per cent and arterial rise time. In the presence of a fixed amount of intrapulmonary shunting, for example, it could be expected that hyperventilation, while affecting the arterial rise time, would have a minimal effect on T_{a-A} 90 per cent, since both alveolar and arterial rise times would decrease. However, T_{a-A} 90 per cent would be expected to decrease with hypoventilation (either generalized or regional), since prolonged alveolar washin would tend to minimize the effects of intrapulmonary shunting.

The effects of changes in cardiac output on T_{a-A} 90 per cent were not demonstrated in the present study; yet, under certain conditions (*i.e.*, a large intrapulmonary shunt) these changes would be expected to have a significant effect. Reduced $C\bar{v}_{O_2}$ secondary to a decreased cardiac output would increase T_{a-A} 90 per cent by further contamination of arterial blood through the shunt. Similarly, an increased cardiac output would decrease T_{a-A} 90 per cent. An increase in cardiac output then, in the presence of intrapulmonary shunt, would improve the efficiency of perfusion relative to ventilation.

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References

1. Briscoe, W. A., and Cournand, A.: Uneven ventilation of normal and diseased lungs studied by an open-circuit method, J. Appl. Physiol. 14: 284, 1959.
2. Nye, R. E., Jr.: Theoretical limits to uneven ventilation, J. Appl. Physiol. 16: 1115, 1961.
3. Theye, R. A.: Blood O_2 content measurement using the O_2 electrode, ANESTHESIOLOGY 28: 773, 1967.
4. Colgan, F. J., and Mahoney, P. D.: The effects of major surgery on cardiac output and shunting, ANESTHESIOLOGY 31: 213, 1969.
5. Colgan, F. J., Whang, T. B., and Gillies, A. J.: Atelectasis and pneumothorax: Effect on lung functions and shunting, ANESTHESIOLOGY 29: 923, 1968.
6. Said, S. M., Davis, R. K., and Crosier, J. L.: Continuous recording in vivo of arterial blood P_{O_2} in dogs and man, J. Appl. Physiol. 16: 1129, 1961.
7. Staub, N. C.: Alveolar–arterial oxygen tension gradient due to diffusion, J. Appl. Physiol. 18: 673, 1963.
8. Briscoe, W. A., Cree, E. M., Filler, J., Houssay, H. E. J., and Cournand, A.: Lung volume, alveolar ventilation and perfusion interrelationships in chronic pulmonary emphysema, J. Appl. Physiol. 15: 785, 1960.
9. Finley, T. H.: The determination of uneven pulmonary blood flow from the arterial oxygen tension during nitrogen washout, J. Clin. Invest. 40(2): 1727, 1961.

Drugs

PARATHYROID POLYPEPTIDES Thyrocalcitonin, a hormone which induces hypocalcemia and hypophosphatemia, has been isolated, analyzed for amino-acid sequence, and synthesized chemically. It is secreted from the perifollicular cells found in mammalian thyroid but related embryologically to the ultimobranchial body of lower vertebrate species. Sensitive radioimmunoassays for thyrocalcitonin and parathyroid hormone have shown that secretion of each is controlled by calcium. Current findings show that the major physiologic parathyroid hormone actions are mediated by adenosine 3′,5′-monophosphate (cyclic AMP) produced as a consequence of direct, specific hormonal stimulation of the enzyme adenyl cyclase in bone and kidney. Thyrocalcitonin acts through another mechanism to inhibit bone resorption. In healthy subjects, but not in patients with pseudohypoparathyroidism, parathyroid hormones cause a marked increase in urinary excretion of cyclic AMP. This observation forms the basis for a useful diagnostic test and suggests that the metabolic abnormality of pseudohypoparathyroidism may be attributable to a genetic lack or defect of a specific adenyl cyclase in renal and skeletal tissue. (*Aurbach, G. D., and others: Polypeptide Hormones and Calcium Metabolism, Ann. Intern. Med. 70: 1243 (June) 1969.*)