《心脏病学——心血管内科学教科书》姊妹篇

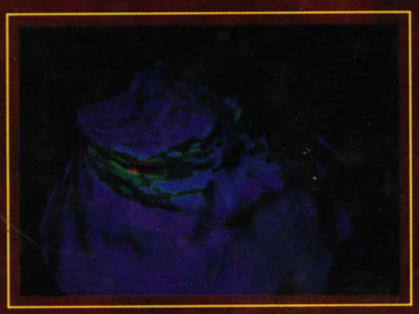
急性冠状动脉综合征

Acute Coronary Syndromes

原　著　Pierre Théroux

主　译　胡大一

副主译　贾三庆　王吉云　华琦　李宏伟



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序

20 世纪初期，冠状动脉疾病是一种严重病变，但是对于它的两种最重要的表现形式——心绞痛与急性心肌梗死的区分并不清楚；到了 20 世纪 20 年代，人们通过确认慢性心绞痛与心肌梗死的临床与病理改变将二者区分开来；30 年代，提出了心绞痛持续时间长可能导致急性心肌梗死的概念；40 年代，界于心绞痛与急性心肌梗死之间的“中间综合征”被认为非常常见；70 年代初期，提出了不稳定心绞痛的概念，现在我们认为它包括初发严重性心绞痛、恶化性心绞痛以及静息心绞痛。在不稳定型心绞痛的定义变得更清晰时，更多的关注直接转移到如何区别 ST 段抬高与非 ST 段抬高的心肌梗死上来，显然，很多不稳定心绞痛可发展成非 ST 段抬高的心肌梗死(NSTEMI)。

到了 20 世纪 90 年代，随着检测心肌坏死更敏感的生化标记物的出现，不稳定型心绞痛与 NSTEMI 之间的界限重新变得不清晰。确实，诊断为不稳定型心绞痛的患者实际存在 NSTEMI 的百分比在不断增加，这一点已经比较明确，即从病理生理学与临床的观点看上述两种状态应当放在一起考虑，合称为“非 ST 段抬高的急性冠状动脉综合征”，有时简称为“ACS”。

ACS 的研究在很多前沿方面有爆炸性进展，这部由 Pierre Théroux 编写的书给我留下了深刻的印象，权威性的捕捉了其中重要的发现，详细探讨了本领域诸如流行病学、临床、病理生理学以及治疗学上的进展。ACS 以其发病率高、病情重，成为心脏病学的“心脏”。确实，每一个心脏病学医师——不管是介入治疗还是非介入治疗、门诊诊疗还是住院诊治，抑或是分别在高血压、心力衰竭、心脏病预防或康复治疗方面有特长的心脏病学医师，都会遇到 ACS 患者。

对 ACS 患者施治的心脏病学医师，将会受惠于 Pierre Théroux 博士与其天才的合作者奉献的这部重要的新书，我的合编者 Douglas Zipes 和 Peter Libby 与我乐于将此书作为《心脏病学——心血管内科学教科书》的姊妹篇。

Eugene Braunwald

波士顿，马萨诸塞州

前　言

急性冠状动脉事件的处置：超越冠心病监护病房(CCU)的范畴

一个世纪的临床观察和最近数十年获取的大量新知识，将急性冠状动脉综合征（acute coronary synndroms，ACS）推到了心脏病临床与科研的最前沿，由于ACS发病率增高且预后差，心脏病专家必须增进自身的知识，改进治疗方法以逆转ACS的动态恶化，防止病变进展到不可逆的心肌损害。

近年来CCU的实践模式发生了根本变化，比如从1990年到2000年，蒙特利尔心脏研究院CCU收住的患者从800名翻倍到1600名，1990年40%的患者为急性心肌梗死，45%为非ST段抬高的ACS，到2000年则分别为20%和75%。随着老龄化社会的来临，患者的平均年龄从61岁增加到64岁，住院天数由8.5天降至4.5天，令人鼓舞的是死亡率从6%降至3%。

CCU的传统作用也发生了转变，这反映在新的治疗目的、富有挑战性的概念以及处置策略上。比如针对ST段抬高的心肌梗死，人们不断寻找更好更快速的血管重建技术和心肌细胞保护方法；对于非ST段抬高的ACS，稳定斑块是关注的焦点；心脏病二级预防，强调危险因素的控制成为所有患者治疗的通则。在新的治疗策略下不断达到这些目标。

炎症与免疫异常是导致斑块活动的主要因素，其作用超过动脉粥样硬化与血管内血栓的影响。血液与血管壁细胞、细胞因子、生长因子以及基质金属蛋白酶之间存在复杂的相互作用，构成了动脉粥样硬化的生物学基础(图1)，随着认识的进一步加深，未知的作用环节与激发因素将必然被揭示，由此产生治疗学上的突破。因此，新的诊断与治疗方法将应运而生。

心脏病学革命性变化与不断扩大的医疗卫生消费必然要求现代的CCU要超越其传统的范围，应当改变原有的目的和概念，今天的CCU需包括：CCU前处置，及时进行心导管检查和血管重建；CCU后处置。所有救治层面上均需要多学科协调一致，共同制

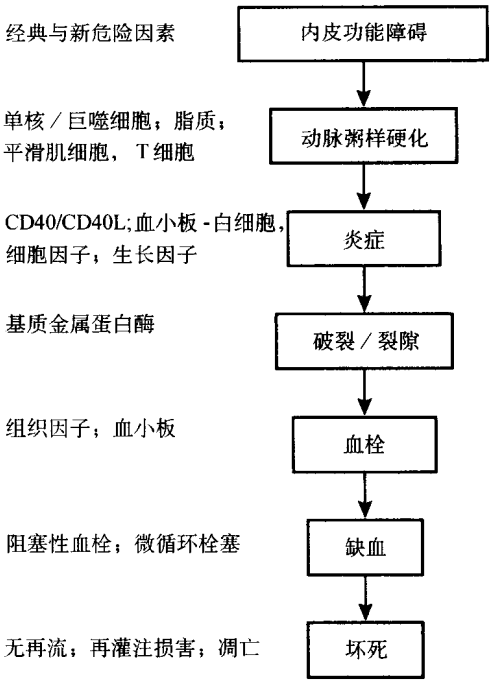

图1　ACS源于一系列病理生理事件并最终导致细胞死亡。一旦斑块严重阻塞冠状动脉或形成血栓造成心肌缺血，则产生瀑布效应样事件，临床表现在此过程中出现的相对较迟，在各个水平上，有机会对其进行干预。当前的治疗指南主要针对有症状的阶段进行治疗，图中显示了瀑布效应的诊断流程和治疗靶目标

订诊断、治疗方案(图2)。

所有相关的专业人员，从负责心脏患者的内科医师到院前急诊救护和急诊室的全体工作人员，都要参与患者进入CCU前的处置。ACS的诊断与危险分层应一体化完成，需要12导联心电图、肌钙蛋白以及心脏负荷试验的证据，很多医院设置胸痛与转送单元完成上述工作。由于救治的时间与生存率相关联，因此需要急诊介入治疗。当前努力要做到的是将诊治手段到达患者(图3)。

CCU内的医务人员要在控制花费的条件下完成

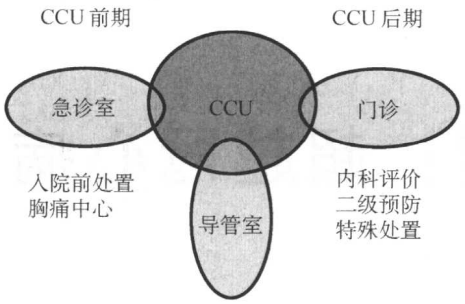

图 2 ACS 的治疗已经延伸出 CCU 前期与 CCU 后期。CCU 前期、CCU 期与 CCU 后期各类参与者共同努力，制订诊断治疗方案，以达到最有效的处置

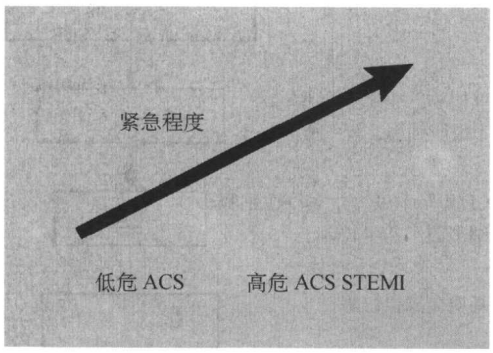

图 3 ACS 救治时间的重要性。箭头梯度反映救治的差异。对于 ST 段抬高的心肌梗死(STEMI)，时间越短，心肌坏死越少；对于非 ST 段抬高的 ACS，治疗的重点是尽快抑制血栓形成，减弱血栓形成的激发点以及对病变严重的患者进行血运重建

大量工作，筛选出高危患者，进行包括血管重建在内的诊治流程，以获得最佳治疗和效益。当今时代，治疗手段含有很多诱人的现代技术，但不能忽视患者的积极参与。除了急性期缓解病情外，治疗的目的还需有效的控制病变的进程，防止动脉粥样硬化进展，改进患者的生活质量。

CCU 的平均住院时间逐渐减少，仅比门诊心导管介入留观时间略长，因此，后-CCU 阶段应运而生。此阶段用于重新评价治疗的成败、药物剂量是否适当以及对药物的耐受性，这些措施能防止患者出院后过多且不必要的急诊室就诊次数。由于患者增加住院时间获得的医学常识依然有限，因此出院后应当强调控制危险因素的再教育，应根据患者的特殊需要与存在的特殊问题安排合适的治疗(图 4)。

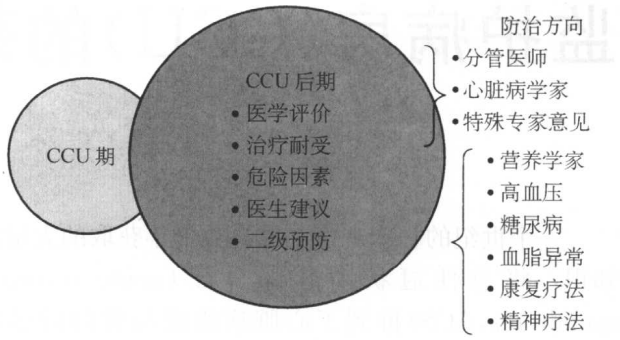

图 4 CCU 后期。控制危险因素对于 ACS 的治疗是必要的，为此要进行积极的教育和实施个人预防方案。ACS 患者动脉粥样硬化不稳定，复发的危险性高，非 ST 段抬高的 ACS 与危险因素未控制的患者长期危险性特别高。心脏病专家与患者的分管医师应实施个体防治方案，必要时与各类专家和特殊门诊合作

与以往相比，现代从事心脏监护工作的医生，有更多机会接触前沿的知识理念，并可以通过国内、国际的临床试验为医疗事业的发展做出贡献。

本书根据以上新概念来安排结构，有些章节涉及 ACS 在个人和社区人群中的发病情况，对于 ACS 的病理生理学、诊断、危险分层、药理学以及血运重建治疗策略则做了详尽的侧重，尚有一些专题是本书所特有的。从不同视角阐述的临床实践反映了 CCU 前期、CCU 期和 CCU 后期多学科参与的治疗方法。本书结尾在回到欧洲心脏病学会(ESC)、美国心脏病学会(ACC)和美国心脏协会(AHA)有关 ACS 最新处置指南具体实践前，展望了 ACS 防治的前景和新一代 CCU 的构想。

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Coronary Angiography and the Culprit Lesion in Acute Coronary Syndromes

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Cocaine and Other Environmental Causes of Acute Coronary Syndromes

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Coronary Angiography and the Culprit Lesion in Acute Coronary Syndromes

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Markers of a Thrombotic State

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What Cardiologists Need to Know on Depression in Acute Coronary Syndromes

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Coronary Angiography and the Culprit Lesion in Acute Coronary Syndromes

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Molecular Mechanisms of the Acute Coronary Syndromes: The Roles of Inflammation and Immunity

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Perspective for Gene and Cell-Based Therapy

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An Epidemiological Perspective: Society, Environment, Risk Factors, and Genetics

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The Elderly, Women, and Patients with Diabetes Mellitus

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Risk Stratification in Non-ST-Segment Elevation Acute Coronary Syndromes

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The Role of Infection

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Triggers to Acute Coronary Syndromes

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The Elderly, Women, and Patients with Diabetes Mellitus; The Modern Coronary Care Unit

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Computed Tomography in Coronary Artery Disease

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β-Blockers and Calcium Channel Blockers: Use in Acute Coronary Syndromes

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Triggers to Acute Coronary Syndromes

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Acute Coronary Syndromes: National and International Dimensions of the Problem

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Emerging Diagnostic Procedures for the Vulnerable Plaque

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Echocardiography in Acute Coronary Syndromes

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The Patient with Disabling Angina Not Amenable to Revascularization Procedures

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Preface; Continuous Risk Stratification; Antithrombotic Management

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Acute Plaque Passivation and Endothelial Therapy

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ATP-Sensitive Potassium Channels, Adenosine, and Preconditioning

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Identifying the Vulnerable Plaque and the Vulnerable Patient

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Revascularization in Acute Coronary Syndromes: Which Patients and When?

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Treatment of Cardiovascular Inflammation

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An Epidemiological Perspective: Society, Environment, Risk Factors, and Genetics

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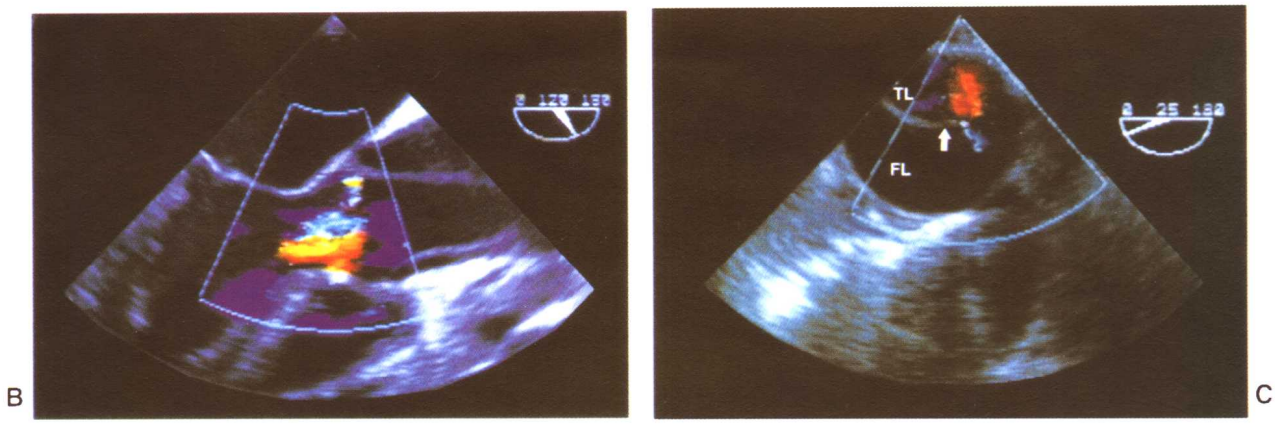

图 18-5 A，经食管超声心动图清楚的显示升主动脉。B，彩色血流 Doppler 显示联合主动脉瓣反流。C，胸降主动脉短轴观显示真腔(TL)和假腔(FL)之间的沟通，二者由内膜瓣(箭头)分割

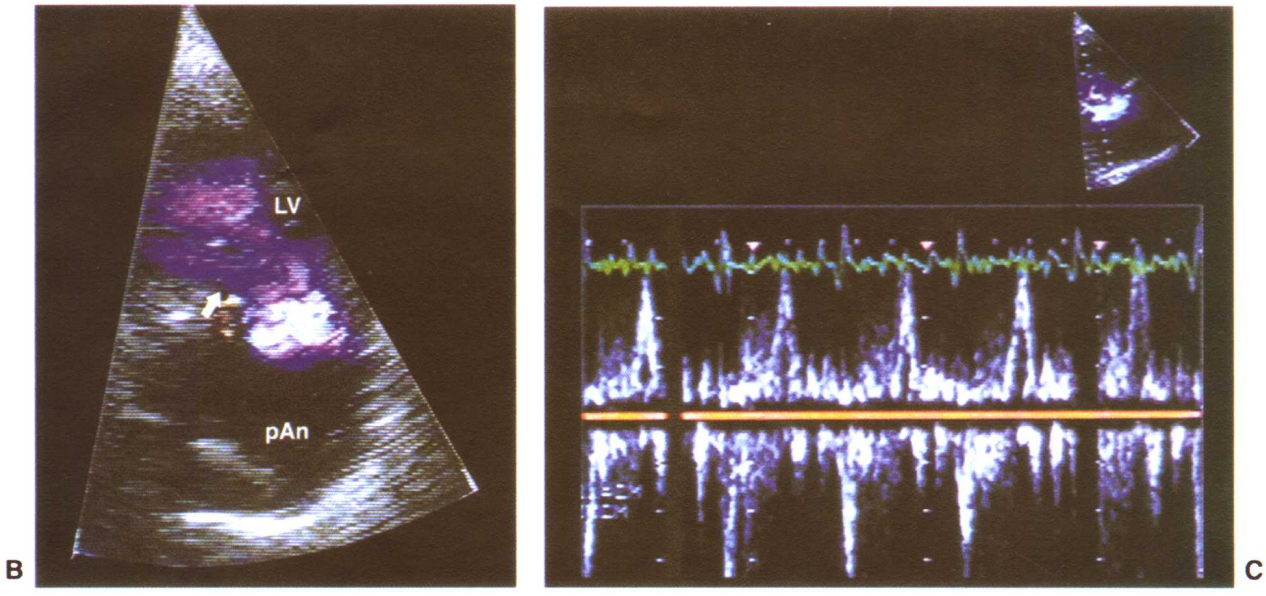

图 18-7 A，胸骨旁长轴观(右侧可见左室心尖部)显示：下侧壁基底段的连续性中断导致了左室与看似假性动脉瘤的大的腔隙之间相交通。箭头所指为左室下侧壁基底段。B，通过彩色 Doppler，可见血流从左心室流向假性动脉瘤的瘤腔(箭头)。C，脉冲频谱 Doppler 显示血流来往的速率。AO，主动脉；LA，左心房；MV，二尖瓣；pAn，假性动脉瘤

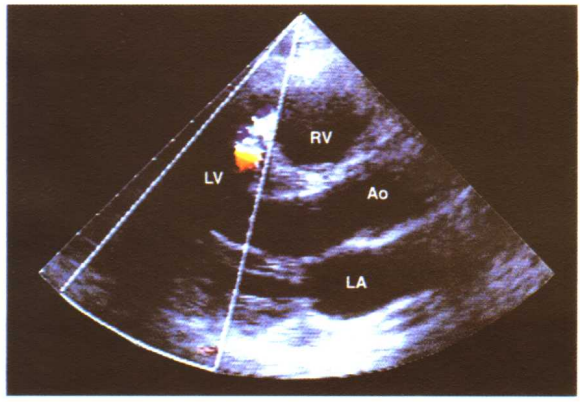

图 18-8 在急性前壁心肌梗死后 3 天，观察到患者临床恶化和出现新的收缩期杂音。彩色 Doppler 胸骨旁长轴观可见到由左到右心室的分流，与室间隔缺损表现一致。AO，主动脉；LA，左心房；LV，左心室；RV，右心室

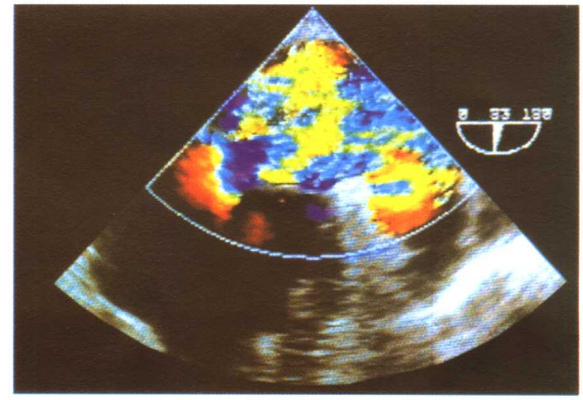

图 18-9 A. 经食管超声心动图两腔观(83 度)显示急性心肌梗死后继发于乳头肌断裂的严重二尖瓣反流

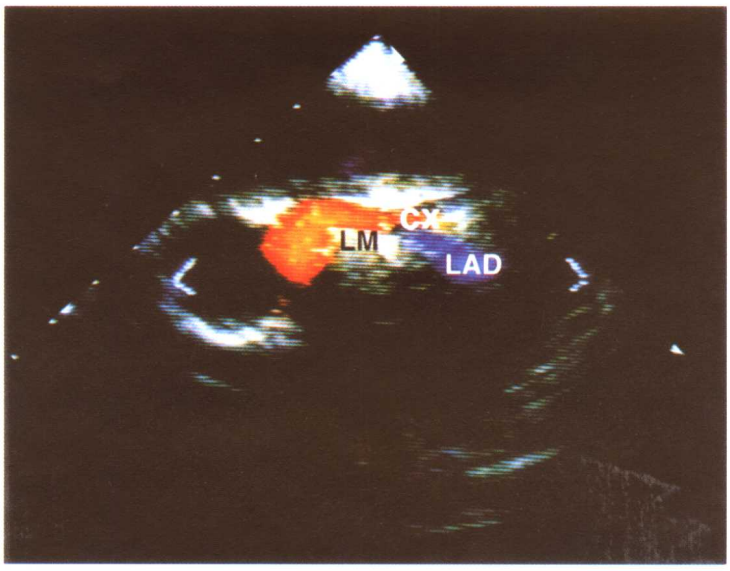

图 18-12　经食管超声心动图显示冠状动脉左主干(LM)及其分支、左前降支(LAD)和回旋支(CX)的血流

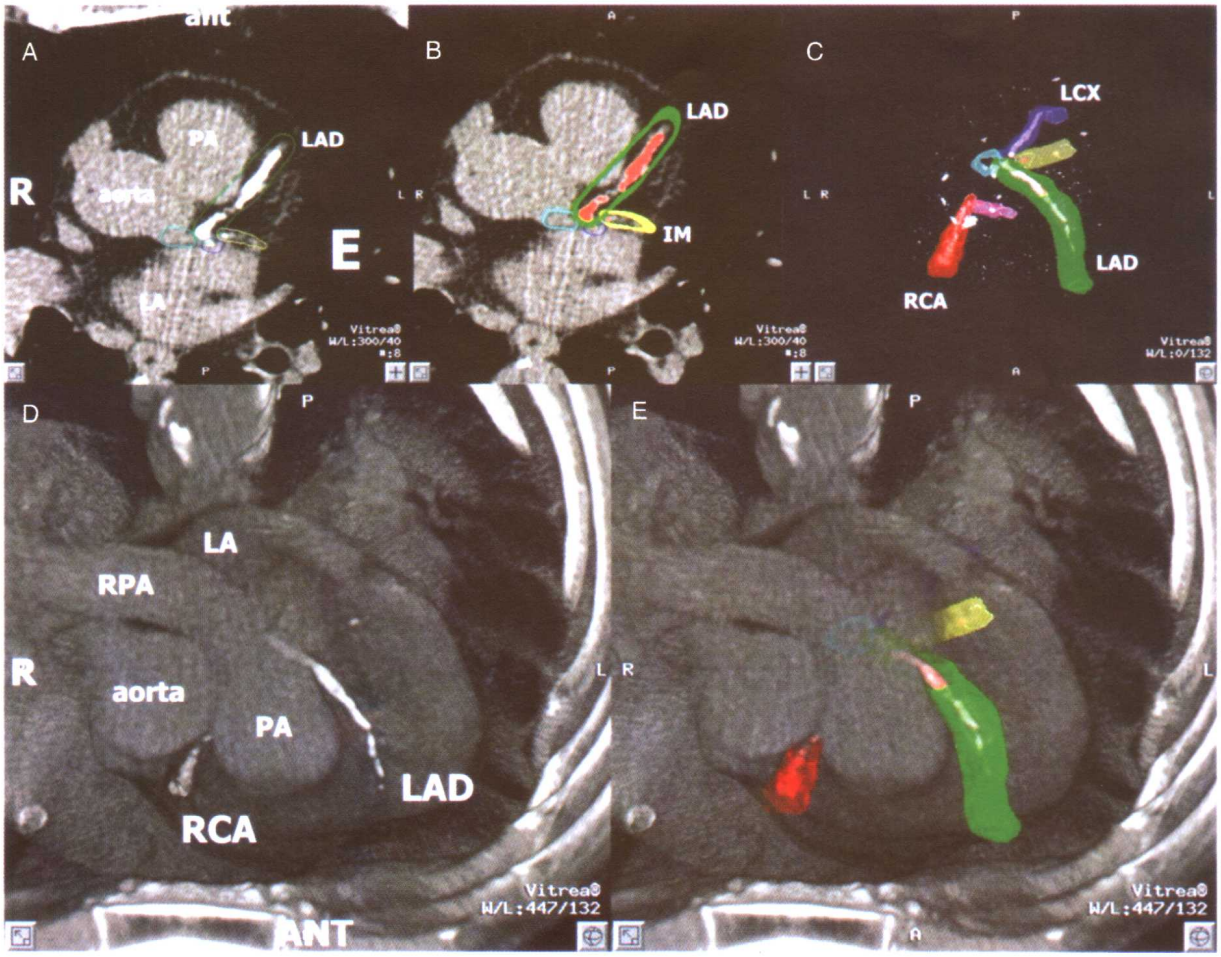

图 20-3　定性及定量检测冠状动脉钙化的实例。A，人为将冠状动脉沿纵轴切面标记出轮廓，以便于有选择地定量分析冠状动脉钙化沉积。B 和 C，将节段性容量密度值超过阈值(130HU)范围内的轮廓连接并区分其容量成分，作出标记(红色)。D 和 E，非增强的 3-维 MSCT 扫描的容量成分示例，显示左前降支动脉(LAD)和右冠状动脉(RCA)钙化。主要分支的代表性积分(Agatston 和容量积分)分别为：左主干 0，0；LAD988，629；左回旋支(LCX)673，445；RCA1629，1061；后降支动脉 40，35。这位患者的总积分为 3330 或 2170，结合患者年龄，考虑钙化积分升高。ANT，前；LA，左心房；PA，肺动脉；R，右；RPA，右肺动脉

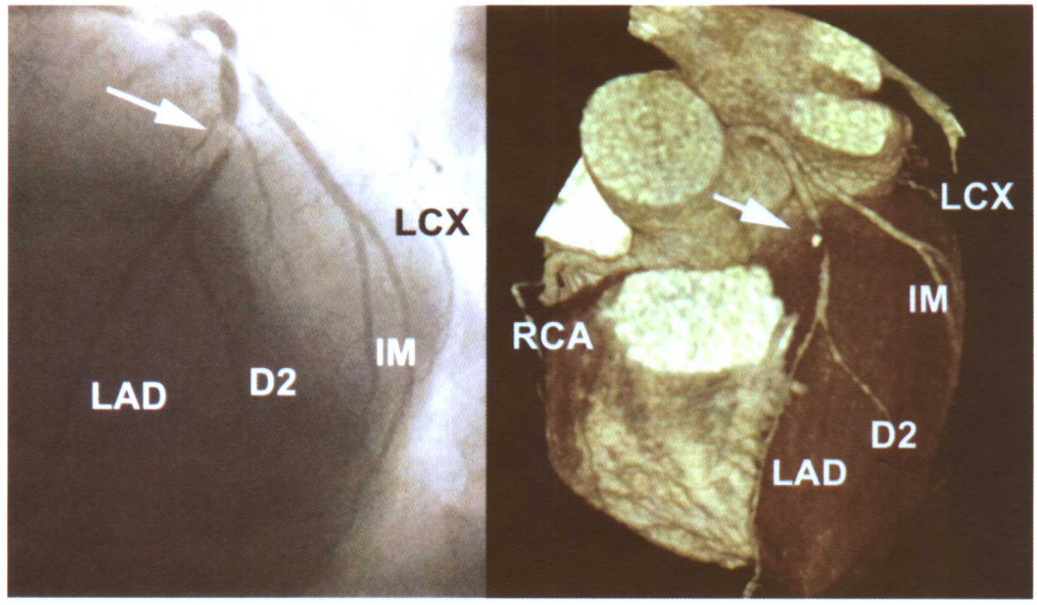

图 20-6 前降支中段，第一对角支远端冠状动脉狭窄的患者传统 CT 和 MSCT 冠状动脉显像(三维容量显像)。MSCT 中可以看见斑块的钙化成分。左回旋支(LCX)，右冠状动脉(RCA)，第二对角支(D2)，中间支(IM)也能看见

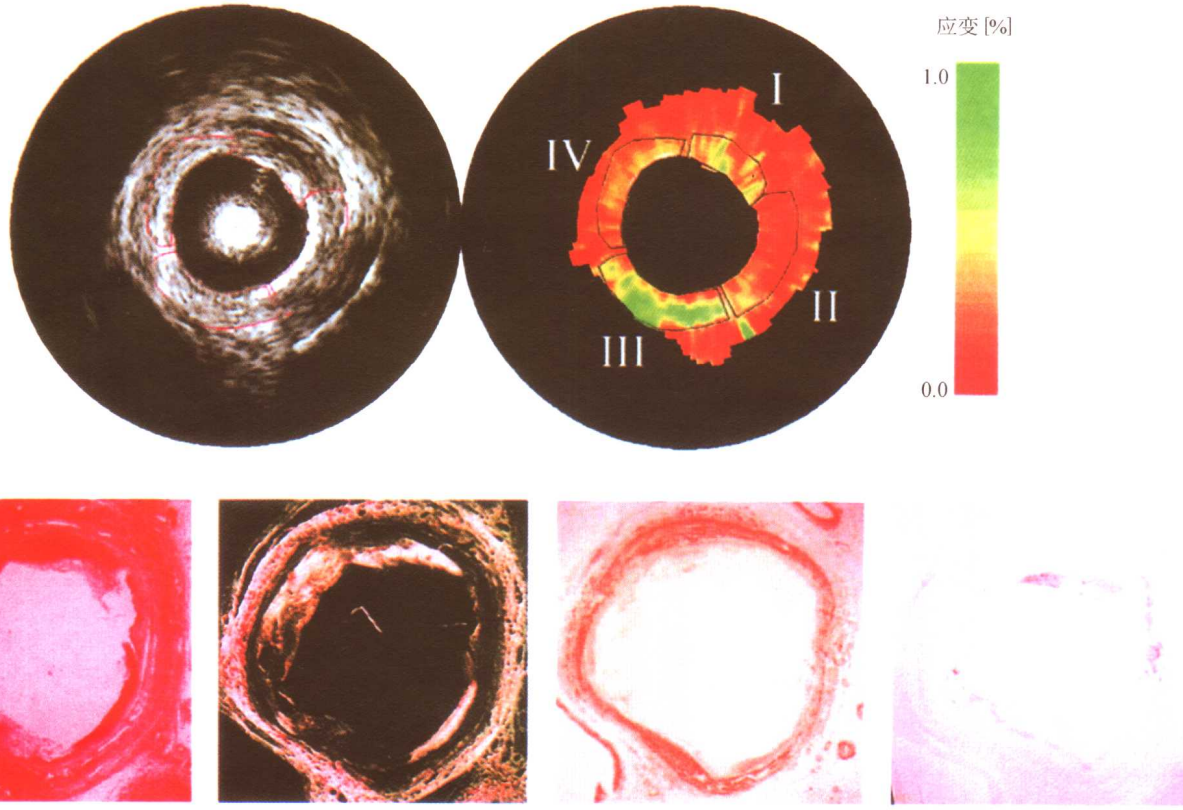

图 47-2 血管内超声(IVUS)(左上)，IVUS 弹性图像(右上)，和相应的人股动脉粥样硬化的组织学影像(底图)。IVUS 弹性图像通过获得两种管腔内压(80 和 100mm Hg)下 IVUS 图像并进行射频测定的局部应力水平的相关分析而完成。红色代表低局部应力；黄色代表中度局部应力；绿色代表高局部应力。标准 IVUS 图像以变化的回波发生性显露出向心性斑块。IVUS 显示出两处深染区和两处柔和区。组织学切片(从左向右)picro-Sirius 红染胶原，picro-Sirius 红配合偏振光显微镜显示脂肪组织、平滑肌细胞的抗-α-肌动蛋白抗体和巨噬细胞的抗 CD-68 抗体。结合组织学图像，柔和区实际上包括巨噬细胞密度增高的脂肪物质，而深染区则由纤维成分构成

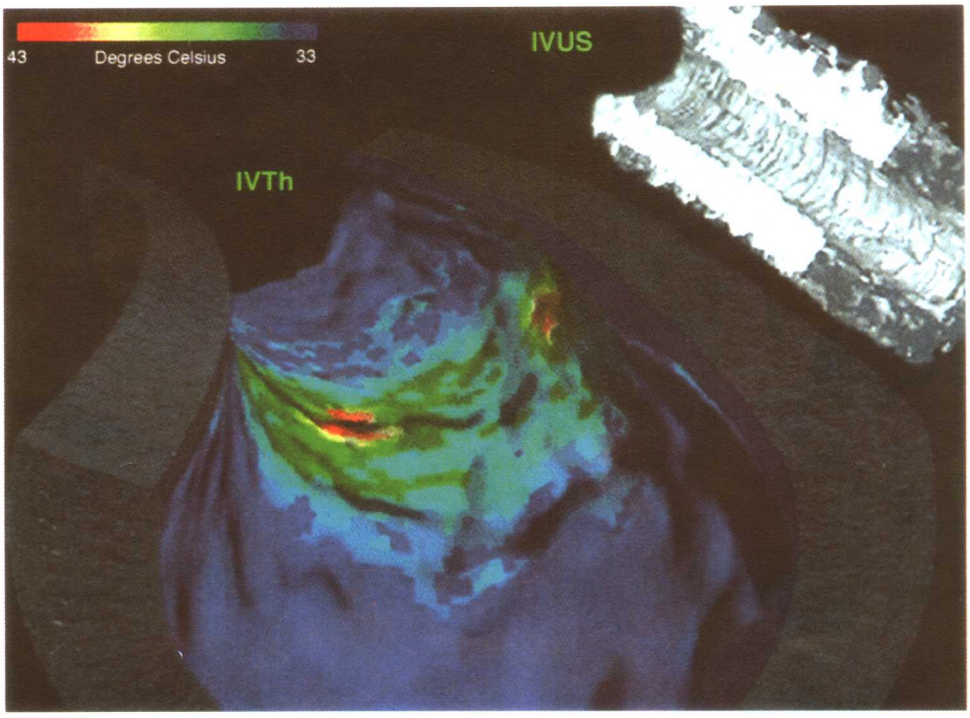

图 47-3 兔主动脉的导管下温度记录的三维重建(左)和血管内超声(右)。颜色标尺(左上)的范围在红(43℃)和深蓝(33℃)之间。三维血管内超声显现出重要的细微结构，而温度记录法测定温差，充分发挥两种成像方法的互补特性，因而可以对炎症部位进行定位

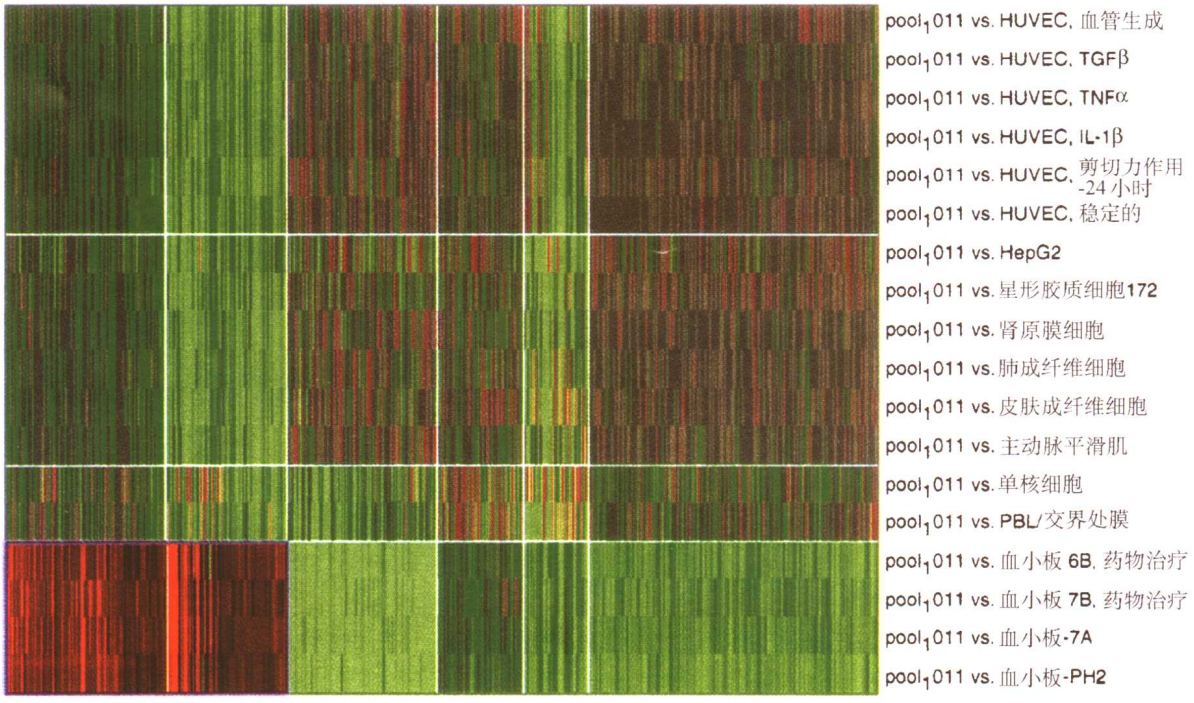

图 47-9 显示 1 700 个血小板选择性 mRNAs 的血小板转录子的基因芯片。HUVEC：人脐静脉内皮细胞

目　录