0122. Omega－3 fatty acids in cardiac biopsies from heart transplantation patients: Correlation with erythrocytes and response to supplementation

Harris W. S. / Sands S. A. / Windsor S. L. et al. [Dr. W. S. Harris, Lipid and Diabetes Research Center, 4320 Wornall Rd, Kansas City, MO 64111, United States] – CIRCULATION 2004, 110/12 (1645－1649)

Background-Omega-3 fatty acids(FAs) appear to reduce the risk of sudden death from myocardial infarction. This reduction is believed to occur via the incorporation of eicosapentaenoic acid(EPA) and docosahexaenoic acid (DHA) into the myocardium itself, altering the dynamics of sodium and calcium channel function. The extent of incorporation has not been determined in humans. Methods and Results-We first determined the correlation between red blood cell (RBC) and cardiac omega-3 FA levels in 20 heart transplant recipients. We then examined the effects of 6 months of omega-3 FA supplementation (1 g/d) on the FA composition of human cardiac and buccal tissue, RBCs, and plasma lipids in 25 other patients. Cardiac and RBC EPA + DHA levels were highly correlated (r = 0.82, P < 0.001). Supplementation increased EPA + DHA levels in cardiac tissue by 110%, in RBCs by 101%, in plasma by 139%, and in cheek cells by 73% (P < 0.005 versus baseline for all; responses among tissues were not significantly different). Conclusions-Although any of the tissues examined could serve as a surrogate for cardiac omega-3 FA content, RBCEPA + DHA was highly correlated with cardiac EPA + DHA; the RBC omega-3 response to supplementation was similar to that of the heart; RBCs are easily collected and analyzed; and they have a less variable FA composition than plasma. Therefore, RBC EPA + DHA (also called the Omega-3 Index) may be the preferred surrogate for cardiac omega-3 FA status.

心脏移植患者心肌活检标本中ω－3脂肪酸与红细胞的相关性及对补充的反应

背景：ω－3脂肪酸(FA)似乎可降低心肌梗死猝死的风险。该作用可能是通过二十碳戊烯酸(EPA)与二十二碳己烯酸(DHA)协同进入心肌细胞，并改变其钠、钙通道动力学而实现的。但在人体中，这种协同作用的程度尚未得到确认。方法和结果：首先探讨20例心脏移植患者红细胞与心肌细胞中ω－3 FA水平的相关性，然后探讨其余25例患者补充ω－3 FA(g/d)6个月对心肌组织、颊部组织、红细胞中FA组分及血脂的影响。心脏与红细胞EPA + DHA水平高度相关(r = 0.82, P < 0.001)。补充后心肌组织EPA + DHA水平增加了110%，红细胞中增加了101%，血浆中增加139%，颊细胞中增加73%(与所有基线值相比P < 0.005，各组织之间相比无显著性差异)。结论：尽管上述任何一种组织均可作为测定心脏ω－3 FA含量的替代物，但红细胞EPA + DHA与心肌EPA + DHA高度相关；红细胞对ω－3 FA补充的反应与心脏相似；红细胞较易采集和分析，且较血浆变化少。因而红细胞EPA + DHA(也称为ω－3指数)可能更适合作为心肌ω－3 FA水平的一个替代指标。

0123. Single-arm study of bridging therapy with low-molecular-weight heparin for patients at risk of arterial embolism who require temporary interruption of warfarin

Kovacs M. J. / Kearon C. / Rodger M. et al. Dr. M. J. Kovacs, London Health Sciences Centre, 800 Commissioners Rd E, London, Ont. N6A 4G5, Canada] – CIRCULATION 2004, 110/12 (1658－1663)

Background-When warfarin is interrupted for surgery, low-molecular-weight heparin is often used as bridging therapy. However, this practice has never been evaluated in a large prospective study. This study was designed to assess the efficacy and safety of bridging therapy with low-molecular-weight heparin initiated out of hospital. Methods and Results-This was a prospective, multicenter, single-arm cohort study of patients at high risk of arterial embolism (prosthetic valves and atrial fibrillation with a major risk factor). Warfarin was held for 5 days preoperatively. Low-molecular-weight heparin was given 3 days preoperatively and at least 4 days postoperatively. Patients were followed up for 3 months for thromboembolism and bleeding. Eleven Canadian tertiary care academic centers participated; 224 patients were enrolled. Eight patients (3.6%; 95% CI, 1.8 to 6.9) had an episode of thromboembolism, of which 2(0.9%; 95% CI, 0.2 to 3.2) were judged to be due to cardioembolism. Of these 8 episodes of thromboembolism, 6 occurred in patients who had warfarin deferred or withdrawn because of bleeding. There were 15 episodes of major bleeding(6.7%; 95% CI, 4.1 to 10.8): 8 occurred intraoperatively or early postoperatively before low-molecular-weight heparin was restarted, 5 occurred in the first postoperative week after low-molecular-weight heparin was restarted, and 2 occurred well after low-molecular-weight heparin was stopped. There