KYPHOSCOLIOSIS AND COR PULMONALE

A Study of the Pulmonary Vascular Bed

Richard L. Naeye, M.D.*

From the Department of Pathology, The University of Vermont College of Medicine, Burlington, Vt.

Certain persons with long-standing dorsal kyphoscoliosis develop pulmonary arterial hypertension and cor pulmonale. A normal pulmonary arterial wedge pressure in these cases suggests that the small pulmonary vessels are the site of an increased resistance to blood flow.[1] However, pulmonary vascular abnormalities have not been established as the basis for this high resistance.[2-9] The present study was undertaken to determine if such vascular changes occur and, if they do, what relationship they may have to other factors thought to influence the pulmonary circulation in kyphoscoliosis. These other factors include: (a) intrinsic pulmonary parenchymal disorder; (b) chronic hypoxemia; and (c) reduction in the pulmonary vascular bed by the thoracic deformity.

Intrinsic pulmonary parenchymal lesions are common in kyphoscoliosis. These may independently increase pulmonary vascular resistance. Included are chronic obstructive emphysema, bronchiectasis, pulmonary fibrosis, abscesses and atelectasis.[2-6,8,10-18] Chronic hypoxemia, often present in kyphoscoliosis, may induce both functional and anatomic changes in the pulmonary vascular bed.[1,9,19-23] Finally, the thoracic deformity itself may restrict the pulmonary vascular bed by reducing thoracic volume. Restriction of the pulmonary parenchyma is presumably associated with a reduction in the pulmonary vascular bed.[24] In adults with kyphoscoliosis the lungs are often less than one-half normal size.[1,9-12]

In the current study, each factor was considered in patients having dorsal kyphoscoliosis and chronic cor pulmonale. To evaluate the influence of the thoracic deformity, pulmonary vessels in these cases were compared with similar vessels in 3 control cases after pneumonectomy. The influence of chronic hypoxemia was assessed by comparison with vascular alterations known to be induced by chronic hypoxemia in other disorders. The influence of intrinsic lung lesions was explored by comparing the pulmonary vascular bed in kyphoscoliosis complicated by

Accepted for publication, December 19, 1960.

* Markle Scholar in Medical Science.

TABLE I

PATIENTS WITH DORSAL KYPHOSCOLIOSIS

Case number:	1	2	3	4	5	6	7	8	9	Normal
Age at death; sex	29M	49M	39F	42M	76F	49F	48M	57M	40F	
Duration of deformity (yr.)	15	49	39	41	76	Child-hood	46	55	38	
Symptoms										
Duration (yr.)	3	*	3	1	0	10	4	1½	2½	
Dyspnea	4+	4+	4+	4+	0	4+	3+	4+	4+	
Orthopnea	0	0	2+	1+	0	1+	0	1+	1+	
Signs										
Respirations per minute	40	25	38	36	30	44	*	26	30	16–22
Pul. 2nd sound, accentuation	1+	2+	1+	2+	0	0	2+	1+	3+	0
Cyanosis	3+	3+	3+	3+	0	3+	2+	3+	3+	0
Peripheral edema	3+	2+	1+	2+	0	*	*	4+	0	0
Laboratory data										
Hematocrit (%)	65.0	48.0	42.0	55.0	41.0	44.0	66.0	49.0	54.0	42–49
Arterial CO_2 cont. (vol. % at 40 mm. of Hg)	55.5	*	68.8	68.0	*	58.0	*	*	75.7	50
Arterial Hb. O_2 saturation in %	59.8	*	87.5	49.7	*	70.0	*	*	42.1	94–97
Right ventricular pressure (mm. of Hg)	58/10	*	*	*	*	53/3	*	*	*	
Vital capacity (cc.)	630	*	600	*	*	460	*	*	750	2 1/2 3400–4000

				Necropsy observations						
Heart										
Weight (gm.)	430	280	280	375	220	450	320	*	500	200–350
Dilatation of rt. atrium	1+	1+	1+	3+	0	2+	1+	3+	3+	0
Dilatation of rt. ventricle	2+	3+	1+	3+	1+	3+	2+	3+	3+	0
Rt. ventricle (thickness in mm.)	10.0	7.5	5.0	10.0	4.0	10.0	6.0	8.5	8.0	1–3
Lungs										
Vol. of lungs combined, in % of predicted	*	*	*	35%	65%	*	30%	*	35%	100%
Emphysema	1+	1+	1+	2+	2+	1+	3+	3+	3+	0
Bronchitis and bronchiolitis	0	0	0	0	0	2+	2+	2+	2+	0
Pleural adhesions	1+	2+	0	2+	0	0	0	2+	2+	0
Other pulmonary parenchymal disorders	Terminal atelectasis	Focal fibrosis, R.L.L.	0	0	Fibrosis, apex rt. lung	0	0	Bronchiectasis, L.U.L. Old atelectasis, L.L.L.	Bronchiectasis, L.U.L., abscess, L.U.L. Fibrosis	0

1+, very mild; 2+, mild; 3+, moderate; 4+, severe; *, unknown.

chronic obstructive emphysema with that in kyphoscoliosis without intrinsic parenchymal disease.

MATERIAL

Nine patients with dorsal kyphoscoliosis and chronic cor pulmonale were selected from the necropsy files at the Presbyterian and Bellevue Hospitals, New York City. There were evidences of pulmonary hypertension in all instances (Table I). Right heart catheterization in 2 of the cases demonstrated a marked elevation of right ventricular pressure which increased with exercise. At necropsy, all 9 had right ventricular myocardial hypertrophy and in 8 the right heart was dilated. None had a deformity of the great vessels sufficient to restrict blood flow through them.

All 9 patients had small lungs. In every case, the thoracic deformity was present long before skeletal growth was complete. This suggested that the skeletal deformities prevented the lungs from growing to the usual adult size. Kyphoscoliosis was usually the most important factor in reducing thoracic volume since it diminished the height of the thoracic cage. In most instances, the lateral rotation due to scoliosis was responsible for one lung being smaller than the other (Fig. 1). In the cases in which measurement was made, combined lung volumes by fluid displacement at necropsy ranged from 30 to 65 per cent of the values found in normal controls matched for body size and weight (Table I). The matched, normal controls were 17 individuals without thoracic deformity; these died with disorders unrelated to the cardiopulmonary system. The 65 per cent value was recorded in one patient who survived until old age and died of noncardiopulmonary causes (Table I). In 4 cases vital capacity measured during life was less than one third of normal.

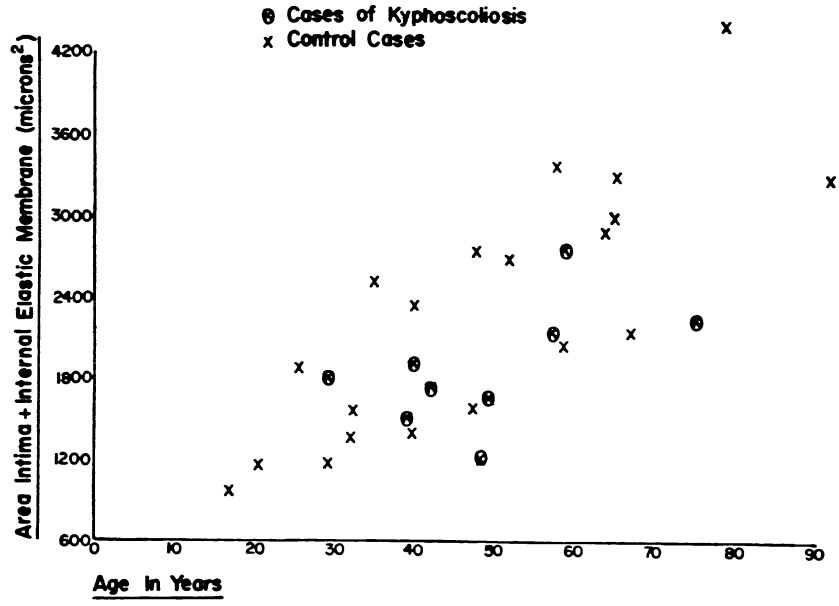
Arterial hypoxemia and hypercapnia were noted in 5 cases in which blood gas studies were made. Eight of the 9 patients were cyanotic, some for protracted periods and others for a short time before death. A secondary polycythemia was present in 5. These phenomena may be attributed to cardiorespiratory failure, a common clinical complication of kyphoscoliosis after the third decade.[1-6] Other common clinical features of cardiorespiratory failure which the patients exhibited were dyspnea, orthopnea and peripheral edema. Several individuals were somnolent during their terminal illnesses. Clinically, this was related to hypercapnia.

Significant intrinsic pulmonary lesions were found in 3 patients at necropsy (Nos. 7, 8 and 9). Two had had repeated respiratory infections during life, with cough and purulent sputum (Nos. 8 and 9). All 3 had diffuse, chronic obstructive emphysema, and bullae were prominent. On microscopic, examination, chronic inflammatory cells could often be seen in bronchiolar walls. Respiratory bronchioles and alveolar ducts were ballooned, and alveolar spaces were dilated, Alveolar walls were narrowed and ruptured. In many cases, fibrosis involved alveolar walls and interstitial tissues. Bronchiectasis was extensive in 2 of the patients, one of whom also had a pulmonary abscess.

Intrinsic pulmonary alterations were few in the other 6 cases. Except for reduced lung size, gross alterations were insignificant. Microscopically, some alveolar spaces were distended and scattered alveolar walls were ruptured. These changes were usually localized and were never associated with bronchitis or bronchiolitis. Patches of atelectasis were found in several of the lungs. The absence of inflammation or fibrosis in

most of the atelectatic areas suggested that they were of short duration. Each of the pulmonary lesions found in these 6 cases was found in some of the matched normal controls.

Lungs from 3 patients who had had pneumonectomy were also examined in an attempt to determine the effect of the single factor, pulmonary vascular bed reduction, on the remaining pulmonary vessels. All 3 individuals had survived operation for an appreciable time without intrinsic disease in the remaining lung. None had evidence of hypoxemia. In these patients there was mild right ventricular hypertrophy and dilatation at death. This suggested that there had been at least latent pulmonary hypertension during life—that is, an abnormal pulmonary arterial pressor response when blood flow was augmented by exercise.[25] At necropsy, the remaining lung was grossly normal. Microscopically the only finding was mild overdistention of alveolar spaces.



TEXT-FIGURE 1. The mean area of intima combined with internal elastic membrane is recorded for pulmonary arteries of all sizes in patients with kyphoscoliosis and in normal controls. Values for the two groups are similar. In both groups the values rise with age because of thickening of the intima.

METHODS

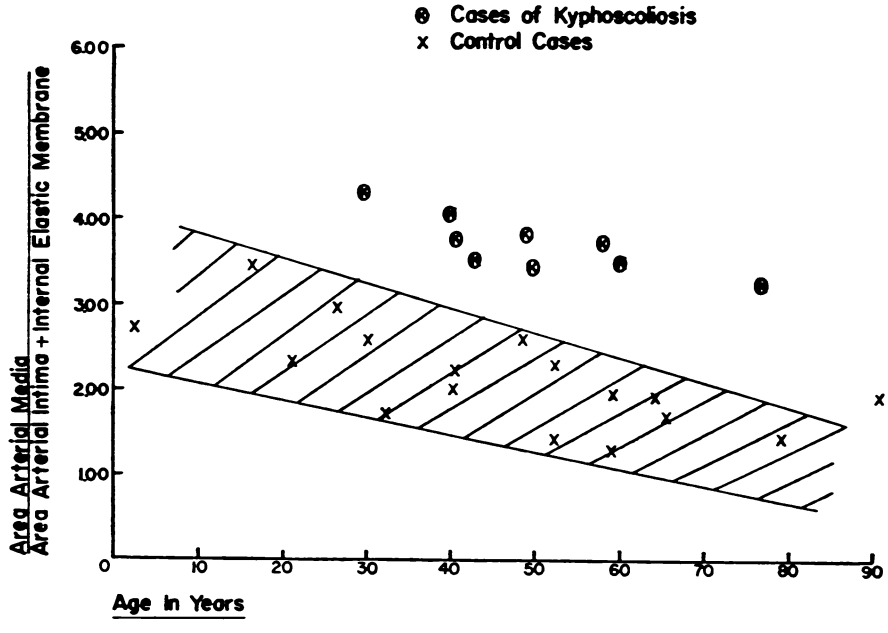
Blocks of tissue were taken from the lungs in each case, a balance being maintained between upper and lower lobes. Every block was serially sectioned so that vascular channels could be traced and examined for some distance. In two cases, bronchial and pulmonary arterial systems were injected with a gelatin preparation [26] in an attempt to demonstrate abnormal communications between the two circuits. Sections were stained with Verhoeff's and van Gieson's stains, a method which helped to differentiate fibrous tissue, elastic tissue and muscle.

A quantitative study of the structure of the pulmonary arterial beds was undertaken. To avoid bias, microscopic sections from control patients and those with kyphoscoliosis were mixed and examined in a random manner. Vascular measurements were made from drawings prepared at constant magnification with the aid of a camera lucida.[27] With a planimeter, the relative cross sectional areas of lumen, intima combined with internal elastic membrane, and media were determined for 15 to 30 muscular arteries and arterioles in each case. All vessels cut in cross section which were encountered were measured.

Results

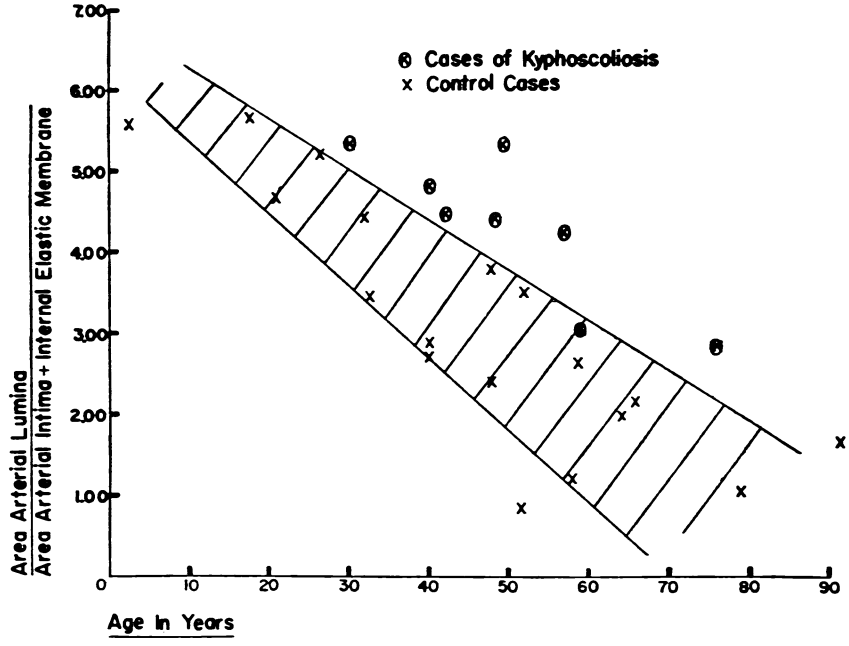
Neither qualitative nor quantitative abnormalities were found in the intima or elastic membranes of pulmonary arteries in patients with kyphoscoliosis when compared with normal controls. The mean combined cross sectional area of intima and internal elastic membrane was charted for muscular arteries and arterioles in each case (Text-fig. 1). The mean values in kyphoscoliosis matched those of normal controls, indicating that these two structures had not thickened in patients with the skeletal deformity. The mean area of these structures did increase with age, however, in both kyphoscoliosis and normal controls as a result of an increase in intimal connective tissue.

For further investigation, arteries and arterioles less than 150 μ in lumen diameter were selected for analysis since these presumably had an important role in controlling vascular resistance. The fact that the



TEXT-FIGURE 2. A ratio which reflects medial muscle mass is recorded for small pulmonary arteries in patients with kyphoscoliosis and in normal controls. The arteries in persons with kyphoscoliosis show a greater muscle mass than do the controls.

intimal area of pulmonary arteries was normal in our cases proved useful; it made possible the use of this value as a base line in the quantitative study of other vascular structures. The area of the media varied markedly and in a very significant manner when compared to this base line. The ratios of media to intima in cases of kyphoscoliosis and in nor-



TEXT-FIGURE 3. A ratio which reflects arterial dilatation is recorded for small pulmonary arteries in patients with kyphoscoliosis and in normal controls. In all but one individual with kyphoscoliosis, the pulmonary arteries are more dilated than in control cases.

mal controls are charted in Text-figure 2. The method employed for calculating this ratio has been previously published.[23,27] The mean ratios in persons with kyphoscoliosis were considerably greater than in the control cases. This indicated that an appreciable increase had taken place in the medial muscle mass of the smaller pulmonary arteries in the kyphoscoliotic group. In some of the cases, the relative medial mass was almost twice that of the controls. One of the principal advantages of this method of measuring medial mass was that such measurements were not affected by stretching of the vessels.[27]

A similar method was employed in evaluating the degree of arterial dilatation. The same base line was used for comparison—namely, the combined area of intima and internal elastic membrane. The area of the lumen was compared with this base line, since an increasing lumen size with dilatation would be associated with an increasing ratio. The mean

ratios of lumen to intima were increased in all but one of the cases of kyphoscoliosis, indicating that terminally, at least, arteries and arterioles were dilated (Text-fig. 3). On casual examination this dilatation obscured the medial hypertrophy because the eye usually evaluates such hypertrophy by comparing medial mass with lumen size (Fig. 2).

In about one half of the cases, the arterioles were normal. They did not have a muscular coat beyond the cuff which surrounded their point of exit from the small muscular arteries. In the other cases, such a coat did extend for varying distances down toward the capillary bed. Alveolar capillaries were markedly congested in all cases. Pulmonary veins and bronchial vessels were structurally normal. In the course of examining serial sections, no abnormal communications were found between the pulmonary and bronchial vascular systems. Likewise, no abnormal communications were found between pulmonary arteries and veins.

Similar studies were made in the lungs of 3 patients who had had pneumonectomy. A comparison was made in each case between the lung removed at operation and that obtained much later at necropsy. Both of the ratios described above were calculated for muscular arteries. In each instance the ratios in the postmortem lung were greater than those calculated for the previously resected lung (Table II). This indicated

TABLE II

VASCULAR ALTERATIONS FOLLOWING PNEUMONECTOMY

	Area media: Area intima + int. elast. mem.		Area lumen: Area intima + int. elast. mem.		Interval between pneumonectomy and death	Age at death
	Before pneumonectomy	After pneumonectomy	Before pneumonectomy	After pneumonectomy		
Case A	2.15	3.43	1.85	5.98	2 yr.	58 yr.
Case B	2.06	4.35	2.24	5.64	3½ yr.	65 yr.
Case C	1.61	4.21	1.53	3.06	5 yr.	48 yr.

The mean ratio $\frac{\text{area media}}{\text{area intima + I.E.M.}}$ for pulmonary arteries is compared in 3 lungs removed by pneumonectomy with the paired lung removed at necropsy. The greater necropsy ratios indicate muscular hypertrophy. An increase in the ratio $\frac{\text{area lumen}}{\text{area intima + I.E.M.}}$ indicates arterial dilatation.

that an appreciable increase in pulmonary arterial muscle mass and in arterial dilatation took place in the years after pneumonectomy. No other vascular abnormalities were found.

DISCUSSION

Both anatomic and clinical evidence indicates that the patients with kyphoscoliosis had pulmonary arterial hypertension. The only abnor-

mality within individual pulmonary vessels which might have been related to hypertension was smooth muscle hypertrophy in the media of arteries and arterioles. Lumen narrowing by intimal fibrosis, so often responsible for pulmonary hypertension in other disorders, was absent. The genesis of the muscular hypertrophy and its possible functional significance must be evaluated in the light of other abnormalities which affect the lesser circulation in kyphoscoliosis.

The reduced size of the lungs in kyphoscoliosis imposes a severe restriction on the pulmonary vascular bed.[1,9–12] This restriction appears to be responsible for at least latent pulmonary hypertension in many cases.[1] The vascular restriction might be compared to that which follows pneumonectomy. In both groups there are individuals without hypoxemia or significant intrinsic lung disease who have an abnormal pulmonary arterial pressor response when pulmonary blood flow is augmented by exercise.[1,25] In both groups the only abnormality in individual vessels is an appreciable hypertrophy of arterial muscle. In both instances the increased muscle mass might be interpreted as a work hypertrophy, a response to normal or increased blood flow through a reduced arterial bed.

Hypoxemia, often chronic, was present in all but one of the cases of kyphoscoliosis in the current study. This was probably most directly related to alveolar hypoventilation associated with an abnormal increase in the total work of breathing.[1,9,19,20] Bergofsky, Turino and Fishman[1] have noted that pulmonary hypertension is most severe in those cases of kyphoscoliosis in which hypoxemia is present. Chronic hypoxemia of this nature is capable of increasing cardiac output and of initiating a general hypervolemia in which the pulmonary circuit participates.[21,23] In the present study, secondary polycythemia and dilatation of pulmonary arteries were evidences of the latter. The high cardiac output and hypervolemia could further increase the circulatory load on the restricted pulmonary bed in kyphoscoliosis and contribute to a work hypertrophy in arterial smooth muscle. However, all of the effects of hypertension associated with hypoxemia cannot be attributed to changes in blood flow and volume.[1,21] It is thought that hypoxemia acts either locally or by reflex means to reduce the caliber or distensibility of the small pulmonary vessels.[21] A plausible target for such an action is the smooth muscle surrounding pulmonary arteries. A consequent increase in the state of contraction of this muscle might well serve as the stimulus for its hypertrophy. This has been postulated in other cases of chronic hypoxemia.[22,23] An alternative explanation would be that the hypertrophied muscle is a consequence of the hypertension rather than part of its cause. If this hypothesis were accepted, it would be necessary

to postulate a vasomotor response to hypoxemia in some more distal segment of the lesser circulation.

Despite small lungs from childhood in most patients with kyphoscoliosis, cor pulmonale is rare before the age of 25 years.[12] The relatively late development of obstructive emphysema and other intrinsic pulmonary diseases may explain this phenomenon in some cases. Chronic obstructive emphysema was prominent in 3 individuals in the present group (Table I). These could not be distinguished from the patients with kyphoscoliosis who had no parenchymal disorders on the basis of pulmonary vascular abnormalities; vascular alterations were identical in the two groups. This similarity can probably be attributed to the factors which might contribute to hypertension in obstructive emphysema, such as hypoxemia and vascular bed reduction. Similar factors were present in the kyphoscoliotic subjects not having emphysema. It seems reasonable to assume that when obstructive emphysema is severe in kyphoscoliosis, it may contribute to pulmonary hypertension. In our cases, other pulmonary parenchymal disorders were too limited in their extent to add significantly to the development of cor pulmonale.

In conclusion, multiple factors may reduce the over-all area and the distensibility of the pulmonary bed in kyphoscoliosis. By increasing pulmonary vascular resistance they could contribute to the development of arterial hypertension. The latent hypertension observed in many younger patients is apparently due to a vascular bed reduced by the limitation of pulmonary parenchyma inherent in the thoracic deformity. Hypertension, in turn, is probably responsible for pulmonary arterial smooth muscle hypertrophy. In later life, other factors contribute to progressive cor pulmonale. Structural changes in the bones and joints of the thoracic cage increase the work of respiration, leading to the development of alveolar hypoventilation and hypoxemia. The hypoxemia may induce a narrowing of some segment of the pulmonary vascular bed and is most likely responsible for hypertrophy of periarterial smooth muscle. Furthermore, the hypoxemia may limit pulmonary vascular distensibility indirectly by increasing the pulmonary blood volume. Finally, intrinsic pulmonary diseases, such as chronic obstructive emphysema, appear to contribute to pulmonary hypertension in a number of individuals.

Summary

Nine patients with dorsal kyphoscoliosis who had evidence of cor pulmonale at death were investigated. Pulmonary arterial and arteriolar changes were similar throughout the group. These consisted of marked hypertrophy of the media and vascular dilatation. Three factors were found which may have contributed to these vascular changes and to pulmonary hypertension:

1. All cases had small lungs and a consequent reduction in the pulmonary vascular bed. The pulmonary arterial muscular hypertrophy found in kyphoscoliosis is apparently similar to that which develops in the remaining lung after pneumonectomy.

2. In all cases there was evidence of chronic hypoxemia. The muscular hypertrophy and vascular dilatation noted were similar to the pulmonary vascular changes attributed to chronic hypoxemia in other disorders.

3. Severe intrinsic pulmonary disease was present in 3 of the 9 cases. It did not appear to have a specific effect on the pulmonary vascular bed in patients with this thoracic deformity.

References

1. Bergofsky, E. H.; Turino, G. M., and Fishman, A. P. Cardiorespiratory failure in kyphoscoliosis. *Medicine,* 1959, **38,** 263–317.
2. Fischer, J. W., and Dolehide, R. A. Fatal cardiac failure in persons with thoracic deformities. *A.M.A. Arch. Int. Med.,* 1954, **93,** 687–697.
3. Reid, W. D. Spinal deformity as a cause of cardiac hypertrophy. *J.A.M.A.,* 1930, **94,** 483.
4. Lewis, C. S., Jr.; Daines, M. C.; Samuels, A. J., and Hecht, H. H. Cor pulmonale (pulmono-cardiac syndrome); a case report. *Dis. Chest,* 1952, **22,** 261–268.
5. Finley, F. G. Spinal deformity as a cause of cardiac hypertrophy and dilatation. *Canad. M.A.J.,* 1921, **11,** 719.
6. Chapman, E. M.; Dill, D. B., and Graybiel, A. The decrease in functional capacity of the lungs and heart resulting from deformities of the chest: pulmonocardiac failure. *Medicine,* 1939, **18,** 167–202.
7. Hertzog, A. J., and Manz, W. R. Right-sided heart failure (cor pulmonale) caused by chest deformity; case report. *Am. Heart J.,* 1943, **25,** 399–403.
8. Kerwin, A. J. Pulmonocardiac failure as a result of spinal deformity; report of 5 cases. *Arch. Int. Med.,* 1942, **69,** 560–572.
9. Hanley, T.; Platts, M. M.; Clifton, M., and Morris, T. L. Heart failure of the hunchback. *Quart. J. Med.,* 1958, **27,** 155–171.
10. Bachmann, M. O. Die Veränderungen der inneren Organne bei hochgradigen Skoliosen und Kyphoskoliosen. E. Nägele, Stuttgart, 1899, 172 pp.
11. Gray, F. D., Jr. Kyphoscoliosis and heart disease. *J. Chronic Dis.,* 1956, **4,** 499–507.
12. Samuelsson, S. Cor pulmonale resulting from deformities of the chest. *Acta med. scandinav.,* 1952, **142,** 399–408.
13. Klawansky, G. Pronounced mechanical stenosis of aorta from kyphoscoliosis. *Klin. Wchnschr.,* 1925, **4,** 831–832.
14. Rieder, J. Die Respirations- und Circulationsstörungen bei Kyphoscoliosis dorsalis. M. Niethe, Berlin, 1881, 32 pp.
15. Traube, L. Ein Fall von Dilatation und Hypertrophie des rechten Ventrikels bei einem mit hochgradiger Scoliose und Verbildung des Brustkorbes behaften Individuum. *Ges. Beitr. z. Path. u. Physiol.,* 1878, **3,** 354–358.
16. Cabot, R. C. Case 19432. The interesting result of severe deformity of the thorax. *New England J. Med.,* 1933, **209,** 854–855.

17. COOMBS, C. F. Fatal cardiac failure occurring in persons with angular deformity of the spine. *Brit. J. Surg.*, 1930, **18**, 326–328.

18. DALEY, R. Morphine hypersensitivity in kyphoscoliosis. *Brit. Heart J.*, 1945, **7**, 101–103.

19. SCHAUB, F.; BÜHLMANN, A., and KALIN, R. Das "Kyphoskolioseherz" und seine Pathogenese. *Cardiologia*, 1954, **25**, 147–152.

20. FISHMAN, A. P.; TURINO, G. M., and BERGOFSKY, E. H. The syndrome of alveolar hypoventilation. *Am. J. Med.*, 1957, **23**, 333–339.

21. FISHMAN, A. P.; MCCLEMENT, J.; HIMMELSTEIN, A., and COURNAND, A. Effects of acute anoxia on the circulation and respiration in patients with chronic pulmonary disease studied during the "steady state." *J. Clin. Invest.*, 1952, **31**, 770–781.

22. NAEYE, R. L., and BICKERMAN, H. A. The effects of hypoxemia on the pulmonary arterial bed of humans and rats. (Abstract) *Fed. Proc.*, 1959, **18**, 497.

23. NAEYE, R. L. Anoxia and pulmonary hypertension, a study of the pulmonary vasculature. *Arch. Path.* (In press)

24. NAEYE, R. L. Kyphoscoliosis and chronic cor pulmonale; a study of the pulmonary vascular bed. (Abstract) *Proc. New England Cardiovascular Soc.*, 1960, **18**, 19.

25. RILEY, R. L.; HIMMELSTEIN, A.; MOTLEY, H. L.; WEINER, H. M., and COURNAND, A. Studies of the pulmonary circulation at rest and during exercise in normal individuals and in patients with chronic pulmonary disease. *Am. J. Physiol.*, 1948, **152**, 372–382.

26. SCHLESINGER, M. J. New radiopaque mass for vascular injection. *Lab. Invest.*, 1957, **6**, 1–11.

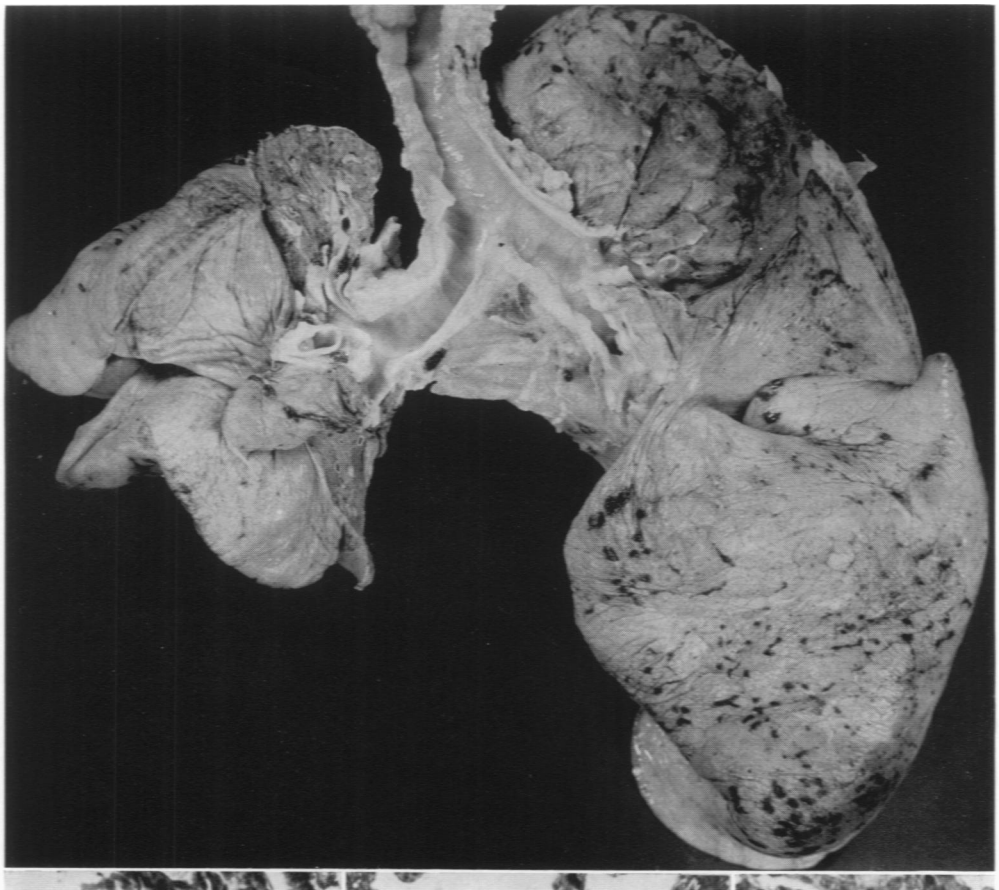
27. NAEYE, R. L. Smooth muscle changes in arteries of the greater and lesser circulations during the perinatal period. *Arch. Path.*, 1961, **71**, 121–128.

I am indebted to Dr. Harry P. Smith, Presbyterian Hospital, and Dr. Marvin Kuschner, Bellevue Hospital, New York, for the necropsy material used in this investigation, and to Dr. Alfred P. Fishman for helpful criticism and for the cardiac catheterization data in two cases.

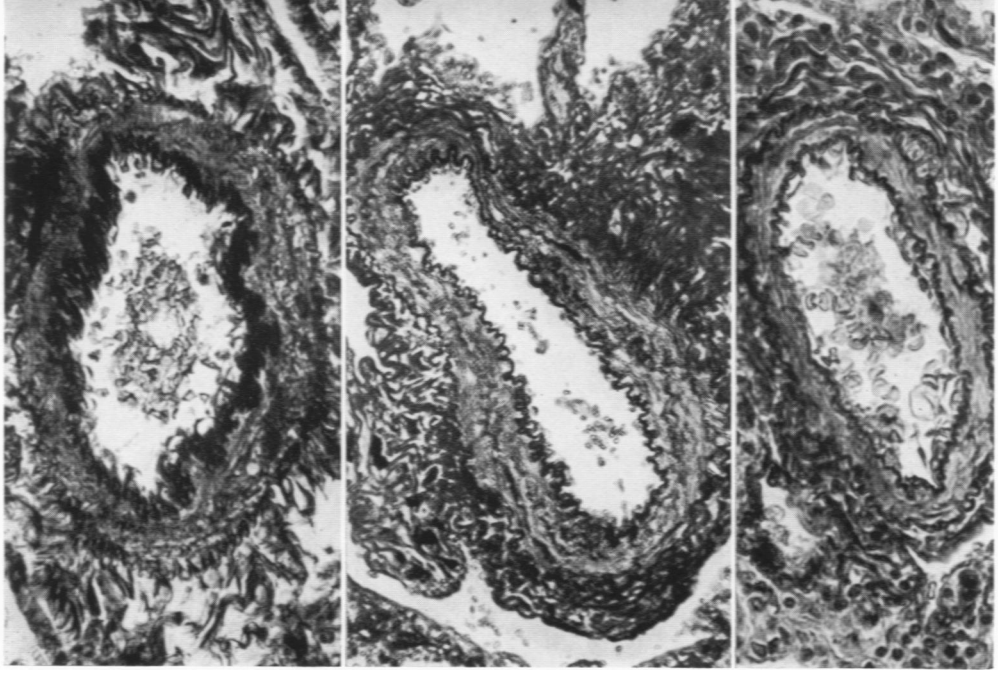
LEGENDS FOR FIGURES

FIG. 1. This posterior view of the lungs in case 5 shows the effect of the thoracic deformity on the lungs. The volume of the right lung is 95 per cent of that predicted and of the left lung 35 per cent of the predicted size. Note the absence of bullae. There is a small area of fibrosis at the apex of the right lung.

FIG. 2. Pulmonary muscular artery (A) is in a normal subject. Arteries (B) and (C) are from a patient with kyphoscoliosis and cor pulmonale. The media appears to be of about equal thickness in arteries (A) and (B). However, vessel (B) is more dilated than vessel (A) as can be determined by observing its internal elastic membrane which is less wrinkled. Artery (C) is still more dilated. The ratio $\frac{\text{area arterial media}}{\text{area intima + internal elastic membrane}}$ is much greater in arteries (B) and (C) than in artery (A). There is significant medial muscular hypertrophy in arteries (B) and (C). Verhoeff and van Gieson stains. × 150.



1



2A

2B, 2C