Mr C

A 28 year old man presented to his doctor in 2003 with increasing shortness of breath and decreased effort tolerance.

He gave a history of occasional palpitations sometimes associated with dizziness.

Initially no formal diagnosis was made. While sitting in the doctors waiting room at a follow up visit he experienced sudden intense dyspnoea and collapsed. He was found to be pulseless.
Examination

Slightly obese young man.

Normotensive with clinical left ventricular enlargement, a grade 2/6 mitral regurgitant murmur and a paradoxically split 2\textsuperscript{nd} heart sound.

ECG

Sinus rhythm. Normal P-R, QRS axis 70\textdegree, LBBB (QRS 160m.secs.), poor R-waves in V5 and V6.
ECGs in the doctors office showed broad complex tachycardias of which 2 were monomorphioic as well as a short run of a non-sustained tachycardia.
Tachycardia 1
Tachycardia 2

P-waves not seen. Rate 214bpm (280m.secs).
QRS= -60°. QR pattern V1. Rs V4 V5 V6.
P-waves probably not seen. c.f. V1
Non-Sustained Tachycardia with some RBBB Configuration
# Cardiac Catheterisation

## Pressures

<table>
<thead>
<tr>
<th>Structure</th>
<th>Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>80/56</td>
</tr>
<tr>
<td>m86</td>
<td></td>
</tr>
<tr>
<td>LV</td>
<td>106/5-22</td>
</tr>
<tr>
<td>PA</td>
<td>37/16 m27</td>
</tr>
<tr>
<td>RA</td>
<td>a=13, c=9, v=9</td>
</tr>
</tbody>
</table>
No mitral regurgitation, coronary arteries normal.
Electrophysiological Study
H-V interval prolonged (106 m.secs)
Frequent Non-Sustained Tachycardia
Runs of Ventricular Tachycardia Induced

Not possible to induce bundle branch re-entrant ventricular tachycardia
**Therapy**

Carvedilol (Dilatrend) up to 25mg bd.
Spironolactone 25 mg
Ramapril 5 mg
Furosemide 40 mg
Amiodarone 200 mg (after loading)
Implantation of Bi-Ventricular Pacing and ICD
Course

Remained symptomatic.
Varying class II–III NYHA.
Constant frequent PVCs – mainly bigeminy

Received both appropriate and inappropriate shocks.
Records From ICD Interrogation

SVT
Mechanism of Low Percentage Pacing
Percentage of pacing low - underestimated on pacer print out.
Not successful except in Pacer Clinic.
Problems

1. Spurious shocks from SVT.

2. Progression of cardiac failure – too little time in bi-ventricular pacing (suppression by PVCs).

Possible Solution

Ablate SVT and PVCs.

? Heart transplant.
Findings

1. AVNRT could not be induced.
2. A-V nodal duality confirmed.
3. Ventricular ectopy is localised to the lateral LV.
RF Ablation to:

- Slow pathway (apex of Triangle of Koch).
- Lateral LV
Subsequent Course

Electrophysiological

No Further spurious shocks.
No further runs of tachycardia (SVT).
Virtually no ventricular ectopy.
Paces V 100%
Haemodynamic

Major improvement

NYHA class III-IV now I-II.

Able to virtually stop diuretics and oral nitrates.

ProBNP levels

<table>
<thead>
<tr>
<th>Date</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>02/08/04</td>
<td>3807 pg/ml</td>
</tr>
<tr>
<td>10/09/04</td>
<td>Ablation</td>
</tr>
<tr>
<td>26/01/05</td>
<td>1059 pg/ml</td>
</tr>
<tr>
<td>23/05/05</td>
<td>678.8 pg/ml</td>
</tr>
</tbody>
</table>

Amiodarone stopped. No further ventricular tachycardias
Conclusions

1. Bi-ventricular pacing is most effective when given 100% of the time.

2. RF ablation is an excellent alternative to anti-arrhythmic drugs in many cases – particularly because of lack of side effects (haemodynamic, proarrhythmic and general).

3. Patients with ICD implantation may benefit from RF ablation if excessive appropriate shocks or spurious shocks occur.

4. In heart failure patients maintenance or provision of ventricular synchrony most of the time is mandatory.

5. Consideration should be given to ablation of excess ventricular ectopy (>7,000/24 hours) in heart failure patients not fully responding to conventional treatment.
Often runs of couplets and triplets.