albumin in the sputum of the non-infected patients was 6-48 times that in the serum, suggesting that local mechanisms exist preferentially to concentrate this protein.

Two-dimensional immunoelectrophoresis studies of the sputum samples showed that various proportions of α1-antichymotrypsin are present as protein/enzyme complex even in the absence of infection. Several of the infected samples showed indistinct precipitation arcs of the inhibitor with increased electrophoretic mobility, compared with the serum protein. This suggests the presence of damaged protein in the presence of infection. The implications of these findings will be discussed.

21. PRODUCTION OF 6-KETO PGF1α BY HUMAN LUNG IN VIVO
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The synthesis of prostacyclin (PGI2), a potent vasodilator and anti-aggregating prostanoid, has been described for guinea-pig [Dawson, Boot, Cockerill, Mallen & Osborne, 1976, Nature (London), 262, 697], rabbit [Moncada, Gryglewski, Bunting & Vane, 1976, Nature (London), 263, 663] and dog (Barnes, Dollery & Hensby, 1979, Fourth International Prostaglandin Conference Proceedings, Washington, p. 8) lung preparations. We now report that the human lung in vivo produces 6-keto PGF1α, a hydrolysis product of PGI2.

Five subjects (37 ± 6.8 years, mean ± SEM) were studied during diagnostic cardiac catheterization. Simultaneous samples of blood (50 ml) were taken from the pulmonary artery and left ventricle and the plasma obtained after centrifugation (4°C) was assayed for 6-keto PGF1α by using a quantitative gas chromatographic–mass spectrometric assay previously described (P. Barnes, C. T. Dollery & C. N. Hensby, 1979, Presentation at British Pharmacological Society, Leeds. 12–14 September) to determine the transpulmonary difference.

In all subjects the concentration of 6-keto PGF1α was greater in the left ventricle (207 ± 33 pg/ml; mean ± SEM, n = 5, P < 0.05) than the pulmonary artery (131 ± 13 pg/ml; mean ± SEM, n = 5), indicating that the lung was secreting prostacyclin into the cardiovascular system. This is in contrast to the removal of most prostaglandins including PGE2 and PGF1α, from the pulmonary circulation [Ferreira & Vane, 1967, Nature (London), 216, 868].

The potential physiological significance of this prostacyclin release in preventing platelet aggregation may explain why the arterial circulation is normally protected from thrombus formation. Factors influencing the prostacyclin release, for example cigarette smoking and the oral contraceptive, are at present under investigation in an effort to explain some of the pathological effects that they have upon the cardiovascular system.

22. COMPARISON OF THE EFFECT OF PROPRANOLOL WITH ATENOLOL AND METOPROLOL ON THE DOSE–RESPONSE CURVES OF CATECHOLAMINE-INDUCED CHANGES IN VENTILATION
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The hyperventilatory response to infused noradrenaline and isoprenaline can be blocked by propranolol (Heistad, Wheeler, Mark, Schmid & Abboud, 1972, Journal of Clinical Investigation, 51, 1469–1475). The specificity of the β-receptor mediating this response is not known.