The presence of a blastema, therefore, is essential for the cessation of dedifferentiation and hence for the establishment of an equilibrium in the cellular interactions which occur during the initial phases of regeneration. This is true, however, only for young undifferentiated blastemas. When a transplanted blastema itself shows unmistakable signs of differentiation, it loses the capacity to control the dedifferentiation of a nerveless amputated limb.

13371 P

Lung Volume and Pulmonary Dynamics in Raynaud's Disease. Effect of Exposure to Cold.

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The occurrence of diffuse pulmonary fibrosis in patients with Raynaud's disease and scleroderma, though rare, has been noted in several instances.[1, 2] The question, therefore, arises as to whether the lung volume and pulmonary dynamics are abnormal in patients with Raynaud's disease and, more particularly, whether exposure to cold affects their pulmonary function. Three patients have been studied; all were females and ranged in age from 15 to 39 years. One of them (Case 3) had cough, dyspnea and X-ray evidence of diffuse pulmonary fibrosis of the type associated with Raynaud's disease.[2] No such findings were present in the other two. All studies were made by the method of Christie,[3] with the patients in the recumbent position. Measurements were made before and again after the patients' legs and at least one hand had been exposed to ice.

Observations. The lung volume and pulmonary dynamics in the 2 patients (Cases 1 and 2) with no clinical or X-ray evidence of pulmonary fibrosis were within normal limits. Case 3, in whom evidences of pulmonary fibrosis were present, exhibited a striking decrease in total pulmonary capacity, vital capacity and complemental air, with a lesser decrease in reserve air; the residual and functional residual airs were within normal limits. The respiratory rate and minute volume were increased.

1 Matsui, S., *Mitt. a. d. med. Fakult. d. k. Univ. zu Tokyo*, **1924**, **31**, 55.

2 Linenthal, H., and Talkov, R., *New Eng. J. Med.*, 1941, **224**, 682.

3 Christie, R. V., *J. Clin. Invest.*, 1932, **11**, 1099.

Exposure to cold caused no change in lung volume or its subdivisions in any of the patients. The respiratory minute volume increased slightly. In addition to the studies here detailed, similar measurements of pulmonary function were made in one case out of doors with the temperature at 40-50°F, with no difference in results.

Discussion. The 2 patients (Cases 1 and 2) with no clinical or X-ray evidence of pulmonary disease showed no physiological evidence of impaired respiratory function. This was in contrast to the findings in the patient with cough, dyspnea and X-ray evidence of fibrosis of the lungs. The complemental air in this patient was greatly decreased, indicating marked impairment of pulmonary distensibility, due apparently to an increased amount of fibrous tissue in the lungs. Attempts to increase the volume of the tidal air in response to the increased demands of exertion would require abnormally strenuous effort and would be only partly successful, thereby leading to dyspnea. Fibrosis also decreases the elasticity of the lungs and further impairs the efficiency of respiration, particularly in regard to expiration; active muscular effort might be required for effective expiration.

The possibility that the increased respiratory activity exhibited by Case 3 might be due to reflexes activated by pathological changes in the pulmonary parenchyma[4] was ruled out by the repeated finding of normal alveolar carbon dioxide concentrations of 5.4 to 5.6%. Dyspnea due to such reflexes is accompanied by a blowing off of carbon dioxide with a resultant decrease in alveolar carbon dioxide concentration. It is probable that the increased respiratory rate and minute volume exhibited by this patient at rest was compensatory to

TABLE I.

Case	Age	Functional residual air cc	Residual air cc	Reserve air cc	Complemental air cc	Vital capacity cc	Total capacity cc	Tidal air cc	Respiratory rate	Respiratory minute volume Liters	Oxygen consumption cc/min	Exposure
1	15	1810	1135	675	2420	3095	4905	260	22	5.7	216	Before
		1695	1100	595	2630	3225	4930	290	22	6.4	207	During
2	24	2310	2030	280	1625	1905	3935	375	18	6.8	180	Before
		2220	1940	280	1585	1865	3805	385	20	7.7	189	During
3	39	1885	1685	200	555	755	2440	345	25	8.6	228	Before
		1910	1705	200	595	795	2500	355	26	9.3	207	During

[4] Porter, W. T., and Newburgh, L. H., *Am. J. Physiol.*, 1917, **43**, 455.

the lowered efficiency of respiration resulting from impaired distensibility and elasticity. That this compensation was adequate at rest is witnessed by the fact that the arterial blood oxygen saturation was normal, *i. e.*, 94%.

It should be noted that clinical manifestations and physiological measurements indicative of seriously impaired pulmonary function were present months before unequivocal X-ray evidence of fibrosis was detected. Roentgenograms at no time reflected the severity of the pulmonary lesion in this case. This is probably due to the fact that the fibrosis is diffuse, with no large masses or nodules of collagen being formed.

The fact that the lung volume and its subdivisions were not changed when the patients were exposed to cold indicates that the blood vessels of the lungs do not react in a manner similar to those of the hands and feet. This conclusion was strengthened by the observation that exposure to cold did not result in a change in the circulation time through the lungs.

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Renin-Like Substance in Blood after Hemorrhage.*

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The stimulus adequate to cause the isolated perfused kidney to release renin has been shown by Kohlstaedt and Page[1] to be a fall in the pulse pressure. It seems probable that a similar fall in pulse pressure in the intact animal would affect the kidney similarly. Wakerlin and Chobot[2] were unable, however, to find any change in the renin content of kidneys after their dogs had been subjected to either hemorrhage or splanchnic stimulation. In view of the considerable variation in the renin content of normal kidneys, and of the possibility that the amount of renin liberated is very small in relation to the amount originally present in the kidney, these results

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1 Kohlstaedt and Page, *J. Exp. Med.*, 1940, **72**, 201.

2 Wakerlin and Chobot, PROC. SOC. EXP. BIOL. AND MED., 1939, **40**, 331.