% procaine. Analysis for oxygen content, oxygen saturation, and total carbon dioxide content of the plasma was by the method of Van Slyke and Neill (1924). Blood pH was measured with a Cambridge pH meter, and the carbon dioxide tension was calculated from the total carbon dioxide content of the plasma and the pH by using the Henderson-Hasselbalch equation.

Pulmonary Function.—Static lung volumes were measured in three patients with a previous history of bronchitis and in four without. Other tests of pulmonary function were carried out in 10 patients, five of whom suffered from chronic bronchitis.

The one-second forced expiratory volume (F.E.V.₁) and the vital capacity (V.C.) were measured by an automatic timing device (Gaensler, 1951). The amount of gas which could be expired in one second was expressed as a percentage of the vital capacity in order to give a measure of airways resistance, while the maximum breathing capacity (M.B.C.) was calculated from the F.E.V.₁, using a factor of 35 (Thomson and Hugh-Jones, 1958). Lung volumes were measured, using the closed-circuit helium technique of Bates and Christie (1950). The evenness of distribution of gas and blood flow in the lungs and the relationship of ventilation to blood flow (ventilation-perfusion ratio) throughout the lungs were studied by recording the tensions of oxygen, carbon dioxide, and argon in expired gas. Gas analysis was carried out according to the single-breath procedure of West, Fowler, Hugh-Jones, and O'Donnell (1957), and using the multichannel respiratory mass spectrometer described by Fowler and Hugh-Jones (1957).

Results

Arterial Blood Gases

Blood gas studies were carried out in nine scoliotic subjects. The arterial oxygen saturation was below the normal range of 95–97% (Sunderman and Boerner, 1949) in all cases. The mean arterial oxygen saturation

Postero-anterior chest radiographs of the two patients with the least severe scoliotic deformity, Cases 1 (left) and 12 (right).

was 87.1%, with a range of 83–91%. Seven of the nine subjects had hypercapnia. The mean carbon dioxide tension was 48.6 mm. Hg, and the range was 36–62 mm.; the normal range is 35–45 mm. Hg (Sunderman and Boerner, 1949). The pH of arterial blood was within the normal range of 7.35–7.45 in every case.

Pulmonary Function

The M.B.C. was below the normal values quoted by Needham, Rogan, and McDonald (1954) in all of the 10 scoliotic patients studied. The reduction tended to be greater in those suffering from bronchitis, but the ranges of the two groups overlapped. The F.E.V.₁, expressed as a percentage of the vital capacity was below the lower limit of the normal range (70% according to Needham *et al.*, 1954) in four of the five bronchitic patients. It was within the normal range in four of the five patients without bronchitis; in the exception (Case 9), the value was 63%.

The total lung capacity (T.L.C.) was below the range of values found by Needham *et al.* (1954) in normal subjects in five of the seven scoliotic patients investigated. Two patients had normal values; both had a previous history of bronchitis. In addition, in these two cases the reserve volume (R.V.) expressed as a percentage of the total lung capacity, was greatly increased, while in the other five the percentage was only slightly increased or within the normal range.

The most striking finding in the present investigation was some unevenness of ventilation or ventilation-perfusion ratio throughout the lung in every case compared with the normal standards of Read (1959). The inequality of ventilation-perfusion ratio was outside the normal range in nine subjects, gross abnormality being present in five. Abnormal inequality of ventilation was found in nine of the cases and inequality of perfusion in seven. The degree of unevenness of ventilation or of ventilation-perfusion ratio appeared to be irrespective of the presence or absence of bronchitis (see Table II).

TABLE II.—Comparison of Pulmonary Function Data of Patients With and Without Bronchitis

Mean Values	M.B.C.	F.E.V. % V.C.	T.L.C. (Litres)	R.V. % T.L.C.	Inequalities % per 500 ml. Expired Gas		
					Ventilation- Perfusion Ratio	Ventilation	Perfusion
With bronchitis	35	54	4.1	57	18	22	7
No bronchitis	50	81	2.7	42	19	23	4
Maximum inequalities for a normal subject aged 45					7	10	9

An unusual finding in two cases (Nos. 4 and 13) was a large negative value for perfusion. This means that the blood flow of the alveoli emptying last was greater than that of the alveoli emptying early in expiration. To call one pattern of expiration indicative of a positive inequality and the other indicative of a negative inequality is merely a convention of the method.

Discussion

In the present study the blood gases were analysed in nine patients with scoliosis in order to determine whether the abnormalities in arterial oxygen saturation and carbon dioxide tensions were those that would be expected to result from alveolar hypoventilation. Two

of the patients had normal arterial carbon dioxide tensions in spite of reduced arterial oxygen saturations. Therefore alveolar ventilation cannot have been inadequate in these instances. In view of this finding in two cases, it is pertinent to check that hypoventilation was the only factor responsible for the hypoxia in the others.

If a subject is hypoxic owing to hypoventilation alone, the value for the arterial oxygen saturation associated with a given arterial tension of carbon dioxide can be deduced from the curves of Rahn and Fenn (1955). This was done for the patients of the present study, assuming a respiratory quotient of 0.8, and in all instances the predicted arterial oxygen saturation was higher than the observed value. In six patients the difference was 6% or more. It would seem likely, therefore, that in most of the patients in the present series some factor other than hypoventilation prevented a full oxygenation of the blood. If the above analysis is applied to the data of the other workers, one of the eight cases reported by Chapman *et al.* (1939), 2 out of 16 cases reported by Schaub *et al.* (1954), and 12 out of the 24 cases described by Hanley *et al.* (1958) had a degree of hypoxia which was out of proportion to the severity of the carbon dioxide retention.

There are three types of disturbances of pulmonary function, other than alveolar hypoventilation, which might be responsible for hypoxia in scoliotic patients. These are a defect in diffusion of oxygen across the alveolar membrane, shunting of blood through non-aerated segments of the lungs, and uneven distribution of gas and blood throughout the lungs. The ability of oxygen to diffuse from the alveoli into the pulmonary capillaries is usually inferred from studies of the behaviour of carbon monoxide. Kjerulf-Jensen and Kruhøffer (1954) found that the diffusing capacity for ¹⁴C-labelled carbon monoxide was abnormally low in a patient with a gross reduction in T.L.C. due to kyphoscoliosis. However, as these authors pointed out, decrease in diffusing capacity to as little as 25% of normal would be associated with a drop in resting arterial oxygen saturation of less than 1%. It seems unlikely, therefore, that a defect in diffusion of oxygen across the alveolar membrane plays a major part in the production of hypoxia in the scoliotic patient.

Necropsy reports on patients with thoracic spinal deformities often stress the occurrence of collapsed and underdeveloped segments of lung (Kerwin, 1942; Lewis, Daines, Samuels, and Hecht, 1952; Fischer and Dolehide, 1954), and it is possible that these might allow unoxygenated blood to pass to the left side of the heart. In the present series a large shunt was excluded in at least two of the subjects (Cases 1 and 4), because their arterial oxygen saturations rose from 86 and 88% to over 100% after inhaling pure oxygen through a valve box.

All but one of the scoliotic patients investigated in the present series had an abnormal degree of inequality in the ratio of ventilation to perfusion throughout the lung. It would seem reasonable to conclude that this predisposed the patients to the development of hypoxia. Unfortunately, blood gas analysis was not performed on the patient with a normal ventilation-perfusion ratio, and hence it is not known whether hypoxia was present in this instance.

The results of the present studies of the evenness of pulmonary ventilation and perfusion in scoliotic patients

differ from those reported by Fishman *et al.* (1956) and Hanley *et al.* (1958). One possible explanation is that there was some difference between the type of patient included in the various series. In fact, the investigation reported here was restricted to patients with a thoracic scoliotic deformity which had developed before adult life, whereas previous workers have tended to include a variety of deformities, some of recent onset.

It has already been pointed out that patients with deformed chests often suffer from chronic bronchitis. Therefore it might be suggested that this condition was responsible for some abnormalities in pulmonary function found in scoliotic patients. Certainly in the present investigation the results of spirometric and static lung-volume studies differed between patients with and without bronchitis. However, the evenness of ventilation, and ventilation-perfusion ratio, were disturbed to a similar extent in both groups of subjects. It would therefore seem likely that the deformity of the lungs associated with thoracic scoliosis was the prime cause of the abnormalities in ventilation and perfusion.

The way in which deformity of the chest produced the abnormalities in distribution of air and blood within the lungs in the subjects of the present study is uncertain. The uneven ventilation might have been due to bronchial distortion or to restriction of some lung segments. It is less easy to understand the factors underlying the uneven blood flow. The large negative value for perfusion which was present in two scoliotic subjects has not been seen in emphysema (Read, 1959) or in interstitial pulmonary disease (Read and Williams, 1959). It implies that in these two cases there were regions of the lung in which poorly ventilated alveoli were relatively well perfused. Such areas might well lie in partially collapsed lung segments.

In conclusion, it is suggested that scoliosis of the dorsal spine may be associated with abnormalities of the ventilation-perfusion ratio throughout the lungs, even in the absence of bronchitis. This disturbance of pulmonary function, in addition to alveolar hypoventilation, predisposes the scoliotic patient to hypoxia.

Summary

Pulmonary function was studied in 14 patients with severe scoliosis of the thoracic spine. Arterial blood gas analysis was performed in nine; estimation of vital capacity, one-second forced expiratory volume, evenness of ventilation, and ventilation-perfusion ratio were made in 10, and static lung volumes were measured in seven.

In all cases the arterial oxygen saturation was found to be lower than would be expected from alveolar hypoventilation alone.

The maximum breathing capacity was reduced in all cases. Patients who suffered from bronchitis in addition to scoliosis tended to have a marked increase in airways resistance and some increase in reserve volume. Those without bronchitis had a normal degree of airways resistance and residual volume, but a reduction in total lung capacity.

Both patients with and without a history of bronchitis showed abnormalities of distribution of gas and blood flow, and ventilation-perfusion ratio, throughout the lungs. It is suggested that these disturbances of pulmonary function were due to scoliosis, and that they predisposed to the development of hypoxia.

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MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION COMPLICATED BY HAEMATEMESIS

BY

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The administration of anticoagulants to patients with coronary thrombosis is almost routine practice, and there is substantial evidence that this treatment significantly reduces the mortality rate and the incidence of thrombo-embolic complications (Tulloch and Gilchrist, 1950; McCluskie and Seaton, 1959).

One of the main contraindications to anticoagulant therapy is the presence of haemorrhage or haemorrhagic tendency in the patient. Severity of the infarct and the presence of shock may outweigh the risk of further haemorrhage and make the use of an anticoagulant justifiable and necessary. The use of long-acting anticoagulants in the presence of a possible further haemorrhage is inadvisable in view of the fact that their action cannot be immediately antagonized, since the administration of intravenous vitamin K₁ leaves a period of hours before the prothrombin time returns to normal. The anticoagulant of choice under these circumstances is heparin; its activity is rapidly antagonized by intravenous protamine sulphate, but it has the disadvantage that for maximum efficiency it has to be given intravenously and at intervals of four to six hours. Even with heparin great care is necessary.