

Cardiac Pacing

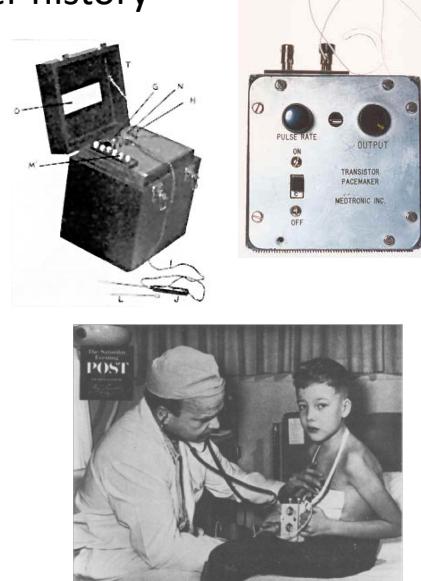
2019/02/21

What you can expect to learn today

- How stimulation concepts can be applied to cardiac pacing
 - Strength-Duration curve
 - Point vs. Field pacing
 - Anodal vs. Cathodal pacing
 - Activating function/virtual electrodes
- Physiological considerations
 - When/where to pace?
 - Class engineering discussion
 - Chronic electrode effects?
 - Pacemaker modes of operation
 - Class engineering discussion
 - ATP (Not what you think.)
 - CRT
 - HBP
 - PITA (Also not what you think!)

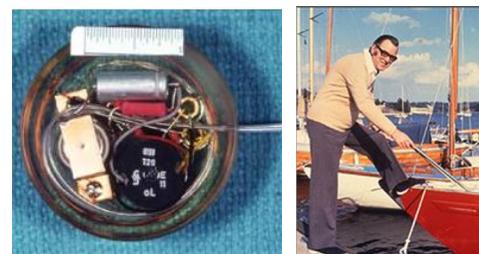
Pacemaker history

- **1932:** Albert Hyman developed the “artificial cardiac pacemaker.” He described his early work: “Finally on April 6, 1930, I received Grant No. 30-2 from the Witkin Foundation to explore the possibility of developing a practical machine, to be used as an artificial pacemaker in experimental animals.”
- **1958:** Earl Bakken made the first wearable external pacemaker for a patient of Dr. C. Walton Lillehei. Allowed adjustment of pacing rate and voltage and was connected to electrode leads which passed through the skin epicardial surface.



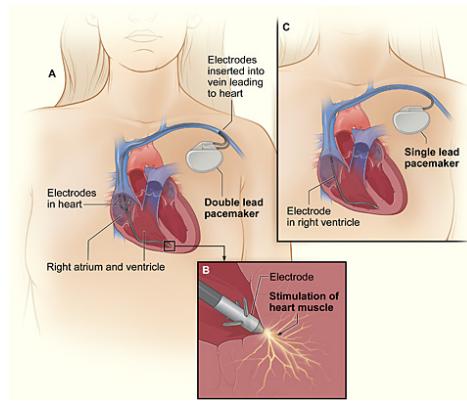
Pacemaker history

- **1958:** The first fully implantable pacemaker, designed by Rune Elmqvist, was implanted by surgeon Åke Senning at the Karolinska Institute in Solna, Sweden.
 - Connected via epicardial electrodes implanted by thoracotomy. The first device failed after three hours and the second lasted two days.
- Arne Larsson: The world's first implantable pacemaker patient. He received 26 pacemakers in his lifetime. He died in 2001, at 86, outliving both Senning and Elmqvist.



Pacing Characteristics and Locations

- Typical pulse is < 1.0 ms and <3.0 V. (~2-3x threshold). The lead is intravascular and can be placed in multiple locations:
 - A single-chamber pacemaker lead is typically in the right ventricle
 - A dual-chamber pacemaker lead is typically in the right atrium and the right ventricle.
 - A biventricular pacemaker typically has leads in the right atrium and both ventricles. This is used for cardiac resynchronization therapy.



Indications for pacemakers

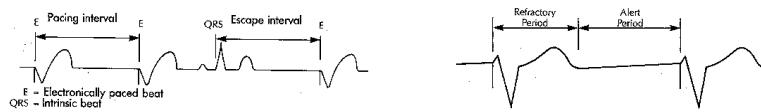
- Symptomatic bradycardia
 - Frequent sinus pause
 - Drug induced
- Symptomatic chronotropic incompetence**
- Unexplained syncope with electrophysiological abnormalities
- AV block
 - 3rd degree (complete)
 - 2nd degree (one or more, but not all, p waves are blocked)
 - Symptomatic 1st degree (slowed conduction --where would we notice this?)
- Post MI

Class discussion 1:

- With your knowledge of electrical measurement and stimulation and cardiac physiology can you design a pacemaker for these different clinical situations?
- In two-three large group discuss each case. Each of you will then present one of the cases and your solution. You should consider any relevant signals you would measure and where/when you would pace. Basically you're creating a pacemaker algorithm.
 - Third degree heart block (complete AV dissociation).
 - Sick sinus syndrome
 - Third degree heart block with AF
 - Symptomatic first degree heart block
 - HF with LBBB

Fixed Rate vs Demand Pacing

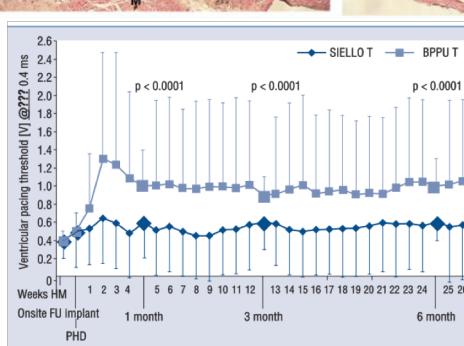
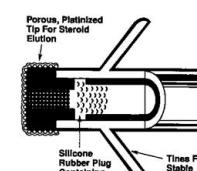
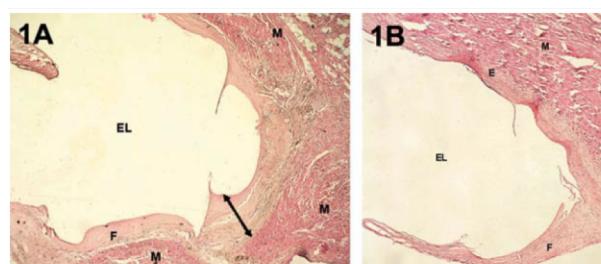
- Fixed-rate pacemakers ignore intrinsic HR and pace at a preset rate all the time.
- Demand pacemakers pace only when needed (on demand). This requires additional engineering:
 - *Sensing*: To detect the intrinsic rate and inhibit pacing if detected
 - *Pacing*: Stimulate when no intrinsic beat is sensed (but only within a fixed period!).
- Demand pacemakers can be temporarily converted to fixed-rate mode by placing a special magnet on the chest wall over the battery. (e.g. to test pacemaker functionality).
- Demand pacemakers are programmable -- the pacing rate can be adjusted once implanted with a special telemetry device on the chest.
- Demand ventricular pacemakers are almost always QRS inhibited.
 - There is a predefined “escape rate” of the pacemaker (e.g., 50 bpm) above which they don’t pace.
 - This occurs on a beat-to-beat basis.
- QRS inhibited pacemakers have a refractory (to sensing) period (~0.4 s) and an alert period (where they look for QRS complexes).



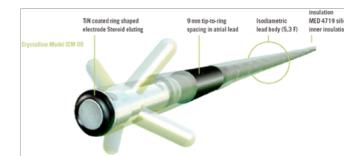
What are the side-effects

- Chronic pacing increases stimulation threshold!

Inflammation at the Electrode



The strength-duration curve is altered!

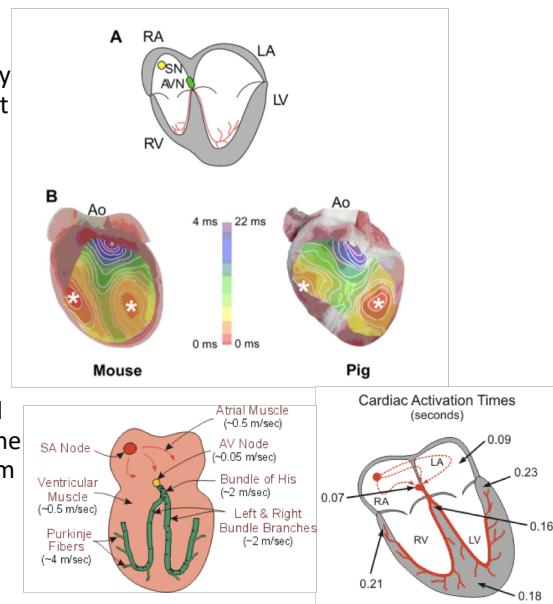


What are the side-effects

- Chronic pacing increases stimulation threshold!
- Pacing activation pattern is often non-physiological.

Activation Pattern in the Human Heart

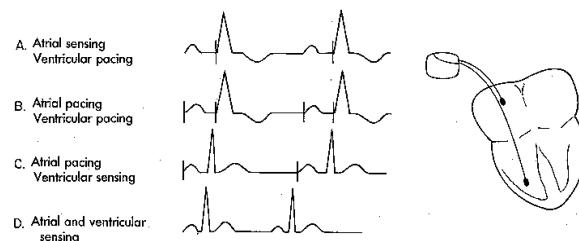
- Depolarization waves initially reach the endocardial aspect of LV at three points simultaneously
 - An area high on the anterior paraseptal wall just below the mitral valve.
 - A central area in the left side of the interventricular septum.
 - The posterior paraseptal area at approximately one third of the distance from the apex to the base.



Dual-Chamber Pacemakers

- Electrodes are inserted into both the right atrium and right ventricle.
- The atrial lead is able to sense the patient's intrinsic P waves and stimulates the atrium when the atrial rate becomes too slow.
 - Can now control *atrioventricular (AV) delay* (analogous to the PR interval on a normal ECG).
- Dual-chamber pacing is used to maintain physiologic timing between atrial and ventricular systole.
 - With ventricular pacing alone, this loss of timing can cause significant reduction in cardiac output.
 - Very useful for exercise. Why?

Dual-Chamber (DDD) Pacemaker Functions



What are the side-effects

- Chronic pacing increases stimulation threshold!
- Pacing activation pattern is often non-physiological.
- What if you need your heart rate to change?

Chronotropic Incompetence

- If the patient's heart cannot increase its rate appropriately in response to increased activity, the patient is chronotropically incompetent
- Classification:
 - Maximum heart rate < 90% x (220 - Age)
 - Maximum heart rate < 120 b p m
- Causes:
 - Aging
 - Drugs
 - Heart disease

Rate-adaptive pacing

United States Patent [19]
Bornzin

[11] Patent Number: **4,467,807**
[45] Date of Patent: Aug. 28, 1984

[54] RATE ADAPTIVE DEMAND PACEMAKER

[75] Inventor: Gene A. Bornzin, Coon Rapids, Minn.

[73] Assignee: Medtronic, Inc., Minneapolis, Minn.

[21] Appl. No.: 323,507

[22] Filed: Nov. 23, 1981

[51] Int. Cl.³ A61N 1/36

[52] U.S. Cl. 128/419 PG

[58] Field of Search 128/419 PG

lished in Biomedizinische Technik, Band 20, Heft 6/1975.

Abstract "Results, Problems and Perspectives in the Autoregulating Pacemaker" by Cammelli et al., published in the May-Jun. 1980 edition of Pace Magazine. Article entitled "A Physiologically Controlled Cardiac Pacemaker" by J. L. Krasner et al., published in the Journal of the American Association for Medical Instrumentation, vol. 1, No. 3, Nov./Dec. 1966.

An article entitled "A New Pacemaker Autoregulating the Rate of Pacing Relation to Metabolic Needs," by Cammelli, et al., published in the Proceedings of the 5th International Symposium, 1976 Tokyo.

Primary Examiner—William E. Kamm
Attorney, Agent, or Firm—Reed A. Duthler; John L. Rooney; Joseph F. Breitmayer

[57] **ABSTRACT**

An implantable pacer having an effective stimulation rate which varies in response to a measured physiolog-

Class discussion 2:

- The healthy heart responds to increased physiological demand to increase HR
- How would you design a rate-adaptive pacer?
 - What do we want to accomplish?
 - What parameters should we measure and how?
 - What are the advantages and disadvantages?
 - How would you implement this (how would you use the parameter to alter rate)?

We need:

- Prompt chronotropic response.
- High specificity and sensitivity.
- Proportional to metabolic demand.
- Appropriate recovery rate decay to match metabolism (i. e. fast after short exercise but prolonged after longer and maximal exercise .
- Reduce the complexity of programming.

What's been used before?

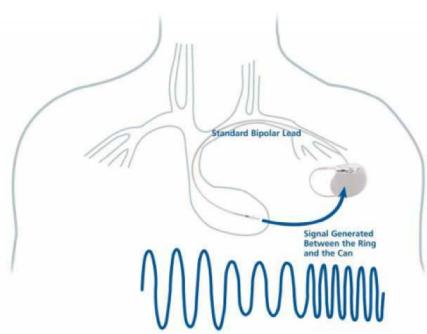
- QT interval shortening
- O₂ tension in intracardiac or pulmonary artery venous blood
- Increase in respiratory or minute-ventilation rate (RR*TV)
- Increased mean atrial rate
- Rise in central venous temperature
- Decrease in venous blood pH
- Increase in right ventricular stroke volume
- Increase in ventricular inotropy
 - (e.g. peak endocardial acceleration and ventricular impedance variation).
- Body motion
- Vibration
- Evoked response

Some pros and cons:

- Body acceleration:
 - Responds rapidly
 - No special pacing leads required
 - Easy to manufacture and program
 - Cannot be “fooled” by pressure on the can
 - Non-physiologic (low specificity, high sensitivity), and miss metabolically demanding isometric activity.
- Vibration:
 - Can be “fooled” by pressure on the can
- QT Interval:
 - Measures the interval between the pacing spike and the evoked T-wave, which should shorten with exercise
 - Highly specific, but can’t be used with acute MI, is affected by drugs and electrolyte imbalance, and many conditions correlated with HF (e.g. circulating catecholamines).
 - Requires no special pacing lead, but works only when pacing
- Temperature:
 - Thermistor on the lead (not can)
 - Responds to metabolism
 - Requires a special lead and can be slow

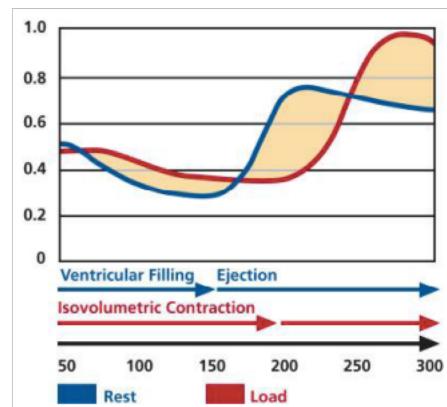
Minute ventilation:

- Uses low-level electrical signals to measure chest impedance
- Impedance increases during inhaling, and decreases during exhaling
- Detect respiration rate and relative change in tidal volume ($MV = RR \times TV$)
- Requires no special sensor
- Provides metabolic info
- Can’t be reliably used for patients with pulmonary disorders
- Requires a relatively high amount of processing power



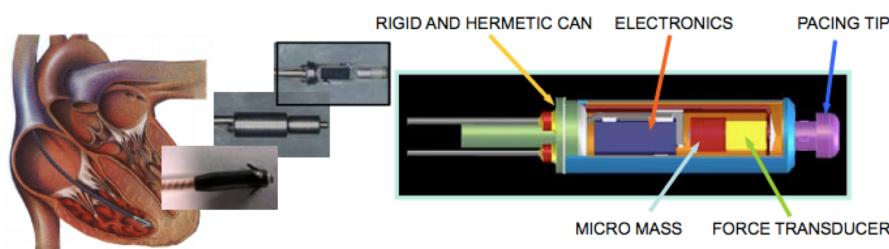
Closed Loop System (CLS)

- Measures changes in cardiac contractility (inotropy) via intracardiac impedance
- Increases during systole and decreases during diastole
- May be affected by postural changes



Peak endocardial acceleration

- An accelerometer inserted in the tip of a pacing lead for the chronic measurement of the Endocardial Acceleration (EA)
- Peak EA is related to the first heart sound, which reflects cardiac contractility
- Measures changes in cardiac contractility
- Proportional to metabolic demand and is fast
- Requires a special sensor and a large lead body
- Susceptible to false positives

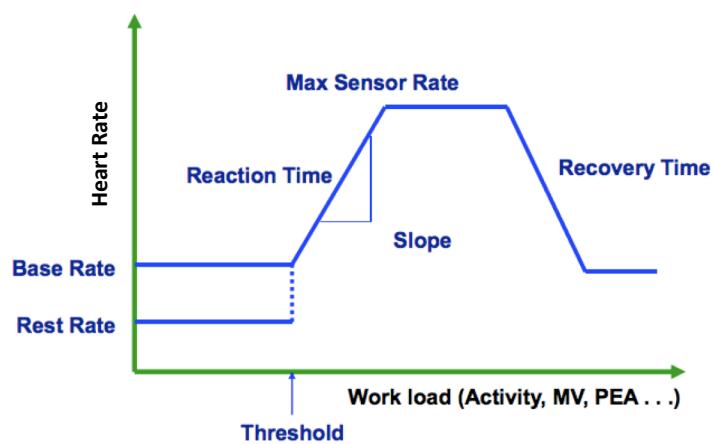


Evoked Response



- Measures the QRS depolarization AUC with the expectation that the QRS AUC decreases in area with exercise
- Requires no special leads
- May be affected by postural changes
- Only works when pacing

How do we use these signals?



Pacemaker codes

Position I	Position II	Position III	Position IV	Position V
Pacing chamber	Sensing chamber	Response to sensing	Programmability	Anti-tachycardia functions
O=none	O=none	O=none	O=none	O=none
A=atrium	A=atrium	I=inhibited	R=rate modulation	P=pace
V=ventricle	V=ventricle		T=triggered	S=shock
D=dual (atrium and ventricle)	D=dual (atrium and ventricle)		D=dual (inhibited and triggered)	D=dual (pace and shock)

Pacemaker Codes

Code	Meaning	When is it used?
AOO	Atrial pace, no sense, no inhibitions	Sick sinus syndrome with intact conduction in the operating room with bovie (electrical cautery). e.g. with heart rate low from narcotics.
AAI	Atrial pace, atrial sense, inhibited by atrium	Sick sinus syndrome with intact conduction system.
VOO	Ventricular pace, no sense, no inhibit	Third degree heart block in OR with atrial fibrillation. Why atrial fibrillation? Because you can't effectively pace the atrium if it is fibrillating.
VVI	Ventricular pace, ventricular sense, ventricular inhibit	Third degree heart block with atrial fibrillation.
DOO	Dual pace, no sense, no inhibitions	Third degree heart block in OR with bovie.
DVI	Dual pace, ventricular sense, ventricular inhibit	Third degree heart block with supraventricular tachycardias
DDD	Dual pace, dual sense, dual inhibit	Third degree heart block.

Heart Failure

- What is it?
 - Pump dysfunction that results in an inability to meet the metabolic demands of the body.
 - Commonly caused by coronary artery disease (including prior MI), high blood pressure, AF, alcoholism, infection, cardiomyopathy.
 - Two primary types:
 - Preserved ejection fraction (HFpEF) – inadequate filling. $EF=SV/EDV = 50-70\%$
 - LV dysfunction ($EF<40\%$)
 - Also characterized in other ways:
 - Left vs. right sided
 - Systolic vs. diastolic dysfunction (same as before).
 - Increased preload vs. increased afterload.
 - Low CO with high resistance vs. high CO with low resistance
 - Graded by the degree of exercise tolerance. (NYHA functional classification).
 - Characterized by stable low-level performance punctuated by periods of acute decompensation.

NYHA Class	Symptoms
I	Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients.

Non-Pharmacological Treatment

- AICD – purely to reduce mortality. Does nothing for symptoms (HF with $EF<35\%$ has much higher rates of VT)
- Cardiac Contractility modulation – for patients NYHA II-IV. Best for patients with normal QRS duration (<120 ms). Improves symptoms QoL, and exercise tolerance.
- CRT uses biventricular pacing to ‘resynchronize’ the heart.
 - ~1/3 of HF with $EF<35\%$ have significant ventricular dyssynchrony, particularly in LBBB.
 - NYHA II or IV, QRS duration >120 ms.
 - In the remaining 2/3 of HF patients it may be harmful.
- Pacing-induced transient asynchrony
 - Could be used in remaining 2/3 of patients without dyssynchrony