IN THE NAME OF GOD

PACE MAKER AND ICDs

Dr. Sima Sayah
Local effects of cardiac electrical stimulation

- Cardiac pacing requires a local stimulus sufficient to depolarize local myocardium during diastole to initiate a self-propagating wave front of depolarization.
- A stimulus that successfully stimulates local myocardium is said to capture it.
According to Ohm’s law:

\[ R = \frac{\text{VOLTAGE } U \text{ (in volt)}}{\text{CURRENT } I \text{ (in ampere)}} \]

comprises:
- lead resistance
- tissue impedance

Note for electricians:
The pacing impedance is not purely resistive (the tissue impedance is capacitive) and should be indicated by a Z. In the clinical practice only the absolute value or magnitude of the pacing impedance is considered and since it is expressed in “OHM” according to Ohm’s law, most people simply call it “resistance”

<table>
<thead>
<tr>
<th>INSULATION DEFECT</th>
<th>NORMAL PACING IMPEDANCE</th>
<th>LEAD FRACTURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 250Ω</td>
<td>ca. 500Ω</td>
<td>&gt; 1000Ω</td>
</tr>
</tbody>
</table>
The stimulus strength for local capture by ATP is higher than that for bradicardia pacing because ATP pulses are delivered during the relative refractory period rather than during diastole.
Always keep in mind! The myocardial refractory period refers to stimulation. In contrast, the pacemaker refractory period refers to the sensing function of the device.

**VENTRICULAR MYOCARDIAL REFRACTORY PERIOD**

- **Ventricular action potential**
- **ECG**

- **Absolute refractory period**: No stimulus can activate the ventricle.
- **Relative refractory period**: Only a stronger than normal stimulus can activate the ventricle.
- **NORMAL**: A normal stimulus activates the ventricular myocardium.
VENTRICULAR DEPOLARIZATION BY PACING

* The depolarization caused by the pacemaker does not occur via the specialized His-Purkinje network and propagates slower through ordinary myocardium
* The QRS complex is therefore wide like a ventricular extrasystole (premature ventricular contraction)

ENDOCARDIAL STIMULATION FROM RIGHT VENTRICLE
ECG resembles LBBB (LBBB = left bundle branch block)

EPICARDIAL STIMULATION FROM LEFT VENTRICLE
ECG resembles RBBB (RBBB = right bundle branch block)
VOLTAGE AND PULSE DURATION MAY BE CHANGED BY AN EXTERNAL PROGRAMMER

CAPTURE WITH

2.5 V
1.8 V
1.2 V
1.05 V

0.3 ms
0.5 ms
1 ms
1.5 ms

spike
paced complex
Threshold for pacing and defibrilation

- A threshold stimulus is the minimum stimulus required to evoke a response. (to depolarize local myocardium and to initiate a propagated response)

- The term defibrillation threshold (DFT) is used as the minimum shock strength that defibrillates during a sequence of fibrillation episodes in which defibrillation test shocks of different strengths are delivered
The pacing threshold is always expressed in terms of both voltage and pulse duration. The pacing threshold can be determined in terms of the smallest output voltage that captures the heart while keeping the pulse duration constant.
Waveform duration is critical

- Typically, the voltage output for pacing is set to 1.5-2 times the threshold at pulse durations of 0.4 -0.5 ms.
SAFETY RATIO CONCEPT FOR CAPTURE

Threshold voltage: 2.5 V
Programmed voltage: 5 V

Safety Ratio = \( \frac{\text{programmed voltage}}{\text{threshold voltage}} = \frac{5V}{2.5V} = \frac{2}{1} \)
BIPOLAR vs UNIPOLAR PACING
SIZE OF THE STIMULUS ON ECG

UNIPOLAR PACING

PM can active
Current through conductor
Current through the body

large pacemaker stimulus on analog ECG recording

A. F. Pinnaeve
BIPOLAR vs UNIPOLAR PACING
SIZE OF THE STIMULUS ON ECG

BIPOLAR PACING

PM can not active

Small pacemaker stimulus on analog ECG recording

A. F. Pinnaeve
ACTIVE LEAD FIXATION

INSULATION

CONDUCTOR

SCREW HOUSING

SCREW-IN ELECTRODE
Voltage is a critical parameter for pacing or defibrillation because it determines the electrical field that interacts with the heart.

- $E = IR$
- $E$: voltage
- $I$: current
- $R$: resistance
EVOLUTION OF PACING THRESHOLD

- Direct contact between electrode and excitable myocardium
- Thicker layer between electrode and excitable myocardium
- Smaller layer between electrode and excitable myocardium

STEROID-ELUTING ELECTRODE

- Dexamethasone diffuses into the myocardium
- Monolithic controlled release device
- TINE
- Insulation
- Porous coating
- Electrode (cross section)
- Conductor
## THREE-LETTER PACEMAKER CODE (ICHD)

<table>
<thead>
<tr>
<th>POSITION</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
</tr>
</thead>
<tbody>
<tr>
<td>CATEGORY</td>
<td>CHAMBER(S) PACED</td>
<td>CHAMBER(S) SENSED</td>
<td>MODE OF RESPONSE</td>
</tr>
<tr>
<td>LETTERS</td>
<td>V = VENTRICLE A = ATRIUM S = SINGLE</td>
<td>V = VENTRICLE A = ATRIUM S = SINGLE O = NONE</td>
<td>T = TRIGGERED I = INHIBITED O = NONE</td>
</tr>
</tbody>
</table>

### EXAMPLES:

AAL = a pacemaker pacing and sensing in the atrium, being inhibited by spontaneous electrical activation of the atrium.

VVT = a pacemaker pacing and sensing in the ventricle and working in the triggered mode (each sensed ventricular event elicits a pacemaker stimulus).

* A. J. Stjernvall
OVERSENSING

Detection Level

7.5 mV

Sensitivity

2.5 mV

time
The voltage refers to the intracardiac electrogram and not the surface QRS complex. Sensitivity refers to a programmable parameter of the pacemaker. A sensitivity of 4 mV means that the pacemaker can only sense a signal equal to or greater than 4 mV. It will sense a signal of 5 mV but not a signal of 3 mV.
Various drugs and metabolic effects can alter pacing and defibrillation thresholds

- acidosis
- alkalosis
- Electrolyte abnormalities, especially hyperkalemia
- drugs: 1C(flecainide), amiodarone
Goals of pacing:

1. Restoration of rate responsiveness
2. Restoration of AV synchrony
AV synchrony

- In patients with CMP & HF, timing of RV and LV contraction and relaxation may differ sufficiently that optimal AV timing for one may not be ideal for the other.

- Patients with severe diastolic dysfunction may benefit most from AV synchrony because they depend on optimal preload.
Lack of AV synchrony as a result of exclusive ventricular pacing can result in hemodynamic impairment caused by VA conduction and atrial contraction against a closed AV valve.

- Sign’s and symptoms: malaise, lightheadedness, atypical chest discomfort

- (Pace maker syndrom)
VENTRICAL DEMAND PACING (VVI)

Note that in the VVI mode, a competitive rhythm is not possible. Moreover the lifetime of the battery is extended because the pacemaker is not pacing during long periods of time when it is on standby.

** TIMER ACTION **
- Output stimulus
- Upper level
- Start
- Stop
- Lower level
- Automatic interval
- Start
- Reset

** Pacemaker response to sensed QRS complex **

** ON-DEMAND or STANDBY PACING **
- Automatic interval
- Escape interval
- Pacemaker stimulus
- Sensed spontaneous QRS complexes
- Paced QRS complex
- Spontaneous QRS inhibits the output & resets the timer
- Reset

The electronic escape interval (starting at the time of intracardiac sensing) is equal to the automatic interval. The escape interval is measured on the surface ECG from the onset of the QRS complex because the time of intracardiac sensing cannot be determined accurately. Therefore the escape interval so measured will be slightly longer than the automatic interval because intracardiac sensing occurs later than the beginning of the surface QRS complex.
1 INTERMITTENT UNDERSENSING OF A DEMAND PACEMAKER

unsensed spontaneous QRS complex

correctly sensed QRS complex

unsensed spontaneous QRS complex

paced QRS complex

-paced QRS complex

automatic interval

escape interval

automatic interval

automatic interval

Automatic interval

time
UNDERSENSING OF VPC BY DEMAND PACEMAKER

unsensed ventricular premature complex

correctly sensed QRS complex

unsensed ventricular premature complex

paced QRS complex

paced QRS complex

automatic interval

escape interval

automatic interval

automatic interval

A. T. Spinnaeel
Magnet application on a demand pacemaker.

Without magnet:
- Spontaneous QRS complexes

Magnet positioned over the pacemaker. This closes the reed switch and converts the pacemaker to the magnet mode (asynchronous mode or VOO).

Stimulus

Wide paced complex

Magnet interval

A. F. Pinnaeve
RV APICAL PACING

The frontal plane axis is usually left superior. It may also be in the right superior quadrant, where it causes leads I, II & III to be negative and lead aVR to show the largest positive deflection.

A typical LBBB pattern in the left precordial leads may not be present and all leads show a QS pattern.

The left precordial leads may show a dominant R wave.
The mean frontal plane axis of the paced beat is directed to the right lower quadrant (right axis deviation). There is a characteristic tall R wave in lead V1 to at least V3 and often further into the left precordial leads.

**Intended LV pacing via coronary sinus and coronary vein**

**Unintended LV pacing:**
* Passage of lead into LV via patent foramen ovale (from right atrium to left atrium and LV)
* Via subclavian artery (across the aortic valve) mistaken for the subclavian vein

**A lead within the LV cavity (endocardial site) may cause thrombus formation, cerebral emboli and stroke.** The diagnosis of LV endocardial lead misplacement should be suspected if there is a tall R wave at least in leads V1 to V3 and sometimes further in the left-sided precordial leads. The definitive diagnosis requires echocardiography especially by the transesophageal method.
Compared to the VVI pacemaker the SENSITIVITY should be higher (i.e. a lower number in mV) because P waves have lower amplitudes than R waves.
**Tracking** is present when the ventricular paced rate follows the spontaneous atrial rate in a 1:1 way.

- **Spontaneous Atrial Rate (SAR in bpm)** = \( \frac{60,000}{\text{Spontaneous Atrial Interval (SAI in ms)}} \)
- **Ventricular paced Rate (VPR in bpm)** = \( \frac{60,000}{\text{Ventricular Paced Interval (VPI in ms)}} \)
THE TREADMILL STRESS TEST

At an atrial rate of 115 bpm and the same paced rate, he still loved his physician....

VPR (bpm)

ventricular paced rate
upper rate limit
lower rate limit

LRL
50
50
30
120
120
120
URL

spontaneous atrial rate
SAR (bpm)

paced tracking
2:1 block
at LRL

But...
at an atrial rate of 120 bpm and a paced rate of 60 bpm he felt himself very unhappy

A. F. Tonnesen
Endless loop tachycardia often occurs at the upper rate. In such a case as the programmed upper rate interval (URI) is longer than the total atrial refractory interval (TARP), the AV delay is extended to conform to the upper rate interval (URI).
THE PACEMAKER REFRACTORY PERIOD TRADITIONAL CONCEPT

VRP = pacemaker ventricular refractory period
p = after pacing
s = after sensing

pacemaker stimulus
paced QRS complex
sensed spontaneous QRS complex

pVRP is often equal to sVRP
FUNCTIONS OF THE PACEMAKER
VENTRICULAR REFRACTORY PERIOD

PACEMAKER VENTRICULAR
REFRACTORY PERIOD
AVOIDS THE SENSING OF:
* its own stimulus
* the paced QRS complex
* the T wave
* (excessive) afterpotential
* the combination of T wave and afterpotential

The duration of the pacemaker ventricular refractory period (VRP) is usually 200 - 300 ms
RIGHT VENTRICULAR PACING & OLD ANTERIOR MYOCARDIAL INFARCT

You cannot imagine what little details these cardiologists are looking at...!

A qR or Qr pattern in leads V5 and V6 often indicates an old anterior myocardial infarct (MI). This pattern may also occur in leads I and aVL.

A shelf-like notch (0.04 sec) on the ascending limb of the S wave, called Cabrera’s sign, in leads V2 - V5 often indicates an old anterior myocardial infarct.

In the case of Cabrera’s sign, rule out ventricular fusion beats and retrograde P waves.
VENTRICULAR PACING & THE MEMORY EFFECT

The underlying ECG cannot be used for the diagnosis of cardiac ischemia because inverted T waves may be due to the memory effect !!!

BEFORE PACING

II

aVF

V4

time

time

time

DURING PACING

II

aVF

V4

time

time

time

AFTER PACING

II

aVF

V4

time

time

time

For some time after pacing, the heart seems to remember the abnormal depolarizations. The duration of the memory effect (negative T waves) depends upon the duration of pacing.
FUNCTIONS OF THE POSTVENTRICULAR ATRIAL REFRACTORY PERIOD (PVARP)

PVARP Interval after a ventricular paced or sensed event during which the atrial channel is refractory !!!

1. Avoids the inappropriate atrial sensing of ventricular events (ventricular stimuli, QRS complexes, aberrant T waves)

Atrial electrogram (A-EGM)

Ventricular electrogram (V-EGM)

2. Avoids sensing of retrogradely conducted P waves

Surface ECG
Setting up pacing (TPM)

- **Threshold**: set the output to 3V and pace at a rate above the intrinsic cardiac rate.
- **Slowly turn down** the box output watching the ECG monitor.
- **Identify** the point where capture is lost. The output where the ventricle is recaptured is the pacing threshold.
- **Aim for a threshold** of <1V.
- **Set output** to at least 3x the pacing threshold to ensure a good safety margin.
Setting the box:

- “output” should be set to 3 times the threshold
- Set to “demand”
- “Sensitivity” should be adjusted to ensure that each intrinsic beat is detected but that skeletal muscle interference does not lead to pacemaker inhibition
HARDWARE

**Hardware**

**Lead Selection**

The choice of leads for transvenous pacing and defibrillation depends on the patient's anatomy and the position of the electrodes. The bipolar leads can be positioned in the left atrium, right atrium, or right ventricle. The unipolar leads can be positioned in the right atrium, right ventricle, or coronary sinus.

**Dwell Time**

The dwell time is the duration that the electric current is applied to the heart. It is typically set to 50 to 200 milliseconds to ensure effective defibrillation.

**Energy Level**

The energy level is the amount of electrical energy delivered to the heart. It is typically set to 10 to 40 joules for monophasic defibrillation and 5 to 20 joules for biphasic defibrillation.

**Sensing and Detection**

Sensing involves detecting the electrical activity of the heart. The defibrillator uses this information to determine the need for treatment. Detection involves identifying the presence of fibrillation or flutter.

**Intracardiac Electrograms**

Electrograms are used to assess the quality of the electrical signal. They are recorded using intracardiac catheters and can be used to identify the location of the electrodes and the presence of electrical activity.
FIGURE 38-9  Schematic representation of the timing cycle interactions of most refractory and blanking periods available on contemporary dual-chamber pacemakers. PVAB, postventricular atrial blanking; PVARP, postventricular atrial refractory period.
FIGURE 38-13  A, Schematic example of AV search hysteresis, which successfully demonstrates intrinsic AV conduction. B, Example of managed ventricular pacing. Initially, AAIR pacing is seen; if an atrial pace event occurs without a ventricular sensed event, a ventricular backup output occurs and the pacemaker then switches to DDDR mode.
**TABLE 38-1 NASPE/BPEG Generic Code for Anti Bradycardia Pacing**

<table>
<thead>
<tr>
<th>POSITION</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category</td>
<td>Chamber(s) paced</td>
<td>Chamber(s) sensed</td>
<td>Response to sensing</td>
<td>Rate modulation</td>
<td>Multisite pacing</td>
</tr>
<tr>
<td></td>
<td>O = None</td>
<td>O = None</td>
<td>O = None</td>
<td>O = None</td>
<td>A = None</td>
</tr>
<tr>
<td></td>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>T = Triggered</td>
<td>R = Rate modulation</td>
<td>A = Atrium</td>
</tr>
<tr>
<td></td>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>I = Inhibited</td>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
</tr>
<tr>
<td></td>
<td>D = Dual (A + V)</td>
<td>D = Dual (A + V)</td>
<td>D = Dual (t + I)</td>
<td>D = Dual (A + V)</td>
<td>D = Dual (A + V)</td>
</tr>
</tbody>
</table>

Manufacturers' designation only

S = Single (A or V)

S = Single (A or V)

See text for explanation of use of the code.

BPEG = British Pacing and Electrophysiology Group; NASPE = North American Society of Pacing and Electrophysiology.


**FIGURE 38-17** The VVI timing cycle consists of a defined lower rate (LR) limit and a ventricular refractory period (VRP, represented by triangle). When the LR limit timer is complete, a pacing artifact is delivered in the absence of a sensed intrinsic ventricular event. If an intrinsic QRS occurs, the LR limit timer is started from that point. A VRP begins with any sensed or paced ventricular activity.

**FIGURE 38-18** The AAI timing cycle consists of a defined lower rate (LR) limit and an atrial refractory period (ARP). When the LR limit timer is complete, a pacing artifact is delivered in the absence of a sensed atrial event. If an intrinsic P wave occurs, the LR limit timer is started from that point. An ARP begins with any sensed or paced atrial activity. In the AAI mode, only atrial activity is sensed. In this example, it may appear unusual for paced atrial activity to occur so soon after intrinsic ventricular activity. Because sensing occurs only in the atrium, this activity would not be expected to reset the pacemaker's timing cycle. (From Hayes and Levine. By permission of Blackwell Scientific Publications.)

**FIGURE 38-19** The timing cycle in DDD consists of a lower rate (LR) limit, an atrioventricular (AV) interval, a ventricular refractory period, a postventricular atrial refractory period (PVARP), and an upper rate limit. If intrinsic atrial and ventricular activity occurs before the LR limit times out, both channels are inhibited and no pacing occurs. In the absence of intrinsic atrial and ventricular activity, AV sequential pacing occurs (first cycle). If no atrial activity is sensed before the ventriculoatrial (VA) interval is completed, an atrial pacing artifact is delivered, which initiates the AV interval. If intrinsic ventricular activity occurs before the termination of the AV interval, the ventricular output from the pacemaker is inhibited, that is, atrial pacing (second cycle). If a P wave is sensed before the VA interval is completed, output from the atrial channel is inhibited. The AV interval is initiated, and if no ventricular activity is sensed before the AV interval terminates, a ventricular pacing artifact is delivered, that is, P-synchronous pacing (third cycle). ID = intrinsic deflection; TARP = total atrial refractory period.
FIGURE 38-23 Electrocardiographic tracing from a patient with a single-chamber pacemaker. The tracing was obtained when the patient was exposed to an external source of electromagnetic interference, which profoundly inhibited ventricular output, that is, ventricular oversensing.
Troubleshooting

1. Failure to capture
2. Failure to pace or output
3. Undersensing
4. Oversensing
5. Pacing at a rate not consistent with the programmed rate
Failure to capture

- Pacing stimulus without subsequent cardiac depolarization
- It may be related to the pacing system, the patient, or patient-system interaction.
- Stimulus occurs in the physiologic refractory period of a depolarization
LOSS OF VENTRICULAR CAPTURE BY VISIBLE PACEMAKER STIMULI

1. FUNCTIONAL
   ✓ Normal situation: stimuli in myocardial refractory period.

2. ELECTRODE-TISSUE INTERFACE
   - LEAD DISPLACEMENT
     ✓ Early displacement or unstable position of pacing leads (commonest cause).
     ✓ Malposition into the coronary venous system.
     ✓ Twiddler’s syndrome causing late displacement.
     ✓ Perforation of right ventricle by ventricular lead.

   - NO APPARENT LEAD DISPLACEMENT
     ✓ Microdislodgement (a diagnosis of exclusion) causes a marked rise in capture threshold but displacement is not apparent on a chest x-ray.
     ✓ Elevated pacing threshold without obvious lead displacement (exit block): Acute or chronic reaction at the electrode-tissue interface.
     ✓ Subcutaneous emphysema.
     ✓ Myocardial infarction or ischemia, hypoxia.
     ✓ Hypothyroidism.
     ✓ Elevation of pacing threshold after defibrillation or cardioversion. This is usually transient for a few minutes or less.
     ✓ Electrolyte abnormalities usually hyperkalemia, severe acidosis.
     ✓ Drug effect: Flecainide and propafenone can elevate the pacing threshold with therapeutic doses.

3. ELECTRODE
   ✓ Fracture, short circuit or insulation break.

4. PULSE GENERATOR
   ✓ Normal pacemaker with incorrect programming of parameters.
   ✓ Pacemaker failure from exhaustion or component failure.
   ✓ Iatrogenic causes: Component failure after defibrillation, electrocautery and therapeutic radiation.
Failure to pace

- Is most commonly due to oversensing of physiologic or nonphysiologic signals, resulting in inhibition of the pacing output.
- It may be caused by failure of the pulse generator or an open circuit (a lead fracture or a loose set screw).
CAUSES OF MISSING STIMULI DURING VVI PACING

Loose connection?
Lead fracture?

PM
Air entrapment in the pocket?
Subcutaneous emphysema?
(poor anodal contact of a unipolar system)

LEAD

Extraneous non-physiologic signals

PM

* total battery depletion?
* component failure?
* sticky reed switch?

* electromagnetic interference (industrial equipment) *
* oversensing of myopotentials, afterpotential, etc.?

* fast intrinsic rate?
* hysteresis ON?
**ANALYSIS OF LEAD PROBLEMS**

Be cunning as a fox! You can get a lot of information about the leads, just by looking at the pacing impedance and the voltage threshold. The secret is to look at both!

<table>
<thead>
<tr>
<th></th>
<th>IMPEDANCE</th>
<th>VOLTAGE THRESHOLD</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL LEAD PLACEMENT</td>
<td>NORMAL</td>
<td>NORMAL</td>
</tr>
<tr>
<td>LEAD DISPLACEMENT OR EXIT BLOCK</td>
<td>NORMAL</td>
<td>HIGH</td>
</tr>
<tr>
<td>LEAD FRACTURE</td>
<td>HIGH</td>
<td>HIGH</td>
</tr>
<tr>
<td>LEAD INSULATION DEFECT</td>
<td>LOW</td>
<td>MAY BE MODERATELY INCREASED</td>
</tr>
</tbody>
</table>
FIGURE 38-22  Three-channel tracing from an ambulatory monitor that was obtained because the patient had recurrent symptoms after pacemaker implantation. The tracing demonstrates intermittent failure to capture, and the pacemaker malfunction correlated with symptoms. The patient was found to have developed excessively high pacing thresholds on epicardial pacing leads.
FIGURE 38-11  Electrocardiographic example of crosstalk. Although crosstalk appears to be the most likely cause of ventricular failure to output when the surface ECG is assessed, it is confirmed by the telemetered ladder diagram. The ladder diagram, a type of diagnostic channel that is not commonly seen with contemporary devices, confirms that the atrial output was detected almost simultaneously on the ventricular sensing channel and that ventricular output was inhibited. P, paced; S, sensed.
Is a specific form of oversensing in which the atrial pacing stimulus is sensed on the ventricular channel, resulting in inhibition of ventricular output.
Pacing at a rate not consistent with the programmed rate

- Pacing with a shorter than expected escape interval indicates undersensing of myocardial depolarization
Failure to Respond to Resynchronization Pacing

- Caused by: -- Patient-related factors
- -- System-related Factors
- -- Interaction between the patient and the system
Resynchronization of less than 90% of R-R intervals caused by conducted AF or frequent PVCs.
System-related:

- Loss of LV capture due to lead dislodgement

- Patient-system interaction:
  Placement of the LV lead at an ineffective site for resynchronization and chronic changes in pacing threshold that can occur in any pacing system
Implant-related complications

- Hematoma
- Traumatic pneumothorax
- Inadvertant arterial puncture
- Air embolism
- A-V fistula
- Thoracic duct injury
- Subcutaneous emphysema
- Brachial plexus injury
- LV lead
- Tamponad (acute- asymptomatic)
Lead–related complications

- Dislodgment
- Header-connector pin problems
- conductor/cable(lead) fracture
- Insulation break
Complications of pacemakers

- Two major groups of complications are associated with pacemaker implantation:
  (a) nonelectrical complications including acute complications at the time of implantation such as pneumothorax and complications of lead placement and pocket formation;
  (b) electrical complications.
# Nonelectrical complications

## Table 7. Nonelectrical or arrhythmic complications.

<table>
<thead>
<tr>
<th>Venous access</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Hemothorax</td>
</tr>
<tr>
<td>Air embolism</td>
</tr>
<tr>
<td>Brachial plexus injury</td>
</tr>
<tr>
<td>Thoracic duct injury</td>
</tr>
<tr>
<td>Trauma to the subclavian artery</td>
</tr>
<tr>
<td>Hematoma</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pacemaker pocket</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection, septicemia, etc. Conservative therapy is often unsuccessful and removal of the entire system may be required</td>
</tr>
<tr>
<td>Hematoma/seroma</td>
</tr>
<tr>
<td>Erosion</td>
</tr>
<tr>
<td>Pacemaker migration</td>
</tr>
<tr>
<td>Twiddler’s syndrome</td>
</tr>
<tr>
<td>Muscle stimulation from either a flipped but normally functioning unipolar or extravascular insulation defect</td>
</tr>
<tr>
<td>Chronic pain including subcuticular malposition of the pulse generator</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intravascular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subclavian or inominate vein thrombosis</td>
</tr>
<tr>
<td>Thrombosis of superior vena cava</td>
</tr>
<tr>
<td>Coronary sinus dissection or perforation during implantation of a left ventricular lead</td>
</tr>
<tr>
<td>Large right atrial thrombus</td>
</tr>
<tr>
<td>Endocarditis with vegetations</td>
</tr>
<tr>
<td>Manifest pulmonary embolism (rare)</td>
</tr>
<tr>
<td>Cardiac perforation</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Entanglement of lead in the tricuspid valve and ruptured chordae</td>
</tr>
<tr>
<td>Tricuspid insufficiency</td>
</tr>
<tr>
<td>Pericardial rub</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lead problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Displacement</td>
</tr>
<tr>
<td>Malposition in the coronary venous system</td>
</tr>
<tr>
<td>Endocardial left ventricular malposition across a patent foramen ovale or via subclavian arterial puncture (or via atrial or ventricular septum defect)</td>
</tr>
<tr>
<td>Right ventricular perforation or lead perforation of the interventricular septum</td>
</tr>
<tr>
<td>Diaphragmatic pacing. Left side with or without right ventricular perforation and right side by phrenic nerve stimulation by atrial pacing</td>
</tr>
<tr>
<td>Intercostal muscle stimulation due to right ventricular perforation</td>
</tr>
<tr>
<td>Post pericardiectomy syndrome (pericarditis etc.) with or without lead perforation</td>
</tr>
<tr>
<td>Intracardiac rupture of lead during attempt to remove old or broken lead</td>
</tr>
</tbody>
</table>