

Prostaglandins and the cardiovascular system

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Our perception of the role of prostaglandins in the cardiovascular system changed dramatically in the 1970's with the discoveries of thromboxane A_2 and prostacyclin (for more extended reviews, see Moncada and Vane,¹ Moncada,² and Vane³). Both of these compounds are chemically unstable and share, in common with the other prostaglandins, the same precursor, arachidonic acid. This fatty acid is released from cell membrane phospholipids by chemical or mechanical perturbations and is then metabolised by a cascade of enzymes to the stable prostaglandins such as PGE_2 and $PGF_{2\alpha}$, thromboxane A_2 (TXA_2), or prostacyclin (PGI_2). On the one hand, in platelets arachidonic acid is mainly converted to thromboxane A_2 , an unstable (half life 30 seconds) vasoconstrictor and platelet aggregating substance. On the other, in the vessel wall arachidonic acid is converted to prostacyclin (PGI_2), an unstable (half life two to three minutes) vasodilator and anti-aggregating substance. We have proposed, therefore, that prostacyclin is the natural and main defence of the vessel wall against deposition of platelet aggregates. Further, prostacyclin and thromboxane A_2 represent in biological terms the opposite poles of a homeostatic mechanism for the regulation of platelet aggregability *in vivo*.⁴ Clearly, manipulation of this control mechanism is likely to affect thrombus and haemostatic plug formation.

Apart from platelets, some other tissues have also been shown to generate TXA_2 , including phagocytosing leucocytes. The role of TXA_2 in tissues other than platelets is uncertain.

Prostacyclin is the main product of arachidonic acid in all arteries and veins so far tested. Not much is known about the microcirculation but microvessels, mainly capillaries, isolated from rat cerebrum generate predominantly prostacyclin.⁵

The ability of the large vessel wall to synthesise prostacyclin is greatest at the intimal surface and progressively decreases towards the adventitia.⁶ Use of cultured cells from vessel walls additionally shows that endothelial cells are the most active producers of prostacyclin. Other cells, including myocytes in culture and mesenchymal cells (probably fibroblasts) also produce prostacyclin, as does the pericardium and the

epicardial surface of the heart. Since the release of prostaglandins is increased by any distortion of cell membranes, it is interesting to speculate that with each beat of the heart the stretching of the parietal and visceral pericardium leads to a release of prostacyclin which may act as a natural lubricant between them. In this respect, the third activity of prostacyclin (termed cytoprotection when applied to the reduction of experimentally induced ulcers in the stomach) could be important. The possibility also exists that any prostacyclin released will condition the surface coronary vessels and perhaps also be responsible for preventing the clotting of blood spilt into the pericardial sac.

Prostacyclin has been implicated in functional hyperaemia, the main evidence for which is in the gastric mucosa,⁷ where prostaglandin synthetase inhibitors such as aspirin and indomethacin reduce mucosal blood flow and cause erosions. The aspirin-like drugs, however, do not cause vasoconstriction in the normal coronary circulation either of animals or man, indicating that a continuous production of prostacyclin is not having a vasodilator activity in the normal heart. Interestingly, Friedman *et al.*⁸ found in nine patients with coronary artery disease (eight with angina) that indomethacin increased the blood pressure by an average of 20 mm, increased myocardial oxygen demand, and decreased coronary flow by 40%. The results are complicated by the fact that the patients were receiving beta-blockers and nitrates, each of which could be a source of interactions with indomethacin, but they do suggest that in coronary vascular disease the endogenous release of prostacyclin takes part in the struggle to maintain the coronary circulation.

There is also experimental evidence that exogenous prostacyclin has a beneficial effect in the acutely ischaemic heart. Infusions of prostacyclin intravenously have an antiarrhythmic effect in experimental infarction⁹ and also limit infarct size (in dogs and cats) and reduce lysosomal enzyme release. Several studies have now confirmed the original observations of Lefer *et al.*¹⁰ including that of Ribeiro *et al.*¹¹ in which they showed a substantial decrease in infarct size and mortality in dogs treated with intravenous prostacyclin for

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