Effect of propranolol on hypoxia induced myocardial vasodilatation

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On the basis that sotalol markedly reduced, or even reversed, the coronary vasodilatation which results from systemic hypoxia in open chest dogs, Folle & Aviado (1965) suggested that β-adrenoceptor blocking drugs in general might seriously interfere with the self-regulating control of myocardial oxygen supply in patients with coronary artery disease. This conclusion clearly has far reaching implications and, since the experimental procedures used by these authors involved considerable surgery, it seemed important to re-examine the effects of β-adrenoceptor blockade on the responses of the myocardial vessels to a decreased arterial oxygen tension.

Anaesthesia was induced in seven dogs with intravenous sodium thiopentone and, after endotracheal intubation, was maintained using 0·5–1·0% trichlorethylene. Under fluoroscopic control, catheters were placed in the coronary sinus, right atrium, descending aorta and either the circumflex or the anterior descending branch of the left coronary artery. Myocardial blood flow was measured using a ^{133}xenon clearance technique (Ross, Ueda, Lichtlen & Rees, 1964; Ledingham, McBride, Parratt & Vance, 1970) with the scintillation counter placed over the praecordium. Hypoxaemia induced by rapidly reducing the inspired oxygen content to 10–11% (arterial pO_2 27 mmHg) increased myocardial blood flow [from (119±10 to 184±8 ml/100g)/min] and mean systemic blood pressure (112±5 to 133±10 mmHg), decreased heart rate (139±10 to 122±11 beats/min) and myocardial vascular resistance and had no significant effect on myocardial oxygen consumption [(10·3±0·8 to 9·6±1·4 ml/100g)/min]. Although a combination of propranolol (0·2 mg/kg) and atropine (0·04 mg/kg) markedly decreased each of these parameters, the cardiovascular responses to hypoxia were essentially unaltered by these drugs. Thus myocardial blood flow was increased by hypoxia from (89±10 to 158±9 ml/100g)/min and mean blood pressure from 96±8 to 117±8 mmHg. Heart rate and myocardial vascular resistance were again reduced. These results indicate that coronary vasodilatation, under our conditions of hypoxia, is due to an effect of the lowered oxygen tension (or the release of some vasodilator metabolite) on vascular smooth muscle and there is no support for the contention of Folle & Aviado (1965) that β-adrenoreceptor blocking drugs interfere with hypoxic induced coronary vasodilatation.

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Effects of β-adrenoceptor blocking drugs on the chronotropic and inotropic actions of isoprenaline on the acutely denervated dog heart

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The effect of β-adrenoceptor blocking drugs on the dog heart is well known (e.g.