·临床病例讨论·
Clinicopathological Conference

An elderly patient with recurrent heart failure after coronary artery bypass grafting surgery

(the 37th case)

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Case presentation

A male patient, 73 years old, was admitted to Chinese PLA general Hospital on June 15, 2009, complaining of paroxysmal chest distress for 22 years, which was getting worse with recurrent shortness of breath in recent 1 month.

The patient had a 22-year history of intermittent chest distress, and the electrocardiogram (ECG) showed abnormal ST-T, so he had diagnosis of coronary artery disease (CAD). Though he got regular treatment for CAD, he still suffered from acute inferior and posterior myocardial infarction in 1991. Since then, his exercise tolerance decreased with progressively worsening dyspnea on exertion. Angiography in 2001 showed that: he had a severe stenosis (about 90%) in proximal left anterior descending, a 90% stenosis in proximal left circumflex, and complete occlusion in right coronary artery. So he received coronary artery bypass grafting (CABG) in 2002. At the beginning of 2009, he felt slight palpitation and dyspnea during hard work, fast walk, or climbing stairs. His legs were swollen in the evening. ECG displayed frequent ventricular premature beats (Fig 1). After taking β-blocker or amiodarone, the patient presented degree Ⅱ artial ventricular block(Fig 2). The echocardiography showed enlarged left ventricle and atrium, inferior wall ventricular aneurysm, and left ventrical ejection fraction (LVEF) of 45%. Since May 2009, his shortness of breath aggravated at night, forcing him to "sit upright". He was unable to complete activities that he could do easily one month ago. He was hospitalized with the diagnosis of cardiac dysfunction. He complained of cough with white sputum, but without blood. There was a 30-year history of hypertension. His blood pressure was controlled at normal range currently by medications. He also had a 15-year history of gout, which was stable, but without history of diabetes, hyperlipemia, and drug allergy.

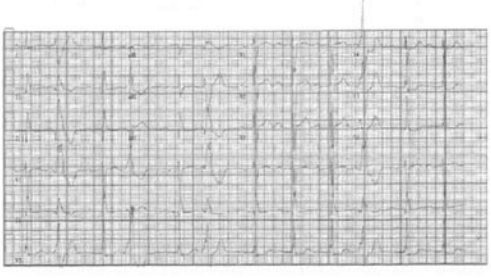

Fig 1 ECG on admission

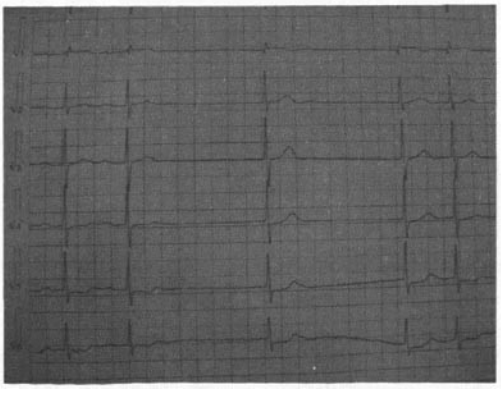

Fig 2 ECG after β-blocker or amiodarone medical treatment
degree Ⅱ atrial ventricular block after β-blocker or amiodarone medical treatment

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Family history The patient was born in Xi'an in 1936. He had been to the south of China but did not contact contaminated water. He gave up smoking for 7 years. He got married in 1959. His wife was healthy. They had a son who was also healthy. Both his father and mother had history of hypertension.

Physical examination Body temperature was 36.3℃, pulse rate 87 beats/min, respiratory rate 24/min, blood pressure 130/70mmHg. He was mentally normal and cooperative in the examination. There was no eruption, no jaundice, no purpura on the skin, and the lymph nodes were not palpable. The head, eyes, nose, ears, mouth were normal while the lips were cyanotic. The neck was soft, and there was mild venous engorgement. Thyroid glands were not palpable. There was no thrill or brunt. The trachea was in midline. The chest and respiratory movements were symmetrical. There was no abnormal dullness but some moist rales were heard in the base areas of the both lungs. The points of maximal impulse were not visible but palpable in the 6th costal interspace, and there was no thrill. The cardiac dullness was enlarged to the left side. The distance from midsternal line to midclavicular line was 10cm. The heart rate was 87 beats/min, with 5 to 8 premature beats in one minute. There was a grade Ⅱ soft blowing like systolic murmur at the apex, and P2 was louder than A2, but no pericardium friction sound was heard. Abdominal wall was soft without tenderness. The liver was palpable 2cm below the costal margin with slight tenderness. The spleen was not palpable and there was no shifting dullness. There was mild edema on both lower extremities.

Accessory examination The complete blood count and levels of electrolytes and glucose, as well as the results of tests of coagulation, renal function, liver function and myocardial enzymes were all within the normal ranges. But brain natriuretic peptide(BNP) level was as high as 1488ng/L. Chest X-ray showed an enlarged heart with pulmonary congestion(Fig 3).

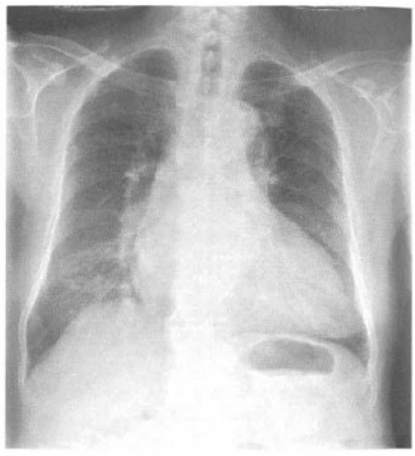

Fig 3 Chest X-ray examination

ECG showed sinus rhythm, fragmented QRS complexes (0.13s), old inferior and posterior myocardial infarction, and frequent ventricular premature beats(Fig 1).

Degree Ⅱ atrial ventricular block after β-blocker or amiodarone medical treatment.

Echocardiogram showed LVEF of 37%-44%, old inferior and posterior myocardial infarction, enlarged left ventricle and atrium, mild pulmonary artery hypertension, and mild aortic valves regurgitation (Fig 4). Contraction coordinated between left and right ventricle, but left intraventricular contraction was dyssynchronized.

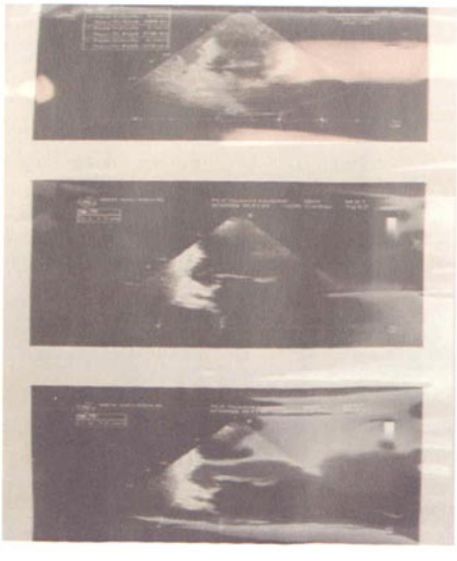

Fig 4 Echocardiography examination

Holter revealed 18 600 multiple ventricular premature beats (VPB) of total 91 133 heart beats within 24h. Sinus rythm and degree Ⅱ A-V block were also shown. The minimal heart rate was 32 beats/min, and the maximal was 95beats/min. Coronary CT angiography showed that the bridge vessels were sclerotic but not obstructed, and the coronary artery was diffuse sclerotic(Fig 5).

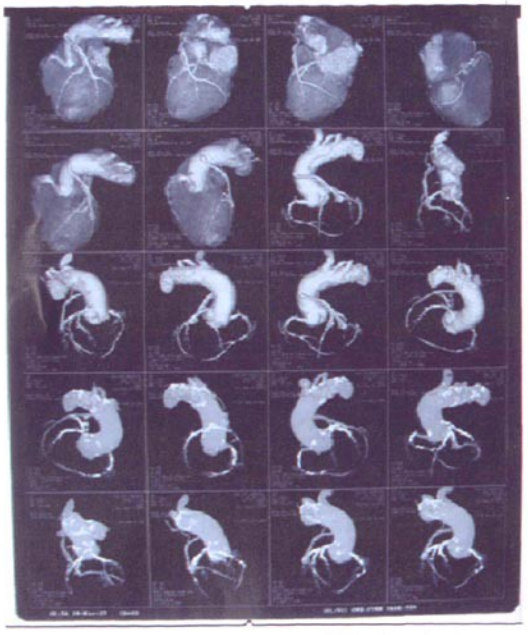

Fig 5 Computed tomography angiography of coronary artery
The bridge vessels were sclerotic but not obstructed

Admission diagnosis (1) Coronary heart disease. Old myocardial infarction with degree Ⅳ heart failure, post CABG. (2) Arrythmia. Multiventricular premature beats. (3) Degree Ⅱ A-V block. (4) Hypertension grade 3 with very high risk. (5) Gout.

Discussion

Dr. CHEN Weiren: This is an old male patient with coronary artery disease, old myocardial infarction, post CABG, NYHA Class Ⅳ heart failure and frequent ventricular premature beats (VPB). The diagnosis was definitely clear. After admission, he was treated with diuretic, digoxin, β-block, ACEI, vasodilators and amiodarone, etc. The symptoms of dyspnea and edema in his lower extremities were improved by regular and standard medical treatment, but his heart function didn't turn better. He suffered repeated acute heart failure, and the LVEF was still around 35% with frequent VPB. Because his bridge vessels were still open, I don't think he should accept revascularization therapy. As the patient's lowest HR was only 32 bpm after taking amiodarone and β-block for VPB treatment, meanwhile bradycardia with degree II A-V block appeared on his ECG, which obviously limited us to increase the dosage of medications to eliminate VPB, therefore medications got restricted. I think his prognosis is poor.

Dr. CAO Jian: The diagnosis is definite, including coronary artery disease, old myocardial infarction, post CABG, ischemic cardiomyopathy, degree Ⅱ A-V block, left ventricular aneurysm, NYHA class Ⅳ heart failure and frequent VPB. His ECG showed the QRS complex was 0. 139s, with transient degree Ⅱ A-V block. There was no obvious aggravation of ischemic change on his ECG during attack of acute heart failure. So we didn't consider of re-PCI for him.

As we could not use the medications in expected doses for his bradycardia and A-V block, I think he had strong indication for cardiac resynchronization therapy (CRT), and CRT -defibrillator (CRT-D) was better for him. CRT-D is recommended to reduce morbidity and mortality in patients in NYHA Ⅲ-Ⅳ class who are symptomatic despite optimal medical therapy, and have a reduced LVEF (35%) and QRS prolongation (120ms). CRT-D implantation could prevent sudden cardiac death. and is commonly preferred in clinical practice in patients satisfying CRT criteria.

Dr. ZHANG Li: I agreed with above analysis. We had no difficulties in diagnosis, but the key problem was that, he repeatedly suffered from acute heart failure despite optimal medical therapy, which may be due to the excessive preload of the heart. There was no more obvious evidence of cardiac ischemia than before during the episode of acute heart failure. So I don't think he needs re-PCI. UCG showed enlarged left atrium and ventricle, LVEDV > 200ml, LVESV > 120ml, LVEF only 37%. Wide QRS, obstinate VPB and elevated

BNP were all highly predictive of poor prognosis, high risk of sudden death, and low long-term survival rate. Though contraction coordinated between left and right ventricles, left intraventricular contraction was dyssynchronized. I also think CRT is suitable for him. CRT is used in order to synchronize interventricular and intraventricular contraction in patients with heart failure in whom there is evidence of electrical dyssynchrony (QRS width $\geqslant$ 120ms).

BNP levels are powerful markers of increased cardiovascular risk. CRT reduces NT-proBNP substantially, and reduction in NT-proBNP is associated with a better outcome. Patients with marked elevation of NT-proBNP receive a relative smaller benefit from CRT, but, due to their higher risk, the absolute benefit is similar.

The first clinical trial investigating the value of CRT in the management of patients with NYHA class Ⅲ and Ⅳ heart failure, a reduced LVEF, and a wide QRS demonstrated that CRT improves functional class, exercise duration, and quality of life. Results from a blind study that enrolled 1 000 NYHA class Ⅲ/Ⅳ heart failure patients with a wide QRS show that CRT dramatically improves patients' perceived quality of life and the clinicians' assessment of functional status. All studies indicate that CRT improves peak value of O_2, and should be regarded as an objective measurement of exercise and functional capacity.

A meta-analysis of CRT showed that CRT could reduce all-cause mortality in heart failure patients. Cardiac resynchronization composite response was effective in about 70% heart failure patients, and in about 30% patients, it was uneffective or even worsened heart function. We should let the patient know the risks of CRT and its efficiency rates.

Dr. YANG Tingshu: This is a 73 years old man, who has an enlarged heart, progressively worsened heart function with repeated acute attack, which strongly predicted that he had very high risk for sudden death. I agree with all of your opinions. He should have CRT-D implantation as soon as possible. I just want to mention something about CRT. The main purpose is to illustrate how the leads are placed to achieve cardiac resynchronization.

1. Achievement of cardiac resynchronization - biventricular pacing

CRT pacemaker has 3 leads: right atrium lead, right ventricle lead and coronary vein of left ventricle (LV) or epicardial lead. Biventricular pacing restores synchrony between left and right ventricles.

Standard pacing leads are placed in the right atrium and right ventricle. The LV lead is placed via the coronary sinus in a cardiac vein, preferably a lateral or postero-lateral vein in the mid-part of the LV. New LV leads can be used in varying patients. A key difference is the placement of a LV lead via the coronary sinus opening. Coronary venous anatomy varies significantly between patients. In a small percentage of cases, it may not be possible to place the LV lead transvenously. Some centers are opting for an epicardial approach if the transvenous approach is unsuccessful.

2. Benefits of CRT on cardiac function

CRT improves systolic function by relieving systolic asynchrony, increases diastolic filling, enables LV reverse remodeling by reducing LV end systolic and diastolic volumes, reduces mitral regurgitation

3. Role of echocardiography in CRT

Echocardiography plays important roles in CRT, such as assessment of cardiac function before and after CRT, investigation of the mechanism of benefit by CRT——cardiac synchronicity, prediction of therapy responses, patient selection. Before CRT, echocardiography can select responders and predict optimal lead placement; during CRT, it can determine optimal lead placement; after CRT, it can optimize A-V delay and V-V delay, and evaluate short/long-term effect.

In a word, the CRT-D was beneficial for improving life quality, exercise tolerence, functional capacity, and LV structure remodeling. Besides, CRT can reduce all-cause mortality and incidence of sudden death. So we strongly recommend CRT-D implantation for this patient, though 30% of patients may get no benefit or worse.

(Translator: ZHANG Li)

老年男性冠状动脉搭桥术后难治性心力衰竭1例

1 病历摘要

患者文某，男性，73岁，主因发作性胸闷气短22年，冠脉搭桥术后7年，加重并下肢浮肿不能平卧1个月余，于2009年6月15日入院。自1987年始，患者在劳累时感胸闷、气短，心电图检查发现ST-T异常，当地诊断为冠心病，虽然长期服用扩冠等药物治疗，仍于1991年经历了急性下壁正后壁心肌梗死。之后其活动耐力明显减低并常感劳累后气短，2001年6月冠脉造影提示三支病变：前降支近端90%狭窄，回旋支近端90%狭窄，右冠中段完全闭塞。于2002年7月行冠状动脉搭桥术，将大隐静脉与后降支、钝缘支和对角支搭桥，乳内动脉和前降支搭桥。之后病情一度平稳，直到2009年初，患者上述症状逐渐加重，伴双下肢浮肿，心电图发现“频发多源室性早搏”(图1)，服用乙胺碘呋酮治疗后早搏虽有减少，但出现心动过缓及Ⅱ度Ⅰ型房室传导阻滞(图2)，并于来我院前1个月内连续两次发生夜间憋醒，端坐呼吸，咯大量白色泡沫痰，下肢浮肿加重，整夜不能平卧，稍微活动心悸气短明显加重，当地超声心动图检查提示：陈旧下壁、后壁心肌梗死，下壁室壁瘤形成，左心明显扩大，射血分数45%。为进一步诊治来解放军总医院。患有高血压病30余年，最高190/90mmHg，长期服用复方降压片降压治疗，目前服用代文、络活喜、安体舒通等药物治疗，血压一直波动在110～130/60～70mmHg。痛风病史15年，间断有右足趾关节肿痛发作。已戒烟7年，无饮酒嗜好，无糖尿病及高脂血症病史。其父母均有高血压病史。

查体：体温36.3℃，脉搏87次/min，呼吸20次/min，血压130/70mmHg。神志清楚，半卧位，口唇轻度紫绀，颈静脉轻度充盈，双肺底可闻及中等量湿性啰音，无哮鸣音。心界向左扩大，心率87次/min，律齐，1min可闻及早搏5～8次，各瓣膜区未闻及病理性杂音。腹平坦，肝肋下2cm，质中等无压痛。无移动性浊音，双下肢胫前及足背轻度凹陷性浮肿。

辅助检查：血常规、电解质、肝肾功能、心肌酶、血糖血脂、凝血功能均正常，但脑钠肽(BNP)水平高达1448ng/L。心电图提示：陈旧性下壁、正后壁心肌梗死；房室传导延迟、房内传导阻滞、室内传导阻滞、QRS波宽0.13s；频发室性期前收缩(图1)。胸片报告心影增大，轻度肺淤血(图3)。心脏超声符合陈旧性左室下壁心肌梗死后改变及慢性左室侧壁心肌缺血性改变，节段性室壁运动减弱，梗死区疤痕形成，左房左室扩大伴射血分数减低，射血分数37%，左室69mm，左房47mm，有室壁瘤。左室乳头肌功能不全，升主动脉扩张伴主动脉瓣轻中度返流(图4)。左右心室同步性检测：主动脉瓣和肺动脉瓣血流频谱起始时间差为8.25ms(正常＜40ms)。左室内同步检测：12个阶段任2个阶段收缩达峰时间的最大差值为126.67ms(下壁基底段-后间隔中间段)(正常＜100ms)，12节段收缩达峰时间标准差44.71ms(正常＜32.6ms)。印象：左右心室收缩基本同步，左室下壁瘢痕形成，左室内收缩不同步。动态心电图报告为窦性心律，房内传导阻滞，房室传导延迟，不全性房室传导阻滞(Ⅱ度Ⅰ型)，频发多源室性早搏(18600次/d，成对330次/d)，有室性融合波，部分呈现二联律、三联律，部分形成短阵室性心动过速，偶发房性早搏。最慢心率32次/min，最快心率95次/min；无长间歇。冠状动脉CT成像结果：桥血管通畅，吻合口未见明显狭窄，远端血管显影尚可；原冠状动脉弥漫性硬化改变(图5)。

入院诊断：(1)冠心病。陈旧性下壁、正后壁心肌梗死，冠脉搭桥术后；慢性心功能不全，心功能Ⅳ级。(2)频发多源室性期前收缩。(3)Ⅲ度Ⅰ型房室传导阻滞。(4)高血压病3级，极高危。(5)痛风。

入院后内科药物治疗主要包括(1)拮抗神经内分泌激活药物：①ACEI类；②β受体阻滞剂(出现Ⅱ度Ⅰ型房室传导阻滞后停药)；③醛固酮拮抗剂；④新活素。(2)强心药物；(3)利尿剂；(4)乙胺碘呋酮抗心律失常治疗(出现Ⅱ度Ⅰ型房室传导阻滞后停药)。

2 讨 论

陈伟任医师：该患者为老年男性，冠心病，陈旧性下壁、正后壁心肌梗死；冠脉搭桥术后；慢性心功能不全，心功能Ⅳ级；频发多源室性期前收缩诊断明确。虽经治疗后自觉症状如夜间喘憋、下肢浮肿等有所好转，但患者夜间仍不能平卧，反复发生急性左心衰，超声心动图显示心功能无明显改善，射血分数35%左右，仍有频发室性早搏出现，目前桥血管通畅，没有再血管化指征。由于患者服用乙胺碘呋酮后心率偏慢，最慢心率达32次/min，并出现Ⅱ度Ⅰ

型房室传导阻滞，小剂量β受体阻滞剂、稳心颗粒等对控制室性早搏无效，不能加大剂量使用β受体阻滞剂、或乙胺碘呋酮等药物抗心律失常治疗，因此内科用药明显受限，患者预后差。

曹剑副主任医师：诊断明确，冠心病，陈旧性下壁、正后壁心肌梗死；冠脉搭桥术后；缺血性心肌病，Ⅱ度Ⅰ型房室传导阻滞；室壁瘤；慢性心功能不全，心功能Ⅳ级；频发多源室性期前收缩；心电图示 QRS 139ms 。出现过一过性Ⅱ度Ⅰ型房室传导阻滞，心衰发作时心电图较前没有明显缺血加重的表现，故心衰发作的病因不能单纯用心肌缺血来解释，考虑可能与容量负荷过重、心室内收缩不同步有关，故目前暂不考虑再介入治疗。由于β受体阻滞剂加量后出现明显心动过缓伴Ⅱ度Ⅰ型房室传导阻滞，内科用药受限。有采用心脏再同化治疗(cardiac resynchronizating therapy，CRT)的适应证，CRT-D 比较合适。CRT-D 对心功能Ⅲ～Ⅳ级、射血分数＜35%、QRS 宽度＞120ms、尽管应用了理想的足量的药物治疗仍有症状的患者可以减少发病率和死亡率，CRT-D 能够预防猝死，通常适用于临床上符合 CRT 指征的患者。

张丽主任医师：同意上述分析。诊断明确，关键问题是尽管在药物治疗方面尽了最大的努力，但患者仍连续几次发生急性左心衰，可能与容量负荷过重有关。发作时心肌缺血并无明显加重，故心衰的病因的确不能用急性心肌缺血来解释，目前暂不考虑冠脉介入治疗。超声心动图示左房和左室均扩大，左室舒张末期容量负荷＞200ml、收缩末容量＞120ml、射血分数仅 37%；心电图示 QRS 增宽、顽固的难以根治的室性早搏，以及血 BNP 明显升高均提示患者预后差、猝死率高、长期生存率低。虽然同步化检测提示双心室尚同步，而左室内收缩不同步，但仍有采用 CRT 的指征。CRT 用于那些有心衰的患者，这些患者均有心电活动不同步的证据(QRS 增宽，＞0.12s)，CRT 的目的是为了使双心室或室内收缩同步。

BNP 是心血管危险性增加的强力预测因子，CRT 可以从根本上减少 NT-proBNP 水平，而 NT-proBNP 的减少可以使预后更佳，NT-proBNP 显著升高的患者从 CRT 获益较小，但相对于其高危险性，绝对获益是相近的。

有关 CRT 的第一个临床试验是在心功能Ⅲ/Ⅳ级、左室射血分数减低和宽 QRS 波的患者中进行的，结果显示，CRT 可以改善心功能等级、运动耐力和生活质量。一项随机双盲研究入选 1000 例心功能Ⅲ/Ⅳ级伴宽 QRS 波的患者，结果显示，CRT 显著改善患者的生活质量及心功能等级。所有研究均显示，与对照组相比 CRT 可提高组织中的峰值氧流量，可以作为改善运动和功能的更好的客观治疗方法。

来自于 CRT 的汇萃分析显示，两个大型 CRT 试验对心功能Ⅲ/Ⅳ级同时有心室收缩不同步的患者的疗效评价显示，CRT 可减少患者的全因死亡率。CRT 显示，约 70% 的心衰患者 CRT 治疗有效，而 30% 的心衰患者安装 CRT 后并没有明显心功能改善甚至使心功能恶化，我们应和患者谈 CRT 手术的风险和有效率。

杨庭树主任医师：这是一例 73 岁老年男性患者，大心脏，反复心衰，有猝死高危存在。同意大家的意见，为防止猝死，该患者应尽早考虑安装 CRT-D。在这里介绍一下 CRT 的方法和步骤。主要目的让临床医生了解电极是怎样放置来获取心脏再同步化的。

(1)获得心脏再同步——双心室起搏：CRT 起搏器有 3 个电极。右房电极、右室电极和左室的冠状静脉或心外膜电极。双心室起搏可恢复左右心室的同步性。标准的起搏电极被放置在右房和右室。左室电极通过心脏静脉进入冠状窦而放置。最好选左室中部的侧壁静脉或侧后壁静脉，新型的左室电极可适用于不同的患者。关键点在于要通过冠状窦开口放置左室电极，不同患者的冠状静脉解剖变异很大。有少部分病例无法经静脉放置左室电极，如果经静脉方法失败，有些医学中心会选择心外膜放置左室电极的方法。

(2)CRT 对心功能的益处：由于改变了收缩期的不协调，收缩功能将得以改善。增加舒张期心室充盈。由于左室收缩末和舒张末容积减小而使左室重塑。减少二尖瓣反流。

(3)心脏超声在 CRT 中的应用：在 CRT 前后评价心脏功能。研究 CRT 获益机制，即心脏同步化。预测疗效。CRT 前，选择适合 CRT 的患者，预测最佳的电极位置。CRT 中，选择最理想的电极位置。CRT 后，房室传导延迟最佳化、室内传导延迟最佳化、评价近期和远期疗效。

鉴于 CRT 能明显改善生活质量、心脏功能和运动耐力，明显减少全因死亡和猝死发生率，且本病例具有 CRT-D 强适应证，所以应在药物治疗的基础上选择 CRT-D 治疗。但 30% 的患者可能无效。

又及：患者于 2009 年 8 月 26 日在局麻下顺利安装了 CRT-D，术者为王玉堂教授、耿仁义教授及阜外医院华伟教授。术后伤口愈合好，无手术并发

症出现。术后恢复乙胺碘呋酮抗心律失常治疗、卡维地洛、硝酸酯类、ACEI、地高辛、间断利尿等药物治疗，未再出现心前区不适或夜间阵发性呼吸困难，可平卧，双下肢浮肿消失。手术1个月后，复查超声心动图显示室内不同步状态较术前略有改善，各房室腔大小无明显变化，左室射血分数46%；24h动态心电图显示室性早搏明显减少至1552次。

（参加讨论医师：张 丽 杨庭树 陈伟任 曹 剑）

（张 丽 整理）

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用[2]，它对心肌有广泛的电生理作用，除明显延长心肌复极过程外，对窦房结AVN-HPS和房室旁路均有明显抑制作用；临床电生理研究提示胺碘酮可明显抑制AVN-HPS和房室旁路的双向传导[3]，故适用于室速及室上速，也适用于W-P-W伴室上速，房扑、房颤。本研究结果显示，静脉注射胺碘酮对WRT的总有效率为80%，室速总有效率为91%。笔者认为，无论是室性或室上性WRT，应用胺碘酮是比较理想的，尤其是抢救患者时，短时间内难以明确是室性或室上性或难以明确是否伴有W-P-W的宽QRS心动过速，故可作为首选药物应用。本研究结果显示，5例静脉注射胺碘酮无效，以室速为多，1例静脉注射维拉帕米，4例电转复成功，说明胺碘酮也有一定的局限性。胺碘酮也有导致肺间质纤维化、肝损害、甲状腺功能亢进或减退、心动过缓、尖端扭转性室速等不良反应，在应用胺碘酮过程中，监测其可能出现的不良反应，如胸部X光片，甲状腺功能五项，Q-T间期等检查，静脉输入时心电监护，监测心率，心律，血压，Q-T间期，注意药物剂量，输入速度（150mg快速静脉注射，之后以1mg/min持续6h，再改为0.5 mg/min维持，总量1000～2000 mg/d），一旦发现其不良反应，及时减量或停药，不良反应大多可逆转，说明应用胺碘酮患者总的耐受性好，安全性好。

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2 讨 论

左、右冠状动脉开口绝大多数分别位于主动脉左、右窦内，只是其开口的前后和高低位置略有不同[1,2]。冠状动脉畸形包括冠脉起源和分布异常、冠脉支数异常、冠状动静脉漏，发生率0.6%～1.6%[5]；最常见的为左前降支和左回旋支双开口畸形，右冠状动脉变异相对少见[1]。单冠脉畸形大约占所有冠脉畸形的3.3%，一般认为是在胚胎时期冠状动脉异常发育或未发育完全造成的，其原因不明确[4]，多在冠脉造影时偶然发现。本例冠状动脉造影诊断为先天性右冠状动脉缺如，明确为单支左冠状动脉。Yamanaka等[5]报道，大多数右冠状动脉先天性缺如患者可发生劳力型心绞痛，静息时可无症状，本患者具有劳力和自发型心绞痛。单支冠状动脉对心肌灌注有潜在影响，被认为临床上具有潜在危险性，多因年龄、应激、感染、创伤等因素诱发心肌缺血，临床表现类似心绞痛、心肌梗死、心力衰竭、恶性心律失常、猝死等临床和心电图表现，冠状动脉造影是惟一确诊手段[6]。本例患者因疑诊"劳力和自发型心绞痛，运动平板试验阳性"住院，冠状动脉造影结果结合临床，综合分析患者心绞痛症状系活动时心肌需氧量增加，引起相对性心肌供血不足和夜间冠脉痉挛所致，一般建议行外科手术矫治，但目前尚缺乏统一的成年人冠状动脉先天性变异治疗标准[7]，患者因经济原因暂拒绝行外科手术矫治而出院。本例患者既往无特殊病史及心脏血管造影检查史，此次造影发现右冠状动脉缺如，进一步明确了患者病情，同时提示心血管介入手术人员应提高对其病理解剖、病理生理和临床表现的认识，在介入手术时应对可能遇到的冠状动脉起源异常类型、发生几率有所了解，以避免漏诊、误诊。药物治疗改善内皮功能和缓解冠脉痉挛尤其重要。

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