Tissue oxygen pressure in normal myocardium and across the border zone during coronary artery occlusion in the pig. Effects of different arterial oxygen pressures

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Summary: Tissue oxygen pressure (pO₂) in the pig heart was measured with two different oxygen electrodes: We measured pO₂ in normal myocardium with the MDO electrode while oxygen gradients across the border zone, during acute coronary artery occlusion, were measured with an array-multiwire-electrode (AME). The aim of the study was to investigate the effects of increased arterial oxygen pressure (pO₂) during repeated, short-lasting (5 min) coronary artery occlusions.

During ventilation with an inspired oxygen fraction (FIO₂) of 0.3 the pO₂ levels in normal myocardium increased significantly, while the distribution type of pO₂ values remained normal. During ventilation with FIO₂ 0.7 there was an uneven distribution of pO₂ values indicating microcirculatory disturbances, however, no ischemic values were seen. We found no indication for any influence on the pO₂ of the border zone.

Key words: coronary occlusion; myocardial ischemia; border zone; oxygen pressure; oxygen administration

Introduction

For many years patients with acute myocardial infarction have been given oxygen in varying concentrations. This is based on the assumption that an increase in arterial oxygen pressure (pO₂) leads to an increase in myocardial oxygenation and also to increased oxygen delivery to peripheral tissues when the circulation is inadequate. Studies from the late 1960's indicate undesirable hemodynamic effects during oxygen administration to patients with myocardial infarction (28, 31). Oxygen administration via a face mask was shown to decrease cardiac output due to a reduction in both heart rate and stroke volume, and to elevate the peripheral vascular resistance (1). It was also found that coronary vascular resistance increased (2).

Neill (23) compared the effects of both arterial hypoxemia and hyperoxemia on myocardial metabolism in patients both with and without coronary artery disease. He found evidence for a disadvantageous effect of both hypo- and hyperoxemia in the form of increased production of lactate under these conditions. In contrast, Maroko et al. (21) in an experimental study found signs of infarct size reduction during oxygen administration. They used an inspired oxygen fraction (FIO₂) of 0.4 and found no further improvement by "pure oxygen breathing" (FIO₂ 1.0). In a later study using a thermographic technique, Malm et al. (20), in experiments on dogs subjected to coronary artery occlusion, found evidence for an enlargement of the ischemic area as an effect of pure oxygen breathing. Several investiga-
tions indicate negative effects of oxygen administration upon various hemodynamic parameters (8, 9, 30, 36, 37). However, oxygen is still widely administered.

We have previously investigated the border zone, i.e., the myocardial tissue that constitutes the transition zone from normal to ischemic myocardium during acute coronary artery occlusion, in an experimental model using pigs (33, 35). We used an array-multiwire-electrode (AME), which is a multiwire surface oxygen pressure sensor with eight individual measuring points arranged in an array (32). We found that the border zone in the pig heart was very narrow, a finding that was in agreement with other investigators (4, 25). The border zone did not appear homogeneous or extremely sharp, in contrast to propositions that the zone might even be cell-to-cell sharp (5).

The aim of the present study was to investigate whether or not increases in FIO2 influenced the border zone. We also wanted to study simultaneous changes in tissue oxygen pressure (pO2) in normal myocardium during changes in FIO2.

Material and methods

Seven Swedish land-race pigs, 28 to 32 kg b.w., were used. Anesthesia was induced with azaperone 4 mg/kg (Stresnil) and atropine 0.05 mg/kg intramuscularly, followed by an initial dose of 15 mg/kg metomidate (Hypnodil) intramuscularly. Metomidate was then given as a continuous infusion in normal saline. Muscle relaxation was achieved with pancuronium (Pavulon), also given as a continuous infusion. A tracheotomy was performed and the animals were ventilated with ambient air with an Engström ventilator. Positive end-expiratory pressure (PEEP) was kept at 5 cm H2O to prevent pulmonary atelectasis. Arterial blood was drawn for gas analysis (ABL-3, Radiometer) and respiratory volume was adjusted to keep the arterial carbon dioxide pressure (paCO2) within normal range (4.4-5.5 kPa (31)). Catheters were placed in a superficial vein and in an external jugular vein, and a pig-tail catheter was placed via the common carotid artery in the left ventricle in a position inducing no persistent arrhythmias. Systolic and end-diastolic left ventricular pressures as well as ECG were registered with a Mingograf recorder (Siemens-Elema).

A midsternal thoracotomy was performed and the heart was suspended in a pericardial cradle to minimize movement due to respiration. A 2-0 silk ligature was placed around the left anterior descending coronary artery (LAD) after the third diagonal branch or in a corresponding position, i.e., approximately two-thirds of the distance from the base to the apex. The LAD was then occluded temporarily for 40-90 s to allow for development of cyanosis in the area distal to the occlusion. The area of cyanosis was thereby clearly delineated from normal tissue. For tissue oxygen pressure measurements the MDO-electrode (Mehrdraht Dortmund Oberfläche) (10, 12) and the AME (array-multiwire-electrode) (33) were used.

Positioning the electrodes on the ventricular surface required a specially designed holding system. This was sutured onto the left ventricle with 5-0 silk sutures. This holding system allowed for controlled movements of the AME along a line 15 mm long. All rotation was effectively prevented. The AME was positioned at the sharp demarcation between cyanotic and normal myocardium, which had been noted after the first short occlusion of the LAD. The artery was again closed to ensure both normal and ischemic tissue present in the measuring area of the electrode. This required only a short-lasting occlusion of 15-45 s. The electrode was then left on the ventricular surface for 45 min of stabilization, and during this time the LAD was left open.

At one end of the holding system, as far away from the ischemic tissue as possible, a holder for the MDO-electrode was mounted in the plastic film. The shortest possible distance from the MDO-electrode to the AME, and thus to the border zone, was 12 mm and the longest was 27 mm. The two electrodes were calibrated immediately after one another and were allowed 45 min of stabilization on the left ventricular surface before any measurements were carried out.

Two ABC 800 computers (Luxor, Sweden) were used. The signals from each electrode were fed via an amplifier into the computers where the pO2-values were calculated (24). The computer program for the AME was designed so that each one of the eight platinum wires was identified. Sampling was done from all eight measuring points simultaneously and time intervals between samplings could be chosen