更易于及时发现高危期，且对于预防性治疗的开始非常重要。

0020. Residual arachidonic acid-induced platelet activation via an adenosine diphosphate-dependent but cyclooxygenase-1- and cyclooxygenase-2-independent pathway: A 700-patient study of aspirin resistance

Frelinger III A. L. / Furman M. I. / Linden M. D. et al. [Dr. M. I. Furman, Division of Cardiovascular Medicine, UMass Memorial Medical Center, 55 Lake Ave N, Worcester, MA 01655, United States] – CIRCULATION 2006, 113/25(2888 – 2896)

BACKGROUND - Thrombotic events still occur in aspirin-treated patients with coronary artery disease. METHODS AND RESULTS - To better understand aspirin "resistance," serum thromboxane B2(TXB2) and flow cytometric measures of arachidonic acid-induced platelet activation(before and after the ex vivo addition of aspirin and indomethacin) were analyzed in 700 consecutive aspirin-treated patients undergoing cardiac catheterization. In 680 of 682 evaluable patients, serum TXB2 concentrations were reduced compared with nonaspirinated healthy donors. Twelve patients had serum TXB2 that was lower than nonaspirinated healthy donors but > 10 ng/mL. Arachidonic acid stimulated greater platelet activation in patients with high serum TXB2(> 10 ng/mL) than in patients with low serum TXB2. Addition of ex vivo aspirin reduced arachidonic acid-induced platelet activation to similar levels regardless of serum TXB2 concentrations, which suggests that patients with high residual serum TXB2 concentrations were either noncompliant or underdosed with aspirin. Among the remaining 98% of patients, ex vivo administration of either aspirin or indomethacin failed to prevent platelet activation across all degrees of arachidonic acid-induced platelet activation and aspirin doses. Although the patients were not randomized with respect to clopidogrel treatment, multivariate analysis showed that arachidonic acid-induced platelet activation was less in patients receiving clopidogrel. CONCLUSIONS - There is a residual arachidonic acid-induced platelet activation in aspirin-treated patients that(1) is caused by underdosing and/or noncompliance in only ≈2% of patients and(2) in the remaining patients, occurs via a cyclooxygenase-1 and cyclooxygenase-2 independent pathway, in direct proportion to the degree of baseline platelet activation, and is mediated in part by adenosine diphosphate-induced platelet activation.

残留花生四烯酸诱发的血小板激活是通过二磷酸腺苷依赖性、环氧合酶1和2非依赖性途径所致：一项包括700例患者的阿司匹林抵抗研究

背景：接受阿司匹林治疗的冠心病患者中仍可出现血栓事件。方法和结果：为了更好理解"阿司匹林抵抗"，作者连续观察了700例进行阿司匹林治疗且接受心脏导管检查的患者，分析其血清血栓素B2(TXB2)，并进行流式细胞检查以确定花生四烯酸诱发的血小板激活(分别在离体加入阿司匹林和吲哚美辛之前和之后)。682例可进行评估的患者中，有680例较之未服用阿司匹林的健康志愿者其血清TXB2浓度有所降低。12例患者的血清TXB2低于健康志愿者，但 > 10 μg/L。与血清TXB2浓度低的患者相比，血清TXB2较高(> 10 μg/L)的患者中花生四烯酸诱发的血小板激活程度更大。而不论其血清TXB2浓度如何，体外加入阿司匹林可减少花生四烯酸所诱发的血小板激活程度至相似水平，这提示具有较高血清TXB2浓度的患者服用阿司匹林的依从性不佳或药物剂量不足。在其余98%患者中，无论花生四烯酸诱发的血小板激活程度如何或阿司匹林剂量如何，体外加入阿司匹林或吲哚美辛未能阻止血小板激活。虽然未就服用氯吡格雷方面对患者进行随机化，但多因素分析显示花生四烯酸诱发的血小板激活程度在服用氯吡格雷的患者中更低。结论：在接受阿司匹林治疗的患者中存在残留花生四烯酸诱发的血小板激活；仅在约2%的患者中是由于阿司匹林服用剂量不足或服用依从性不佳所致，而在其余的患者中是通过环氧合酶1和环氧合酶2非依赖性途径所致，与基线血小板激活的程度成正比，部分是由二磷酸腺苷诱导的血小板激活所介导。

(0014～0020 杜 媛 译 马 超 校)

0021. Marginal cardiac allografts do not have increased primary graft dysfunction in alternate list transplantation

Lima B. / Rajagopal K. / Petersen R. P. et al. [Dr. C. A. Milano, DUMC Box 3043, Department of Surgery, Duke University Medical Center, Durham, NC 27710, United States] – CIRCULATION 2006, 114/SUPPL. 1 (I27 – I32)

BACKGROUND - Clinical success with modern heart transplantation(HT) has led to the development of an al-

ternate list(AL) HT strategy, matching marginal cardiac allografts with recipients who do not meet standard criteria for HT. Marginal allografts may be at an increased risk for primary graft dysfunction(PGD), the leading cause of early mortality after HT. The incidence of PGD in AL HT relative to standard list(SL) HT has not been evaluated, and may contribute to the greater mortality associated with AL HT. The objective of this study was to determine the incidence of and predictors for PGD. METHODS AND RESULTS - A retrospective analysis was performed on 260 consecutive adult patients undergoing either SL HT(n = 207) or AL HT(n = 53) at our institution from 1/2000 to 1/2005. PGD was defined by requirement for mechanical circulatory support immediately post-HT or more broadly as the need for either mechanical support or high-dose inotrope(epinephrine ≥0.07 μg/kg/min). Donor hearts allocated to AL recipients were turned down for SL HT for reasons that included coronary disease, left ventricular dysfunction or hypertrophy, and high-dose inotropic requirement. AL HT recipients were significantly older, with a higher proportion of diabetes mellitus and ischemic cardiomyopathy. Both groups experienced a similar incidence of significant rejection, but overall mortality was higher in the AL HT group. The incidence of PGD did not differ between AL and SL HT recipients. Pre-transplant VAD and prolonged total ischemic times(≥4.5 hours) were independent predictors of PGD. CONCLUSION - Select marginal donor hearts used in AL HT do not have an increased incidence of PGD. Pre-transplant VAD and prolonged ischemic times are more important determinants of PGD. These data support continued aggressive utilization of marginal donor hearts in AL HT.

在替代的心脏移植手术中边缘心脏移植并不增加原发性移植物功能障碍发生率

背景：现代心脏移植(HT)**的成功临床经验使替代心脏移植**(AL HT)**策略得以发展，它是指不符合标准心脏移植条件的边缘心脏移植。边缘心脏移植可能会增加原发性移植物功能障碍**(PGD)**的发生率，**PGD**是心脏移植术后早期死亡的首要原因。但** AL HT **的** PGD **发生率与标准心脏移植**(SL HT)**是否有区别尚未被评价过，偏向于认为** AL HT **有较高的病死率。本研究旨在探讨** PGD **的发生率以及预测因素。方法和结果 纳入本中心** 2000 **年** 1 **月至** 2005 **年** 1 **月共** 260 **例** HT **成人患者，其中** SL HT 207 **例，**AL HT 53 **例，对所有资料进行回顾性分析。**PGD **的定义是** HT **后需立即行机械循环支持，或者更广义地指需要机械支持或大剂量的变力性药物[肾上腺素≥**0.07 μg/(kg·min)**]。配给** AL **受者的供体心脏不符合** SL HT **的原因有：冠心病、左室功能障碍或肥厚以及需要大剂量应用变力性药物。**AL HT **受者其年龄明显较大，伴有糖尿病或缺血性心肌病的比例较高。两组患者的显著排斥反应事件发生率相似，但总死亡率在** AL HT **组较高，而** PGD **的发生率在两组间并无差异。移植前应用心室辅助装置**(VAD)**和缺血总时间延长**(≥4.5 h)**是发生** PGD **的独立预测因素。结论 选择边缘供体心脏用于** AL HT **并不增加** PGD **的发生率，移植前** VAD **和缺血总时间延长是发生** PGD **的更重要决定因素。以上结果支持今后将边缘供体心脏继续用于** AL HT**。**

0022. Low-dose dobutamine tissue-tagged magnetic resonance imaging with 3-dimensional strain analysis allows assessment of myocardial viability in patients with ischemic cardiomyopathy

Bree D. / Wollmuth J. R. / Cupps B. P. et al. [Dr. M. K. Pasque, Division of Cardiovascular Surgery, Queeny Tower, Barnes-Jewish Hospital, One Barnes-Jewish Hospital Plaza, St. Louis, MO 63110, United States] –
CIRCULATION 2006, 114/SUPPL. 1(I33 – I36)

BACKGROUND - Tissue-tagged magnetic resonance imaging(MRI) with 3-dimensional(3D) myocardial strain analysis allows quantitative assessment of myocardial contractility. We assessed the hypothesis that 3D strain determination at rest and with low-dose dobutamine would discriminate between viable and nonviable myocardium in patients with ischemic cardiomyopathy(ICM). METHODS AND RESULTS - MRI with radiofrequency tissue-tagging at rest and with low-dose dobutamine was performed in 16 normal volunteers and 14 patients with ICM. Three-dimensional global and regional circumferential strains(Ecc) were computed for all subjects at rest and with dobutamine. Results were compared with clinically indicated conventional viability studies. Compared with normal volunteers, global left ventricular Ecc was significantly decreased in patients with ICM at rest(−0.15 ± 0.06 versus −0.27 ± 0.03; $P < 0.001$) and with dobutamine (−0.17 ± 0.08 versus −0.37 ± 0.10; $P < 0.001$). Ecc was significantly decreased in nonviable regions compared with viable segments at rest(−0.08 ± 0.06 versus −0.17 ± 0.10; $P < 0.001$) and with dobutamine (−0.07 ± 0.06 versus −0.21 ± 0.11; $P < 0.001$). Ecc in viable segments increased significantly in response