General:

- In a dual chamber system with atrial and ventricular pacing and ventricular sensing (DVI, DDD, DDI), it is possible that the atrial pacemaker spike will be sensed by the ventricular wire, misinterpreted as a ventricular depolarisation and thereby inhibit the ventricular pacemaker output. In the absence of AV conduction, this will lead to ventricular standstill.

- In atrial sensing systems, the ventricular spike can be similarly misinterpreted, leading to inhibition of the atrial spike; however, ventricular pacing persists, making this less serious.

- Of more concern is the system that allows such atrial sensing 'cross-talk' to trigger a ventricular pacemaker spike.

What to do:

The simplest approaches to eliminating cross-talk are to:

- reduce the sensitivity in the atrial or ventricular channel;
- reduce the power delivered to the ventricular or atrial pacing wire.

NB: Most temporary pacemakers employ a fixed ventricular blanking period and default to set parameters for ventricular safety pacing. As such, cross-talk is rarely if ever problematic as long as reasonable sensitivity and output is set.

- This is a potential problem in only VDD or DDD pacing.

- The simplest form is far-field atrial sensing of a ventricular pacing spike, which is interpreted as an endogenous atrial depolarisation, leading to another ventricular impulse.

- This is overcome by use of an atrial blanking period, during which the atrial channel will not sense any depolarisation.

Temporary pace generators have a preset atrial blanking period that should be sufficient to guard against this.

- A more difficult problem exists when there is retrograde conduction between the ventricle and atrium, through either the AV node or an accessory pathway.

- The conduction may be intermittent and so may not be appreciated when the pulse generator is first set. In addition, it is often only a premature ventricular contraction that is initially conducted back up into the atrium. This may be sensed in the atrial wire as an endogenous atrial depolarisation, which (after the AV delay) triggers another ventricular depolarisation. This 'endless loop' continues with a periodicity that is the sum of the programmed AV delay and the time taken for retrograde conduction.

- This problem is overcome by having an adjustable post ventricular (pacing spike) atrial refractory period, the PVARP. The atrial sensing channel must be refractory when the retrograde depolarisation arrives.

- The PVARP is set to a default value in all pacing generators. However, as the speed of conduction in the retrograde pathway is variable between individuals, it is not uncommon to need to adjust the PVARP. The disadvantage of setting a very long PVARP is that it limits the maximum rate of atrial tracking.

- There is a simpler, but in some circumstances less ideal, solution to pacemaker mediated tachycardia. The re-entrant pathway will be terminated if the mode is switched to VVI or DVI, but this may incur the penalty of losing AV synchrony.

tracking of atrial arrhythmias:

- In dual chamber modes with atrial tracking (DDD or VDD) the pacemaker emits a ventricular spike for every atrial impulse detected.

- In the absence of a protection mechanism, a tracked rapid atrial arrhythmia would rapidly lead to VF.

oversensing:

- Oversensing can cause failure to pace, as already described. In DDD, external electrical impulses can also be misinterpreted as atrial activity, causing pacemaker mediated tachycardia. If the electrical interference is likely to continue, it may be necessary to reduce the sensitivity of the pacemaker (i.e. increase the sensitivity threshold) or switch to an asynchronous mode.

General:

- 'Failure to capture' is when there is electrical output at the pacemaker wire tips (confirmed by visible pacing spikes on the ECG), but this does not cause a cardiac contraction, as shown by the absence of a mechanical cardiac impulse on the arterial pressure or pulse oximeter waveform.

What to do about it:

(i) Correction of any of the exacerbating causes
(ii) Reversing the polarity of both bipolar and unipolar lead systems may help.
(iii) In a bipolar lead system, the distal (negative) electrode usually develops fibrosis first. If this occurs, the proximal electrode may remain adequate to use as a unipolar electrode (now connected to the negative terminal), with a return electrode inserted into the subcutaneous tissues.

NB: If the threshold is progressively increasing and the patient is dependent on the pacemaker, it is wise to place an alternative means of stimulus delivery (such as a temporary transvenous wire) before capture is entirely lost.

- In essence the same mechanisms of failure to capture can cause failure to sense.

- True failure to sense must be distinguished from normal pacemaker function with inappropriate settings, such as over-long refractory periods.

- Other causes of inappropriate tachycardia

- Inappropriate atrial tachycardia

- Inappropriate ventricular tachycardia

- Ventricular fibrillation

Causes:

- Increase in the resistance at the wire / myocardium interface, most commonly due to fibrosis around the pacemaker lead.
- Myocardial ischaemia
- Electrolyte imbalance, particularly hyperkalaemia, acidosis and alkalosis;
- Following defibrillation;
- Medications, including flecainide, moricizine, propafenone, sotalol, and possibly beta blockers, lidocaine, procainamide, quinidine and verapamil.

General:

- 'Failure to pace' occurs when there is no electrical output at the pacing wire tips when the set pacing mode calls for such an output.

Causes:

- Failure to pace can be due to:
  - Lead malfunction or an unstable connection between the lead and the pulse generator
  - Insufficient power in the pulse generator (which should be apparent from the battery indicator)
  - Cross-talk inhibition
  - Oversensing. Any electrical potential across the sensing wires can be misinterpreted as endogenous depolarisation, with resulting inhibition of the pacing spike. Such potentials can be caused by electromagnetic interference (from electrocautery, or even mobile telephones), skeletal muscle activity (including fasciculations caused by suxamethonium), or intermittent contact between the pacing wires, which can generate small 'make and break' potentials

- (Apparent failure to pace) Detection of endogenous extrasystoles, which are of insufficient amplitude to register on the surface electrode, but which inhibit pacemaker output.

Approach:

(i) Make sure that the pacemaker is switched on and connected to the pacing lead(s)
(ii) Increase output to its maximum setting (20mA for atrial lead; 25mA for ventricular lead)
(iii) Switch to an asynchronous mode to prevent oversensing
(iv) Connect the pacemaker directly to the pacing lead as occasionally the connecting wires may be faulty
(v) Prepare for replacing batteries or pacemaker unit
(vi) Prepare for transcervical pacing
(vii) Prepare for CPR and administer positive chronotropic drugs such as atropine or adrenaline

troubleshooting temporary pacemakers [created by Paul Young 02/10/07]