PULMONARY ARTERY DIASTOLIC PRESSURE: ITS RELATIONSHIP TO PULMONARY ARTERIOLAR RESISTANCE AND PULMONARY "CAPILLARY" PRESSURE [1]

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In the systemic circulation, one ordinarily may assume increased peripheral resistance when the diastolic pressure is elevated (1). It is the purpose of this paper to ascertain whether a similar relationship exists within the lesser circulation of man.

MATERIAL

Subjects were selected at random from the medical wards of the Cincinnati General Hospital. Some were normal convalescents; others were suffering from various types of heart and lung disease (Table I). Satisfactory data were obtained in 54 patients.

METHOD

Subjects were studied in the fasting condition, sedated by 0.1 or 0.2 gm. of seconal. Catheterization of the right side of the heart was done as described previously (2). Pulmonary "capillary" pressure was obtained by the method of Hellems and co-workers (3). The criteria of satisfactory pulmonary "capillary" pressures were *1)* the nature of the pressure curve, showing "a" and "c" waves; *2)* the peripheral location of the catheter; and *3)* the securing of blood saturated with oxygen from the wedged catheter tip. In some instances no blood could be obtained from the wedged catheter. In the majority of instances a double lumen catheter was used, so that pulmonary artery and pulmonary "capillary" pressures could be recorded simultaneously. In some instances, a single lumen catheter was employed; in these cases pulmonary "capillary" pressure was recorded first; then the catheter tip was quickly withdrawn into the proximal portion of the pulmonary artery so that almost simultaneous recording of the pulmonary artery pressure could be made. Recordings of pressures were made with the Hathaway blood pressure recording apparatus.

Cardiac outputs were determined by the direct Fick method as outlined before (2). Mixed venous samples were obtained from the proximal portion of the pulmonary artery. Duplicate samples of expired air were analyzed for CO_2 and O_2 and required to check within 0.03%. Duplicate blood samples, obtained during the collection of expired air, were analyzed for O_2 on the Van Slyke manometric apparatus and required to check within 0.2 vol. %.

Resting cardiac outputs were obtained after the catheter had been in place 15 to 30 minutes or more. Immediately after taking blood and gas samples for the output, pressure recordings were made. Mean pulmonary artery and pulmonary "capillary" pressures were determined by planimetry. Measurements were made over two or more respiratory cycles. A point 10 cm. above the table on which the subject lay was taken as the zero point in pressure measurements.

Pulmonary arteriolar resistance was calculated from the formula

$$R = \frac{PA - PC}{CO} \times 1{,}332,$$

where R = arteriolar resistance in dynes sec./cm.$^{-5}$,
PA = mean pulmonary artery pressure, mm. Hg,
PC = mean pulmonary "capillary" pressure, mm. Hg,
CO = cardiac output in cc./sec.

RESULTS

These are shown in Table I and Figures 1 through 7. In Figure 1 is shown a scatter diagram plotted logarithmically indicating the observed relationship between pulmonary artery diastolic pressure and pulmonary arteriolar resistance. The correlation (4) between the two was high ($r = 0.92$; $p = < 0.001$). In Figures 2 and 3 are logarithmic graphs indicating the relationships between pulmonary arteriolar resistance and pulmonary artery systolic and mean pressures. It is noteworthy that even closer correlation was found here. The correlation coefficient of resistance and systolic pressure was 0.95 ($p = < 0.001$); the

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TABLE I

	Pulmonary "capillary" mean pressure, *mm. Hg*	Pulmonary artery pressure, *mm. Hg*		Cardiac output, *L/min.*		Pulmonary arteriolar resistance, *dynes sec./cm.*$^{-5}$	Arterial O_2 sat., *% of capacity*
		Mean	Systolic/diastolic	Index	Total		
1. B. M. (F, 57) Bronchiectasis	9	29	38/17	2.7	4.0	397	84.1
2. I. W. (F, 29) Post-partum myocardosis	18	25	37/15	3.3	4.9	114	94.2
3. M. C. (M, 73) Normal	10	13	22/6	2.3	3.5	78	85.9
4. S. J. (M, ?68) HCVD, no failure	14	25	36/20	2.3	4.5	202	94.7
5. C. C. (M, 46) Normal (conv. empyema)	12	17	25/11	2.8	5.0	74	90.3
6. J. S. (M, 75) Hypertension	22	26	39/22	2.6	4.3	89	93.5
7. J. B. (M, 63) Carcinoma of lung	15	26	36/21	2.7	4.2	202	94.4
8. O. B. (M, 37) Conv. pneumonia	7	15	26/9	3.2	5.5	106	89.4
9. J. H. (M, 62) Hypertension	6	14	19/9	2.5	5.2	110	86.5
10. G. A. (M, 59) Cor pulmonale, diaphragmatic hernia	18	45	61/35	2.9	4.9	452	75.5
11. N. J. (M, 22) Conv. rheum. fever	10	14	22/8	3.3	5.5	57	90.9
12. N. M. (F, 42) Rheumatoid arthritis	7	17	24/11	2.6	4.1	209	91
13. A. H. (F, 56) HCVD	8	17	30/11	4.8	6.9	103	94.1
14. C. P. (M, 30) Conv. pneumonia	11	18	25/13	2.5	4.7	116	95.1
15. M. S. (M, 49) Mitral stenosis	23	54	70/30	2.0	3.7	666	86.5
16. M. B. (M, 60) HCVD, Silicosis	7	18	27/14	2.3	4.2	213	96.1
17. P. S. (M, 43) Malnutrition	4	10	18/7	3.7	5.9	85	99.3
18. J. R. (M, 36) Conv. pneumonia	4	16	23/9	3.0	5.5	162	89.8
19. W. B. (M, 37) Conv. pleurisy	7	13	21/9	4.5	8.0	62	89.6
20. C. M. (M, 49) Rheumatoid arthritis	7	18	26/9	3.0	5.1	170	90.3
21. J. S. (M, 39) Hemoptysis ? cause	7	19	32/10	2.6	4.8	189	83
22. L. J. (M, 20) Fibrosis lung	14	27	36/21	3.0	7.3	133	92.7
23. L. A. (M, 43) Neuritis, malnutrition	9	14	24/9	4.3	7.9	47	93.1
24. V. H. (M, 27) Epilepsy, lung abscess	6	10	15/6	3.2	5.1	69	95.6
25. E. B. (F, 28) Beri-beri	22	32	36/25	6.1	9.9	87	86.6
26. H. H. (M, 39) Bulbar paresis	9	15	16/7	3.0	4.9	83	95.6
27. F. H. (F, 38) Nutritional cirrhosis	7	16	23/10	5.4	7.6	93	92.3
28. G. Y. (M, 46) Conv. pneumonia	11	17	24/13	2.3	4.3	113	94.1
29. C. F. (M, 50) Conv. pneumonia	15	24	37/18	1.9	3.2	217	96.6
30. H. P. (M, 48) CNS lues	6	16	23/11	3.0	5.4	142	94.1
31. C. H. (F, 49) Paresis	8	20	23/10	3.4	5.8	160	96.3
32. S. M. (M, 40) Conv. pneumonia	7	14	21/9	3.1	5.2	101	96.5

TABLE I (*Continued*)

	Pulmonary "capillary" mean pressure, *mm. Hg*	Pulmonary artery pressure, *mm. Hg*		Cardiac output, *L/min.*		Pulmonary arteriolar resistance, *dynes sec./cm.*$^{-5}$	Arterial O_2 sat., *% of capacity*
		Mean	Systolic/diastolic	Index	Total		
33. E. J. (M, 43) Normal heart, chronic leukemia	7	16	24/12	3.3	6.5	113	93.7
34. M. L. (F, 52) Emphysema	6	21	34/16	2.4	3.6	337	89.3
35. F. B. (F, 20) Sickle cell anemia	13	19	25/10	5.6	8.6	56	83.0
36. R. J. (M, 69) Pneumoconiosis	7	17	30/10	2.1	3.6	223	89.1
37. F. J. (M, 46) HCVD, LMCA, thrombosis	11	23	34/12	2.1	4.2	214	97.4
38. O. M. (M, 39) HCVD, failure	17	35	50/29	1.5	2.4	593	94.9
39. A. B. (M, 46) Comp. HCVD, lues, optic atrophy	12	22	36/14	3.9	7.8	102	95
40. G. A. (M, 60) Cor pulmonale	19	74	119/51	1.8	3.3	1331	55.2
41. I. R. (M, 31) Mitral stenosis	19	29	45/18	2.3	3.9	211	90.8
42. C. G. (M, 66) Normal heart, frontal lobe atrophy	6	14	23/7	3.0	5.5	126	91.9
43. W. E. (M, 56) Carcinoma lung, multiple myotonia	16	35	52/28	3.7	5.9	256	73.7
44. J. M. (M, ?47) Pneumonia, carcinoma lung	8	12	22/7	3.6	5.7	53	90.8
45. E. M. (M, 37) Paresis	10	18	24/9	3.6	6.1	98	90.4
46. G. Y. (M, 48) Normal	6	17	24/8	4.7	8.9	104	92.2
47. F. T. (M, 52) Metastatic carcinoma lung	6	25	36/14	3.3	5.6	270	91.6
48. J. S. (M, 52) Emphysema	9	20	28/15	2.1	3.7	223	92.5
49. J. B. (M, 53) Emphysema, cor pulmonale	12	34	55/20	3.2	5.5	321	78.1
50. R. M. (M, 50) Paresis, luetic A.I.	8	14	25/10	2.3	4.2	110	100
51. H. F. (M, 43) Silicosis, ?pulmonary fibrosis	11	29	53/19	2.2	4.2	351	94
52. H. W. (M, 60) LMCA, thrombosis	5	12	20/8	2.2	3.3	150	95.9
53. H. S. (M, 51) Emphysema	6	32	64/17	3.2	4.8	415	85.6
54. C. D. (M, 26) Eisenmenger's tetralogy	9	102	140/80	2.2	4.2	1790	84.8

correlation coefficient for resistance and mean pressure was 0.94 ($p = < 0.001$).

When pulmonary "capillary" pressure was plotted against pulmonary arteriolar resistance on a logarithmic graph (Figure 4), no significant correlation was found ($r = 0.23$; $p = > 0.05$). In Figure 5, pulmonary arteriolar resistance is plotted against arterial oxygen saturation on semilogarithmic paper. A significant negative correlation was found ($r = -0.54$; $p = < 0.001$). Correlation between pulmonary artery diastolic pressure and pulmonary "capillary" pressure was determined and is shown on semilogarithmic paper (Figure 6). A significant correlation was observed ($r = 0.45$; $p = < 0.001$). No significant correlation between arterial oxygen saturation and cardiac output was observed (Figure 7; $r = 0.007$; $p = > 0.9$).

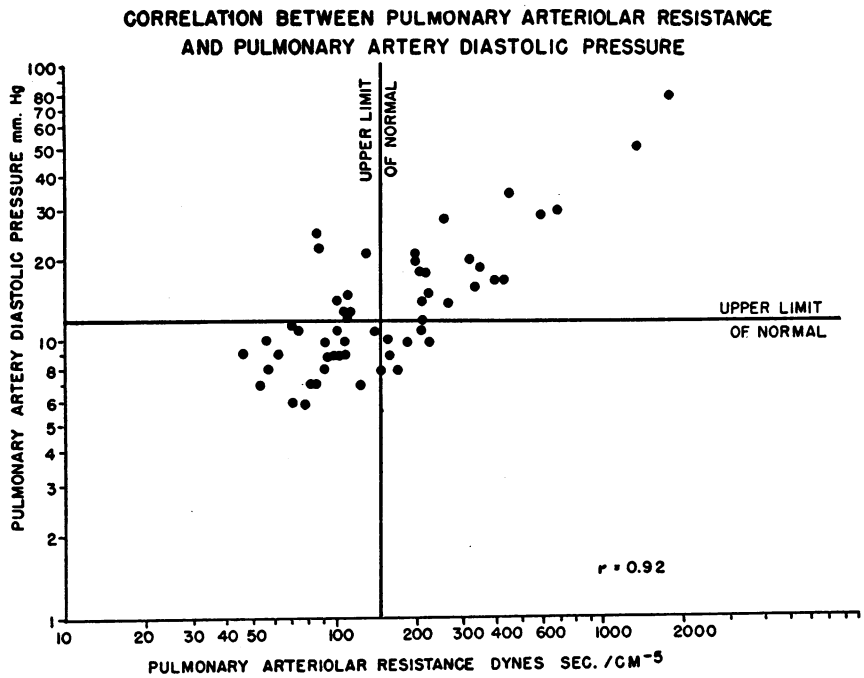

FIG. 1

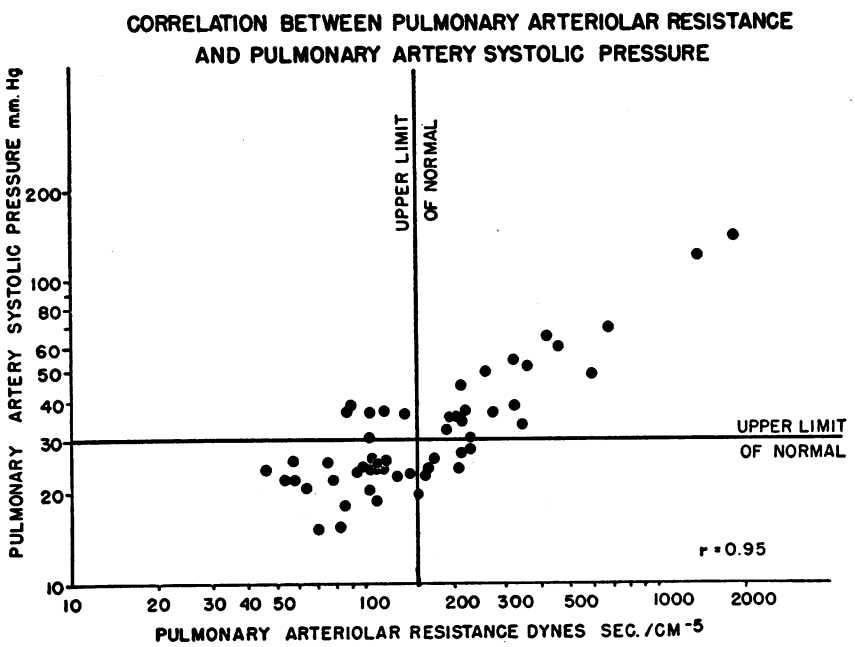

FIG. 2

DISCUSSION

It has been suggested that the elevation of pulmonary artery diastolic pressure commonly seen in emphysema is a reflection of the increased pulmonary vascular resistance found in that condition (5). Although this may well be true, it would appear from our data that it would be dangerous to predict arteriolar resistance from the pulmonary artery diastolic pressure. Although a high degree of correlation is shown between pulmonary

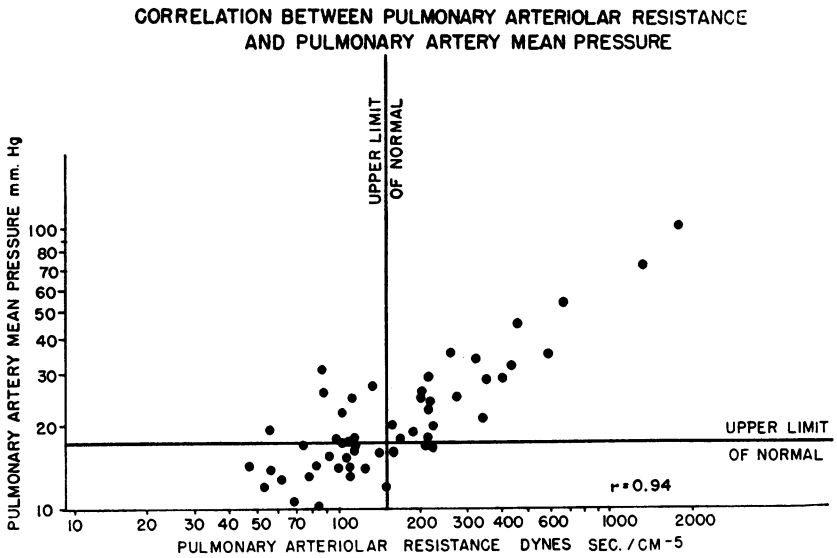

FIG. 3

artery diastolic pressure and pulmonary arteriolar resistance, there is considerable scatter; furthermore, eight patients had normal resistance with high diastolic pressure and seven patients had high resistance with normal diastolic pressure. In addition, an even higher degree of correlation with pulmonary arteriolar resistance was found with regard to pulmonary artery mean pressure and pulmonary artery systolic pressure than with regard to the pulmonary diastolic pressure. It must be borne in mind that the pulmonary artery pressure depends upon cardiac output and left atrial pressure as well as pulmonary vascular resistance. One would interpret the above findings to indicate that a rise in pulmonary arteriolar resistance increases the pulmonary artery pressure, and that

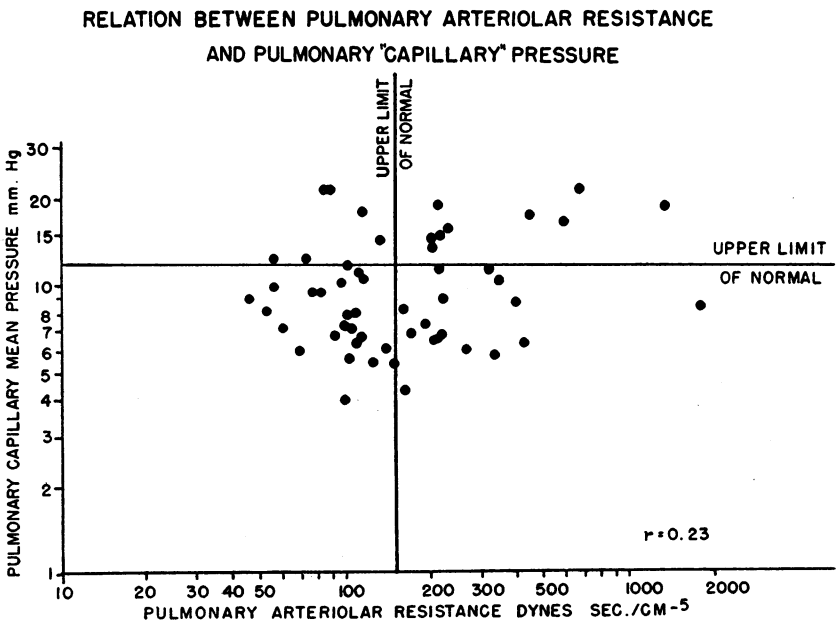

FIG. 4

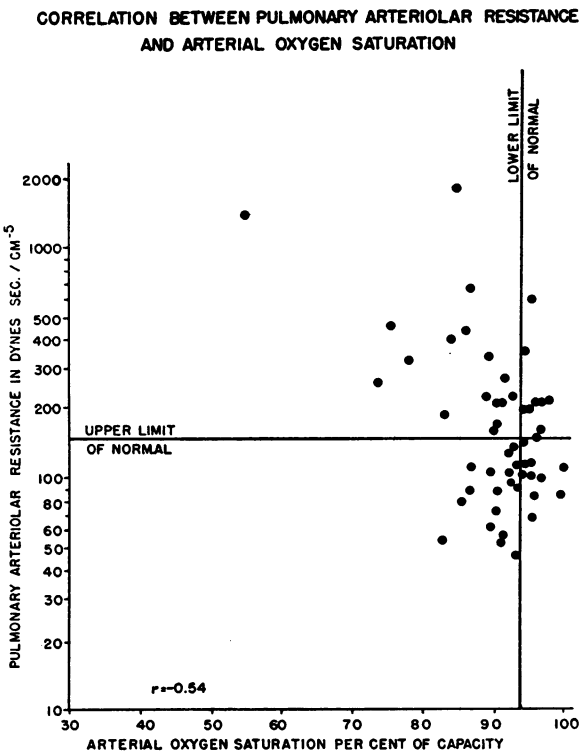

FIG. 5

the increase is as great or greater in the mean and systolic pressures than in the diastolic.

In view of the statement that a rise in left atrial pressure would be expected to produce an increase in pulmonary artery diastolic pressure (6), the significant correlation observed between this pressure and pulmonary "capillary" pressure is of interest since the latter is considered to vary with left atrial pressure. There was a fairly wide range of scatter here, however. It is also important to note that in only seven of the 54 subjects did pulmonary "capillary" pressure exceed the pulmonary artery diastolic pressure. Even in these instances the differences were small and could be explained by the error of the experimental method.

The failure to find correlation between the height of pulmonary "capillary" pressure and the amount of pulmonary arteriolar resistance does not necessarily conflict with the observations of Dexter (7), since our data contain no pulmonary "capillary" pressures above 25 mm. Hg at which level protective constriction of the pulmonary arteries is presumed to occur in order to prevent pulmonary oedema.

The finding of a significant negative correlation between arterial oxygen saturation and pulmonary arteriolar resistance is of interest, in view of the evidence that hypoxia produces pulmonary artery vaso-constriction (6, 8). We believe, however, that these data alone would not necessarily permit one to draw that conclusion. Correlation and causation are not the same. It is likely that conditions such as pulmonary emphysema which are commonly associated with arterial hypoxia produce pulmonary arterial hypertension through a mechanism of which hypoxia is but a part.

Our failure to find correlation between cardiac output and arterial oxygen saturation is in agreement with the observations of Borden and coworkers (5) who found no evidence of such correlation in 24 cases of emphysema, but is in disagreement with some theories as to the cause of high cardiac output in cor pulmonale (6). One should not conclude, however, that our results necessarily indicate no relation between cardiac output and hypoxia in emphysema, since our data were ob-

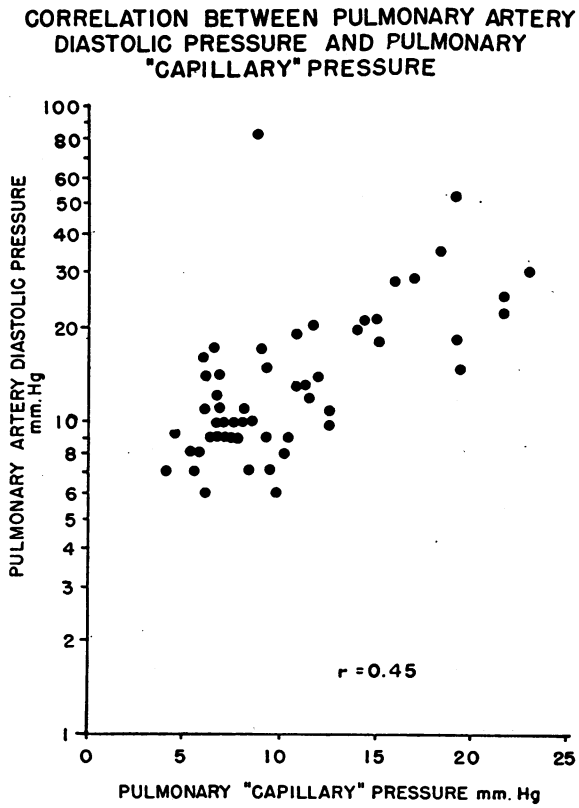

FIG. 6

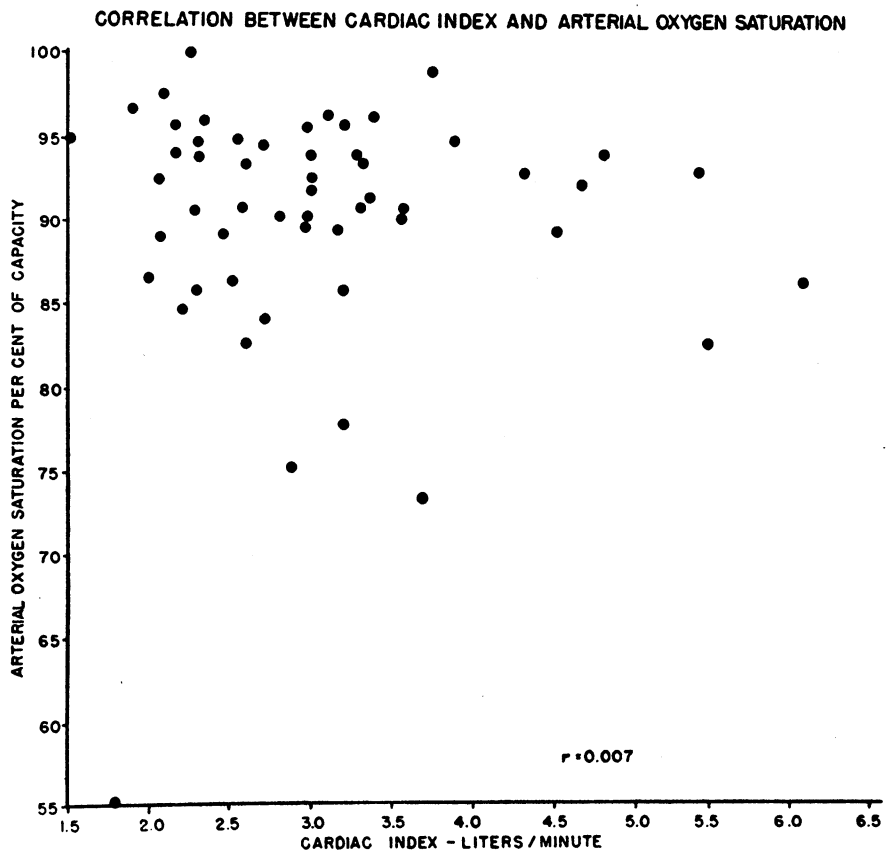

FIG. 7

tained from patients suffering from a variety of heart diseases. Unlike the heart in emphysema, the hearts in some of our subjects would be unable to respond to a stress situation by an increase in output.

SUMMARY

A study of pulmonary arteriolar resistance, pulmonary artery systolic, diastolic, and mean pressures, and pulmonary "capillary" pressure was made in 54 human subjects, some normal and some suffering from a variety of diseases. Significant correlation was found between pulmonary arteriolar resistance and each of the following: pulmonary artery systolic, mean, and diastolic pressures. The correlation of resistance with diastolic pressure was no closer than with mean or systolic pressure. A significant negative correlation between pulmonary arteriolar resistance and arterial oxygen saturation was found. Significant correlation between pulmonary "capillary" pressure and pulmonary artery diastolic pressure was observed. No significant correlation was found between arterial oxygen unsaturation and cardiac output. It is concluded that an increase in pulmonary arteriolar resistance produces an elevation in pulmonary artery mean, systolic, and diastolic pressures. There is no evidence that increased resistance produces a disproportionate elevation in pulmonary diastolic pressure as compared to mean and systolic pressures.

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