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We investigated the changes of coronary sinus blood flow (CSBF) and myocardial oxygen consumption (MVO_2) before and during anginal pain (AP) and to find the correlations between these parameters with indices of myocardial oxygen consumptions in 13 chronic stable angina who could provoke AP by bicycle ergometer. Brachial arterial (BA) pressure was obtained by an Amplatz needle and CSBF was measured by thermodilution technique. Bloods were sampled from both BA and CS to calculate MVO_2 . Coronary vascular resistance (CVR) was calculated by the formula of mean BAP/CSBF. When AP were provoked, CSBF was decreased from 206 ± 56 ml/min (mean SD) to 183 ± 67 (NS, $n=10$) and MVO_2 was decreased from 24.3 ± 7.5 mmHg/min to 20.6 ± 8.6 ($p < 0.05$, $n=8$), whereas CVR was increased from 0.63 ± 0.17 mmHg/ml/min to 0.74 ± 0.18 ($p < 0.01$, $n=10$) and pressure rate product (PRP) was increased from 242 ± 36 mmHg.bpm $\cdot$ min to 256 ± 48 (NS, $n=9$). Correlations of CSBF with MVO_2 at all determinations was 0.87 ($p < 0.01$, $n=72$), with PRP was 0.86 and with HR $\times$ mean BA was 0.70 , whereas correlations of MVO_2 with PRP was 0.67 and HR $\times$ mean BA was 0.69 . These data suggested that (1) during angina, reduction of MVO_2 and increment of CVR were more remarkably changed than CSBF and PRP, (2) lower values of correlations between CSBF and MVO_2 with indices of MVO_2 comparing with previous reporters were probably due to the difference of coronary bed anatomy.

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ROLE OF ADENOSINE IN REGULATION OF CORONARY BLOOD FLOW IN PATIENTS WITH EFFORT ANGINA

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To test the hypothesis that adenosine plays a pivotal role in the regulation of coronary blood flow at rest and during increased myocardial oxygen demand in coronary artery disease, adenosine release (ADR) into the coronary vein was determined at rest and during rapid atrial pacing in 14 patients (pts) with effort angina (EA), who had severe narrowings in the LAD artery. Seven pts with normal coronary arteriograms were also studied as the controls. The great cardiac venous flow (GCF, thermodilution), lactate extraction and ADR were determined before and during pacing. In resting state, the average ADR ($1.93 \pm .29$ n mol/min, $\pm$ SE) in pts with EA was significantly higher than that ($.27 \pm .29$ n mol/min) in controls. During pacing, ADR increased in the pacing-rate dependent manner in both groups. However, ADR in pts with EA was significantly higher at any pacing rate, although increases in GCF were not significantly different. At maximal pacing, which induced anginal pain and lactate production, a marked ADR was observed (8.70 ± 1.87 n mol/min). In 9 pts who underwent PTCA, ADR decreased ($p < 0.01$) both at rest and during pacing. These results indicate that in patients with severe coronary narrowings, adenosine plays a major role in the regulation of coronary blood flow both at rest and when increased myocardial oxygen demand.

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RELEASE OF ADENOSINE AND LACTATE FROM THE HEART DURING ATRIAL PACING IN PATIENTS WITH ISCHEMIC HEART DISEASE

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In patients with coronary stenosis, an increase in oxygen demand produces anginal pain and release of lactate from the heart. Animal experiments have revealed that myocardial ischemia caused release of adenosine as well. Therefore, we examined whether human hearts with coronary stenosis release adenosine during angina induced by atrial pacing. Patients who were diagnosed or doubted as having ischemic heart disease were subjected to routine cardiac catheterization. The patients were divided into three groups, group I: patients without coronary stenosis, who had no anginal pain even after pacing; group II: patients without coronary stenosis having anginal pain on pacing; and group III: patients with coronary stenosis having anginal pain on pacing. In group I, there was no significant release of adenosine and lactate during pacing, although heart rate was increased by pacing from 74.5 to 150.0 beats/min. In group II, heart rate was increased by pacing from 71.6 to 144.4 beats/min. Adenosine and lactate were released but insignificantly. In group III, pacing increased heart rate from 66.1 to 130.0 beats/min, and produced release of adenosine and lactate significantly, the release of adenosine being more marked than that of lactate. In conclusion, human hearts with coronary stenosis release a significant amount of adenosine and lactate during pacing-induced angina.

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INTRAOPERATIVE MEASUREMENT OF MYOCARDIAL BLOOD FLOW BY ELECTROLYTIC HYDROGEN GAS CLEARANCE METHOD

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We have measured MBF by electrolytic hydrogen gas clearance method intraoperatively and evaluate the effect of coronary revascularization.

Seventeen cases with IHD (AP:OMI = 10:7) and 8 with non-IHD as control are studied. MBF measurement was performed before and after cardiopulmonary bypass at the LV ant. wall (LAD region) and RV ant. wall.

MBF of non-IHD group was 176 ± 27 ml/min/100gr and non-ischemic area (under 75% stenosis) of IHD group was 186 ± 48 ml/min/100gr. Whereas MBF of ischemic area (over 75% stenosis) was 131 ± 100 ml/min/100gr, which was significantly low ($p < 0.05$). Preoperative MBF at LAD region with or without OMI was 80 ± 55 ml/min/100gr and 157 ± 52 ml/min/100gr. Severity of LAD stenosis and MBF was evaluated. In 75% stenosis MBF of 195 ± 40 ml/min/100gr was ob-