Dr. David Martínez Duncker R.
DMDR·MDPhD®kardiagnostx.com

Facultad de Medicina
Universidad Autónoma del Estado de Morelos

Curso - Fundamentos Básicos de Electrocardiografía Clínica 2019

Recomendaciones para la Estandarización en la Interpretación del ECG AHA / ACC

Entrada libre
Auditorio - Facultad de Medicina. UAEM
Constancia de Asistencia
Valor curricular 6 horas

Profesor Titular
Dr. David Martínez Duncker R.
Fisiología I y Cardiología

Cupo Limitado

- Introducción.
- Electrofisiología Cardiaca y Correlación entre la Curva del PAT y el ECG.
- Sistemas de Inscripción y Nomenclatura de las Ondas.
- Derivaciones y Hemicampos, Eje Eléctrico y Frecuencia Cardiaca.
- Cardiopatía Isquémica - isquemia, lesión, necrosis.
- Crecimiento de Cavidades.
- Bloqueos Auriculo-Ventriculares y de Rama.
- Mecanismo de las Arritmias.

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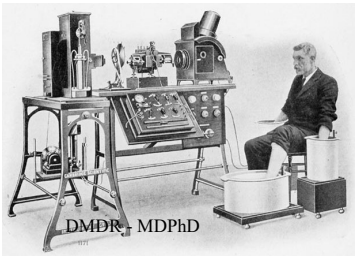
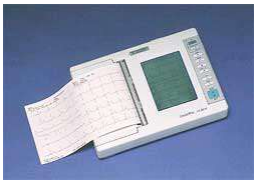
ELECTROCARDIOGRAFIA

El electrocardiograma (ECG) es un registro lineal de la activación eléctrica del corazón (despolarización y repolarización del músculo cardiaco) a lo largo del tiempo. Por cada ciclo cardiaco se registran, sucesivamente, la curva de **despolarización auricular** (asa de P), la curva de **despolarización ventricular** (asa de QRS) y la curva de **repolarización ventricular** (asa de T).





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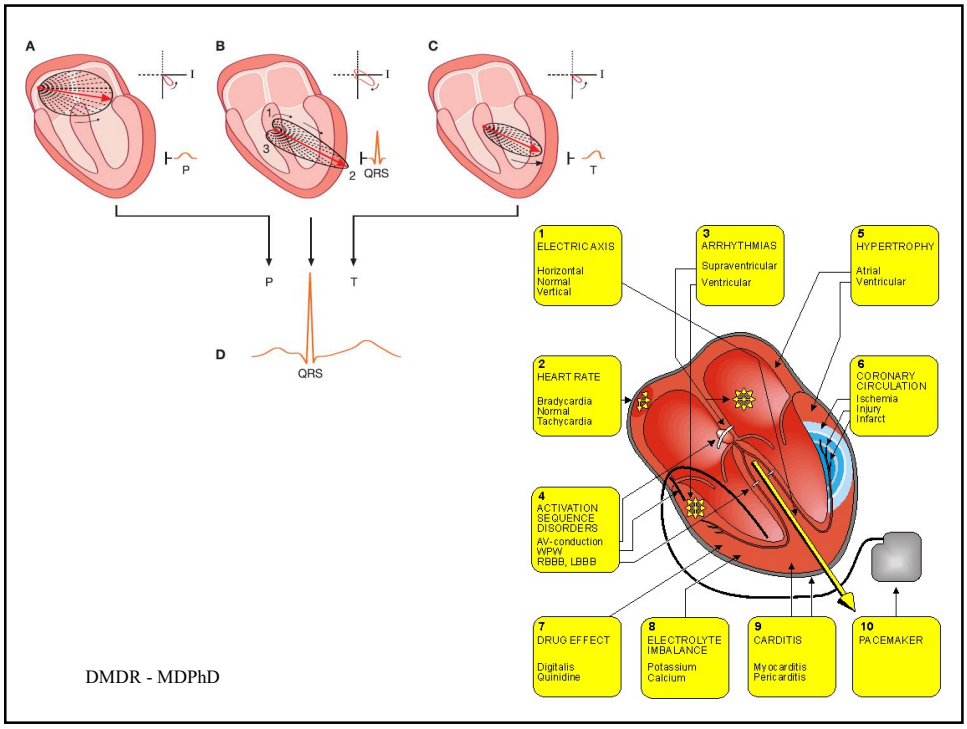

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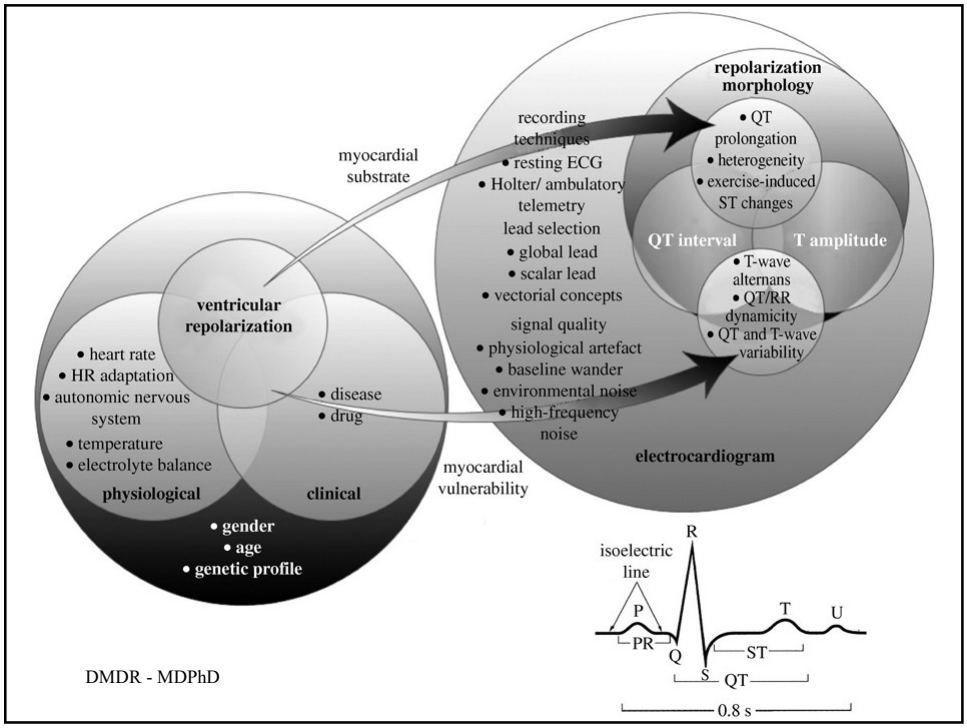

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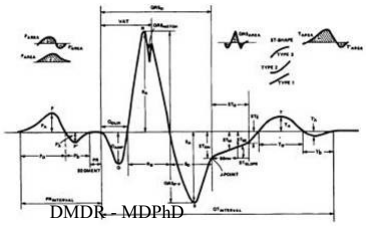
CLINICIAN UPDATE

Electrocardiogram

Still the Cardiologist's Best Friend

Shlomo Stern, MD

The 12-lead surface ECG can indicate pathological changes even before structural changes in the heart can be diagnosed by other methods. The recording of an ECG was of great value for several past generations of cardiologists and continues to provide vital information. Researchers should further scrutinize Einthoven's ingenious method, and clinicians should continue to tap this important and reliable source of information.



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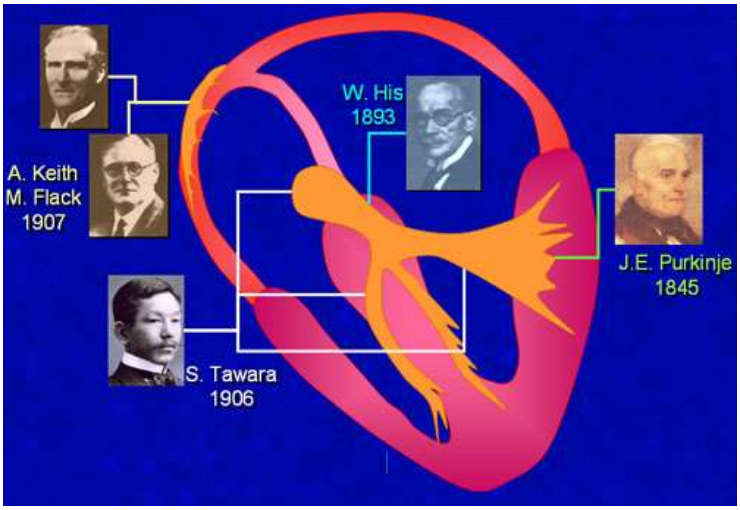
(*Circulation.* 2006;113:e753-e756.)

Why Does the Heart Beat?

The Discovery of the Electrical System of the Heart

Mark E. Silverman, MD; Daniel Grove, MD; Charles B. Upshaw, Jr, MD



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… de la instalación eléctrica del corazón

(*Circulation.* 2006;113:2775-2781.)

1873-1952

Sunao Tawara

Whenever any hypothesis is proposed, the system involved should be taken into consideration as a whole. Moreover, a conclusion derived from the anatomical architecture must eventually accord with the results of physiological experiments regarding the system."

—Sunao Tawara, 1906[1]

Descubrió el Nodo AV y su conducción.

1. Tawara S. *Das Reizleitungssystem Des Säugetierherzens [The Conduction System of the Mammalian Heart]*. Jena: Gustav Fischer; 1906. Suma K, Shimada M, trans. London, UK: Imperial College Press; 2000.

1863-1934

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Wilhem His Jr.

Descubrió la conducción entre el Nodo AV y las ramas del Haz de His

His W Jr. The story of the atrioventricular bundle with remarks concerning embryonic heart activity. *J Hist Med Allied Sci*. 1949;4:319–333.

1787-1869

Jan Evangelista Purkinge

Descubrió la conducción distal de las fibras ventriculares

Schweitzer P. Jan Evangelista Purkinje. *Clin Cardiol*. 1991;14:85–86.

1847-1914

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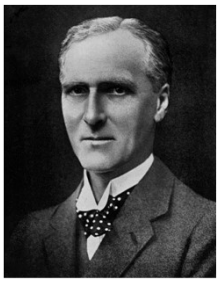
Walter Gaskell

Fue el primero en demostrar fibras de conducción
especializadas entre las aurículas y los ventrículos

Silverman ME, Upshaw CB Jr. Walter Gaskell and the understanding of atrioventricular block. *J Am Coll Cardiol*. 2002;39:1574–1580.



1866-1955

Heart 2007;93:1184–1187.

Arthur Keith

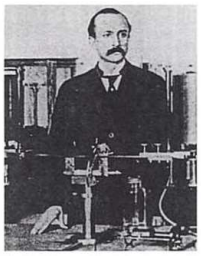
y

Martin Flack

"There is a remarkable remnant of primitive fibres persisting at the sino-auricular junction in all the mammalian hearts examined… in them the dominating rhythm of the heart is believed to normally arise."
Arthur Keith and Martin Flack[1]

Descubrieron el nodo sinusal

1882-1931



1866-1955

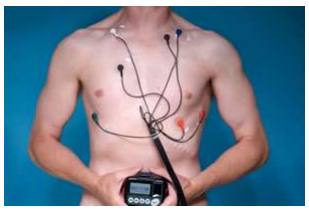
Jean George Bachmann

Un grupo de fibras del tracto internodal anterior que penetran en septo interatrial y diverge en la atrio izquierdo conectan ambos atrio.

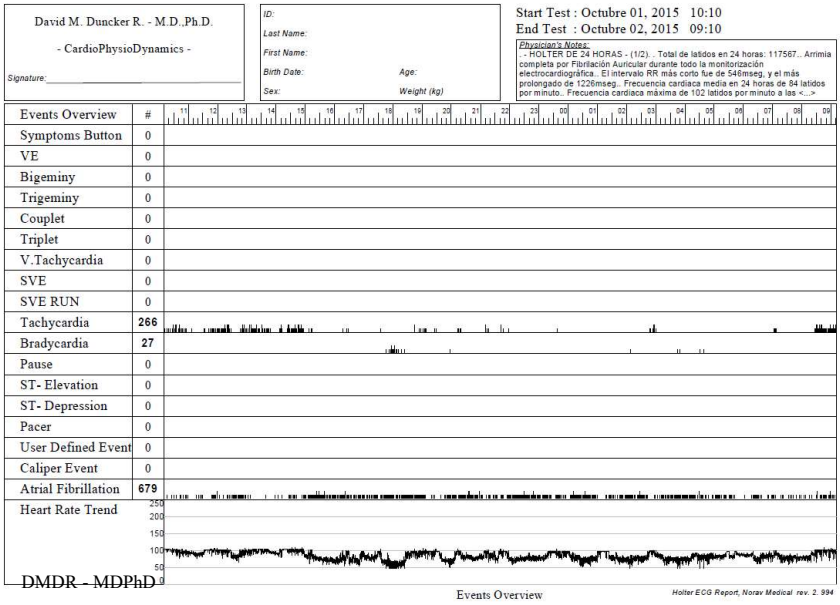
Descubrió las fibras interauriculares

Holter

Norman Jefferis Holter (1914–1983).



Holter de 24 horas - Monitorización ECG Ambulatoria



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- CardioPhysioDynamics -

Signature:

ID:
Last Name:
First Name:
Birth Date: Age:
Sex: Weight (kg)

Start Test : Octubre 01, 2015 10:10
End Test : Octubre 02, 2015 09:10

Physician's Notes:
...HOLTER DE 24 HORAS - (1/2)... Total de latidos en 24 horas: 117567... Arritmia completa por Fibrilación Auricular durante todo la monitorización electrocardiográfica...El intervalo RR más corto fue de 546mseg, y el más prolongado de 1226mseg... Frecuencia cardiaca media en 24 horas de 84 latidos por minuto... Frecuencia cardiaca máxima de 102 latidos por minuto a las <...>

Events Overview	#
Symptoms Button	0
VE	0
Bigeminy	0
Trigeminy	0
Couplet	0
Triplet	0
V.Tachycardia	0
SVE	0
SVE RUN	0
Tachycardia	266
Bradycardia	27
Pause	0
ST- Elevation	0
ST- Depression	0
Pacer	0
User Defined Event	0
Caliper Event	0
Atrial Fibrillation	679
Heart Rate Trend	

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Events Overview

Holter ECG Report, Norav Medical rev. 2. 994

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Signature:

ID:
Last Name:
First Name:
Birth Date: Age:
Sex: Weight (kg)

HOURLY TABULAR REPORT

Time	Symptoms Button	VE	Bigeminy	Trigeminy	Couplet	Triplet	V.Tachycardia	SVE	SVE RUN	Tachycardia	Bradycardia	Pause	ST-Elevation	ST-Depression	Pacer	User Defined Event	Caliper Event	Atrial Fibrillation
10:10	0	0	0	0	0	0	0	0	0	28	0	0	0	0	0	0	0	12
11:00	0	0	0	0	0	0	0	0	0	16	0	0	0	0	0	0	0	14
12:00	0	0	0	0	0	0	0	0	0	36	0	0	0	0	0	0	0	28
13:00	0	0	0	0	0	0	0	0	0	40	0	0	0	0	0	0	0	4
14:00	0	0	0	0	0	0	0	0	0	**47**	0	0	0	0	0	0	0	14
15:00	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	**56**
16:00	0	0	0	0	0	0	0	0	0	3	0	0	0	0	0	0	0	36
17:00	0	0	0	0	0	0	0	0	0	1	8	0	0	0	0	0	0	37
18:00	0	0	0	0	0	0	0	0	0	5	**13**	0	0	0	0	0	0	38
19:00	0	0	0	0	0	0	0	0	0	7	1	0	0	0	0	0	0	24
20:00	0	0	0	0	0	0	0	0	0	5	0	0	0	0	0	0	0	47
21:00	0	0	0	0	0	0	0	0	0	8	0	0	0	0	0	0	0	24
22:00	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	55
23:00	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	29
00:00	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	48
01:00	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	20
02:00	0	0	0	0	0	0	0	0	0	10	1	0	0	0	0	0	0	36
03:00	0	0	0	0	0	0	0	0	0	1	2	0	0	0	0	0	0	36
04:00	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0	0	33
05:00	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	27
06:00	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	18
07:00	0	0	0	0	0	0	0	0	0	4	0	0	0	0	0	0	0	15
08:00	0	0	0	0	0	0	0	0	0	37	0	0	0	0	0	0	0	22
09:00	0	0	0	0	0	0	0	0	0	14	0	0	0	0	0	0	0	6
09:10																		
Total	0	0	0	0	0	0	0	0	0	266	27	0	0	0	0	0	0	679

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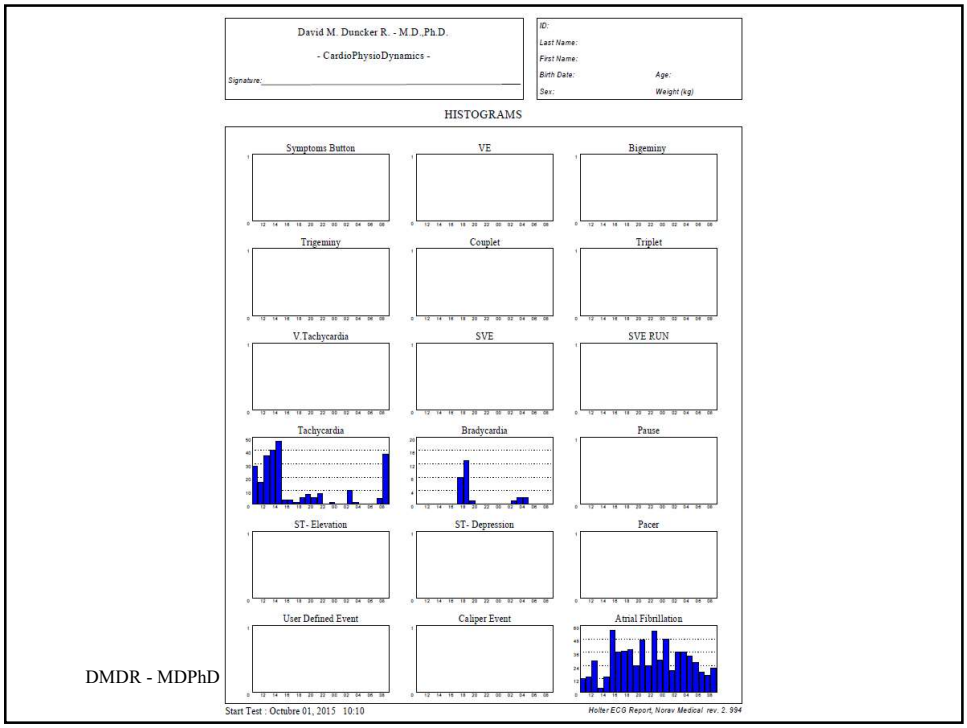

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- CardioPhysioDynamics -

Signature:_____

ID:
Last Name:
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HISTOGRAMS

Symptoms Button | VE | Bigeminy
Trigeminy | Couplet | Triplet
V.Tachycardia | SVE | SVE RUN
Tachycardia | Bradycardia | Pause
ST- Elevation | ST- Depression | Pacer
User Defined Event | Caliper Event | Atrial Fibrillation

Start Test : Octubre 01, 2015 10:10

Holter ECG Report, Norav Medical rev. 2. 994

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Table 1. Applying Classification of Recommendations and Level of Evidence

ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	SIZE OF TREATMENT EFFECT: CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	CLASS III *No Benefit* or CLASS III *Harm* Procedure/ Test / Treatment COR III: No benefit — Not Helpful — No Proven Benefit COR III: Harm — Excess Cost w/o Benefit or Harmful — Harmful to Patients
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care
Suggested phrases for writing recommendations	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit is not recommended is not indicated should not be performed/ administered/ other is not useful/ beneficial/ effective	COR III: Harm potentially harmful causes harm associated with excess morbidity/mortality should not be performed/ administered/ other
Comparative effectiveness phrases†	treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B it is reasonable to choose treatment A over treatment B			

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AHA/ACC/HRS SCIENTIFIC STATEMENTS

J. Am. Coll. Cardiol. 2007;49;1109-1127

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part I: The Electrocardiogram and Its Technology

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part II: Electrocardiography Diagnostic Statement List

J. Am. Coll. Cardiol. 2007;49;1128-1135

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

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EXPERT CONSENSUS DOCUMENTS

J. Am. Coll. Cardiol. 2009;53;976-981

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part III: Intraventricular Conduction Disturbances

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

J. Am. Coll. Cardiol. 2009;53;982-991

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part IV: The ST Segment, T and U Waves, and the QT Interval

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

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Endorsed by the International Society for Computerized Electrocardiology

J. Am. Coll. Cardiol. 2009;53;992-1002

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

J. Am. Coll. Cardiol. 2009;53;1003-1011

AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part VI: Acute Ischemia/Infarction

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

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GUIDELINES

2019 ESC Guidelines for the management of patients with supraventricular tachycardia The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC): Developed in collaboration with the Association for European Paediatric and Congenital Cardiology (AEPC)

FREE

Josep Brugada, Demosthenes G Katritsis, Elena Arbelo, Fernando Arribas, Jeroen J Bax, Carina Blomström-Lundqvist, Hugh Calkins, Domenico Corrado, Spyridon G Deftereos, Gerhard-Paul Diller ... Show more

European Heart Journal, ehz467, https://doi.org/10.1093/eurheartj/ehz467

Published: 31 August 2019

European Heart Journal (2019) **00**, 1–65
doi:10.1093/eurheartj/ehz467

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Clinical Research in Cardiology (2019) 108:577–599
https://doi.org/10.1007/s00392-018-1377-1

REVIEW

German Cardiac Society Working Group on Cellular Electrophysiology state-of-the-art paper: impact of molecular mechanisms on clinical arrhythmia management

Dierk Thomas[1,2,3] · Torsten Christ[4,5] · Larissa Fabritz[6,7,8] · Andreas Goette[9,10] · Matthias Hammwöhner[9,10] · Jordi Heijman[11,12] · Jens Kockskämper[13] · Dominik Linz[14,15] · Katja E. Odening[16,17,18] · Patrick A. Schweizer[1,2,3,19] · Reza Wakili[20] · Niels Voigt[21,22]

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Heart, Lung and Circulation (2019) 28, 155–163
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.10.004

REVIEW

Sudden Death and Ventricular Arrhythmias in Athletes: Screening, De-Training and the Role of Catheter Ablation

M. Darragh Flannery, MBBS [a,b], André La Gerche, MBBS, PhD [a,b,c*]

Heart, Lung and Circulation (2019) 28, 1–5
1443-9506/04/$36.00
https://doi.org/10.1016/S1443-9506(18)31972-3

EDITORIAL

The Contemporary Era of Sudden Cardiac Death and Ventricular Arrhythmias: Basic Concepts, Recent Developments and Future Directions

Haris M. Haqqani, MBBS, PhD [a,b,c,d*], Kim H. Chan, MBBS, PhD [a,b,e,f], Saurabh Kumar, BSc(Med), MBBS, PhD [a,f,g], A. Robert Denniss, MD, MSc [f,g,h,i,j], Anne T. Gregory, MBBS, Grad Cert Pop Health [k]

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Heart, Lung and Circulation (2019) 28, 57–64
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.08.027

REVIEW

Sudden Death Risk-Stratification in 2018–2019: The Old and the New

Sarah Zaman, MBBS, PhD [a,b], Jeffrey J. Goldberger, MD, MBA [c], Pramesh Kovoor, MBBS, PhD [d,e*]

JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY VOL. 73, NO. 1, 2019

PUBLISHED BY ELSEVIER

THE PRESENT AND FUTURE

JACC STATE-OF-THE-ART REVIEW

New Concepts in Sudden Cardiac Arrest to Address an Intractable Epidemic

JACC State-of-the-Art Review

Sanjiv M. Narayan, MD, PhD,[a] Paul J. Wang, MD,[a] James P. Daubert, MD[b]

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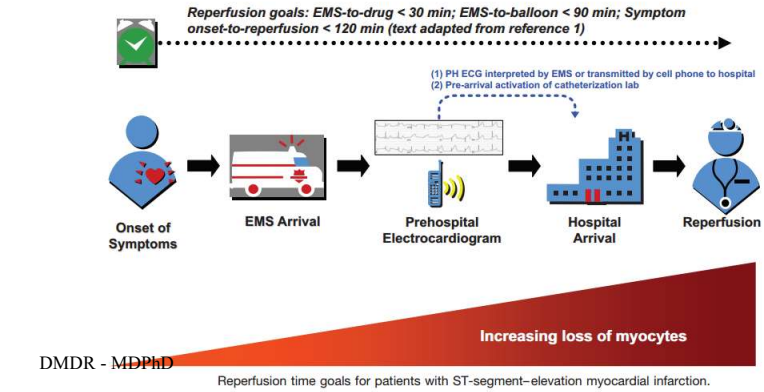
AHA Scientific Statement

Implementation and Integration of Prehospital ECGs Into Systems of Care for Acute Coronary Syndrome

A Scientific Statement From the American Heart Association Interdisciplinary Council on Quality of Care and Outcomes Research, Emergency Cardiovascular Care Committee, Council on Cardiovascular Nursing, and Council on Clinical Cardiology

Henry H. Ting, MD, MBA, Chair; Harlan M. Krumholz, MD, SM, FAHA, Co-Chair; Elizabeth H. Bradley, PhD; David C. Cone, MD; Jeptha P. Curtis, MD; Barbara J. Drew, RN, PhD, FAHA; John M. Field, MD; William J. French, MD; W. Brian Gibler, MD; David C. Goff, MD, PhD, FAHA; Alice K. Jacobs, MD, FAHA; Brahmajee K. Nallamothu, MD, MPH; Robert E. O'Connor, MD; Jeremiah D. Schuur, MD, MHS

(*Circulation.* 2008;118:1066-1079.)



Reperfusion time goals for patients with ST-segment–elevation myocardial infarction.

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AHA/ACCF Scientific Statement

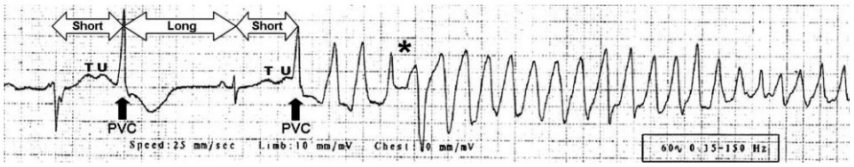
Prevention of Torsade de Pointes in Hospital Settings

A Scientific Statement From the American Heart Association and the American College of Cardiology Foundation

Endorsed by the American Association of Critical-Care Nurses, the International Society for Computerized Electrocardiology, and the Heart Rhythm Society

Barbara J. Drew, RN, PhD, FAHA, Chair; Michael J. Ackerman, MD, PhD, FACC; Marjorie Funk, RN, PhD, FAHA; W. Brian Gibler, MD, FAHA; Paul Kligfield, MD, FAHA, FACC; Venu Menon, MD, FAHA, FACC; George J. Philippides, MD, FAHA, FACC; Dan M. Roden, MD, FAHA, FACC; Wojciech Zareba, MD, PhD, FACC; on behalf of the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology, the Council on Cardiovascular Nursing, and the American College of Cardiology Foundation

(*Circulation*. 2010;121:1047-1060.)



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AHA/ACC Scientific Statement

Assessment of the 12-Lead ECG as a Screening Test for Detection of Cardiovascular Disease in Healthy General Populations of Young People (12–25 Years of Age)

A Scientific Statement From the American Heart Association and the American College of Cardiology

Endorsed by the Pediatric and Congenital Electrophysiology Society and American College of Sports Medicine

Barry J. Maron, MD, FACC, Chair; Richard A. Friedman, MD, FACC, Co-Chair; Paul Kligfield, MD, FAHA; Benjamin D. Levine, MD; Sami Viskin, MD; Bernard R. Chaitman, MD, FAHA; Peter M. Okin, MD, FAHA, FACC; J. Philip Saul, MD, FAHA, FACC; Lisa Salberg; George F. Van Hare, MD; Elsayed Z. Soliman, MD, FAHA, FACC; Jersey Chen, MD, MPH; G. Paul Matherne, MD, FAHA, FACC; Steven F. Bolling, MD, FAHA; Matthew J. Mitten, JD; Arthur Caplan, PhD; Gary J. Balady, MD, FAHA; Paul D. Thompson, MD, FAHA, FACC; on behalf of the American Heart Association Council on Clinical Cardiology, Advocacy Coordinating Committee, Council on Cardiovascular Disease in the Young, Council on Cardiovascular Surgery and Anesthesia, Council on Epidemiology and Prevention, Council on Functional Genomics and Translational Biology, Council on Quality of Care and Outcomes Research, and American College of Cardiology

(*Circulation*. 2014;130:1303-1334.)

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Table 1. The 14-Element AHA Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes

Medical history*
Personal history
1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope†
3. Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing for the heart, ordered by a physician
Family history
8. Premature death (sudden and unexpected, or otherwise) before 50 y of age attributable to heart disease in ≥1 relative
9. Disability from heart disease in close relative <50 y of age
10. Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies, Marfan syndrome, or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members
Physical examination
11. Heart murmur‡
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting position)§

AHA indicates American Heart Association.

AHA/ACC Guideline

2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

(*Circulation*. 2014;130:e344-e426.)

Developed in Collaboration With the Society for Cardiovascular Angiography and Interventions and Society of Thoracic Surgeons

Endorsed by the American Association for Clinical Chemistry

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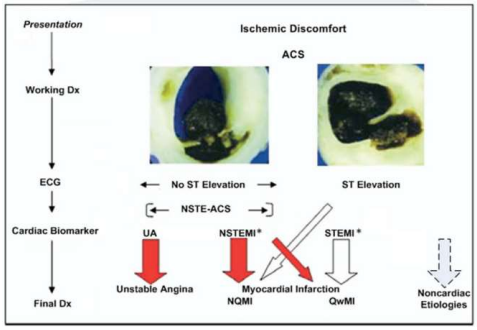

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Sudden Deaths in Young Competitive Athletes

Analysis of 1866 Deaths in the United States, 1980–2006

Barry J. Maron, MD; Joseph J. Doerer, BS; Tammy S. Haas, RN; David M. Tierney, MD; Frederick O. Mueller, PhD

(*Circulation*. 2009;119:1085-1092.)

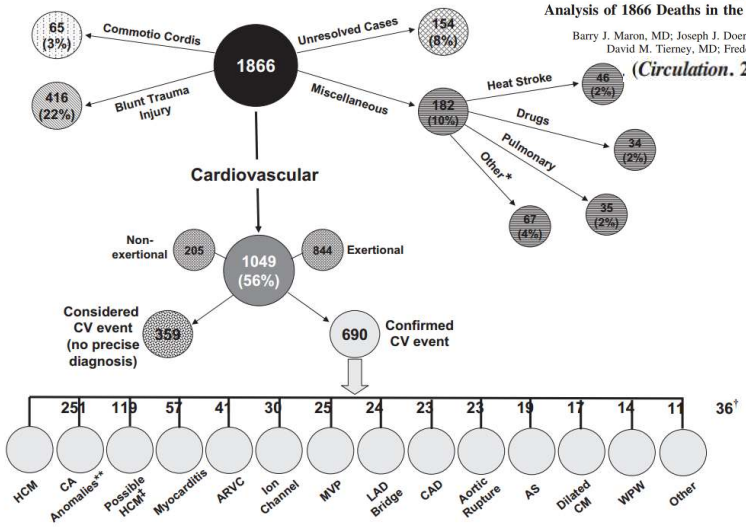

Figure 2. Flow diagram summarizing causes of death in 1866 young competitive athletes. *Suicide (n=22); lightning (n=12); drowning (n=10 and 3 during the swimming segment of triathlon events); cerebral aneurysm (n=9); rhabdomyolysis (n=8); epilepsy (n=2); and miscellaneous (n=4). †Congenital heart disease (n=8); myocardial infarction (n=6); Kawasaki disease or related conditions (n=5); sickle cell trait (n=5); sarcoidosis (n=4); stroke (n=3); cardiac tumor (n=1); conduction system disease (n=2); and miscellaneous (n=2). ‡Regarded as possible (not definitive) evidence for hypertrophic cardiomyopathy at autopsy with mildly increased left ventricular wall thickness (18±4 mm) and heart weight (447±76 g). **Of wrong sinus origin coursing between aorta and pulmonary trunk; most commonly, anomalous left main coronary artery from right (anterior) sinus of Valsalva (n=65) and anomalous right coronary artery from the left sinus (n=[illegible]). ARVC indicates arrhythmogenic right ventricular cardiomyopathy; AS, aortic stenosis; CA, coronary artery; CAD, coronary artery disease; CM, cardiomyopathy; CV, cardiovascular; HCM, hypertrophic cardiomyopathy; LAD, left anterior descending coronary artery; MVP, mitral valve prolapse; and WPW, Wolff-Parkinson-White.

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2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC)

Borja Ibanez, Stefan James, Stefan Agewall, Manuel J Antunes, Chiara Bucciarelli-Ducci, Héctor Bueno, Alida L P Caforio, Filippo Crea, John A Goudevenos, Sigrun Halvorsen ... Show more

European Heart Journal, Volume 39, Issue 2, 07 January 2018, Pages 119–177,
https://doi.org/10.1093/eurheartj/ehx393
Published: 26 August 2017

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Incidence, Cause, and Comparative Frequency of Sudden Cardiac Death in National Collegiate Athletic Association Athletes

A Decade in Review

Kimberly G. Harmon, MD; Irfan M. Asif, MD; Joseph J. Maleszewski, MD; David S. Owens, MD, MS; Jordan M. Prutkin, MD, MHS; Jack C. Salerno, MD; Monica L. Zigman, MPH; Rachel Ellenbogen, MS; Ashwin L. Rao, MD; Michael J. Ackerman, MD, PhD; Jonathan A. Drezner, MD

(*Circulation*. 2015;132:10-19.

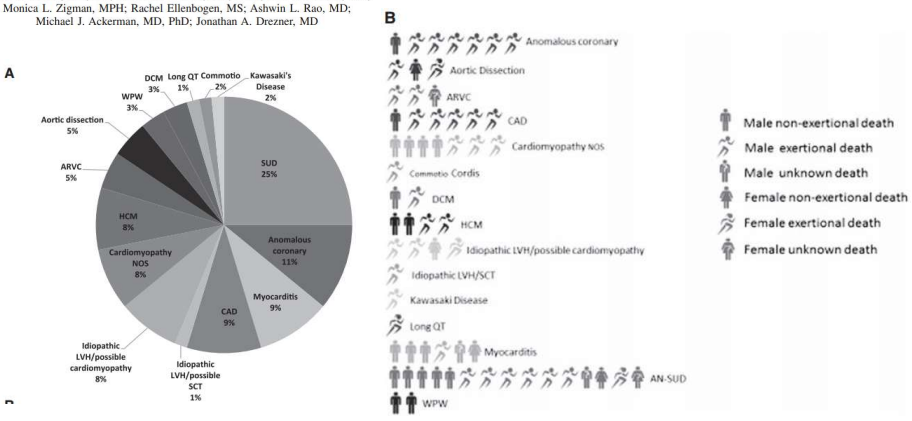

Figure 2. A, Causes of sudden cardiac death in athletes. **B**, Cause and activity at time of death. One person figure equals 1 death; female figures follow male figures, unless no male deaths were present. AN-SUD indicates autopsy-negative sudden unexplained death; ARVC, arrhythmogenic right ventricular cardiomyopathy; CAD, coronary artery disease; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; LVH, left ventricular hypertrophy; NOS, not otherwise specified; SCT, sickle cell trait; SUD, sudden unexplained death; and WPW, Wolff-Parkinson-White.

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Journal of the American College of Cardiology
Volume 72, Issue 14, October 2018
DOI: 10.1016/j.jacc.2017.10.053

PDF Article

CLINICAL PRACTICE GUIDELINE: EXECUTIVE SUMMARY

2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society

Sana M. Al-Khatib, William G. Stevenson, Michael J. Ackerman, William J. Bryant, David J. Callans, Anne B. Curtis, Barbara J. Deal, Timm Dickfeld, Michael E. Field, Gregg C. Fonarow, Anne M. Gillis, Christopher B. Granger, Stephen C. Hammill, Mark A. Hlatky, José A. Joglar, G. Neal Kay, Daniel D. Matlock, Robert J. Myerburg and Richard L. Page

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Journal of the American College of Cardiology
Volume 72, Issue 14, October 2018
DOI: 10.1016/j.jacc.2017.10.053

Table 3 Definitions of commonly used terms

Term	Definition	Ref[a]
Sudden death	Non-traumatic, unexpected fatal event occurring within 1 hour of the onset of symptoms in an apparently healthy subject. If death is not witnessed, the definition applies when the victim was in good health 24 hours before the event.	1
SUDS and SUDI	Sudden death without an apparent cause and in which an autopsy has not been performed in an adult (SUDS) or in an infant <1 year of age (SUDI).	14
SCD	The term is used when: • A congenital, or acquired, potentially fatal cardiac condition was known to be present during life; OR • Autopsy has identified a cardiac or vascular anomaly as the probable cause of the event; OR • No obvious extra-cardiac causes have been identified by post-mortem examination and therefore an arrhythmic event is a likely cause of death.	1, 14, 15
SADS and SIDS	Both autopsy and toxicology investigations are inconclusive, the heart is structurally normal at gross and histological examination and non-cardiac aetiologies are excluded in adults (SADS) and in infants (SIDS).	16
Aborted cardiac arrest	Unexpected circulatory arrest, occurring within 1 hour of onset of acute symptoms, which is reversed by successful resuscitation manoeuvres (e.g. defibrillation).	-
Idiopathic ventricular fibrillation	Clinical investigations are negative in a patient surviving an episode of ventricular fibrillation.	17, 18
Primary prevention of SCD	Therapies to reduce the risk of SCD in individuals who are at risk of SCD but have not yet experienced an aborted cardiac arrest or life-threatening arrhythmias.	-
Secondary prevention of SCD	Therapies to reduce the risk of SCD in patients who have already experienced an aborted cardiac arrest or life-threatening arrhythmias.	1

SADS = sudden arrhythmic death syndrome; SCD = sudden cardiac death; SIDS = sudden infant death syndrome; SUDI = sudden unexplained death in infancy; SUDS = sudden unexplained death syndrome.
[a]References

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Journal of the American College of Cardiology
Volume 72, Issue 14, October 2018
DOI: 10.1016/j.jacc.2017.10.053

Table 4 Diagnostic approach for family members of sudden unexplained death syndrome or sudden arrhythmic death syndrome victims

Approach	Action[a]
History taking and physical examination	• Personal clinical history • Family history focused on cardiac diseases or sudden deaths
ECG	• Baseline 12-lead ECG with standard and high precordial leads • 24-hour ambulatory ECG • Exercise stress test • Signal-averaged ECG • Provocative test with ajmaline/flecainide (when Brugada syndrome is suspected)
Cardiac imaging	• Two-dimensional echocardiography and/or CMR (with or without contrast)
Genetic testing	• Targeted molecular testing and genetic counselling if there is the clinical suspicion of a specific disease • Referral to a tertiary centre specialized in evaluation of the genetics of arrhythmias

CMR = cardiac magnetic resonance; ECG = electrocardiogram.
[a]The recommendations in this table are based on the consensus of this panel of experts and not on evidence-based data.

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The official 2015 AHA Guidelines Update for CPR & ECC are now available!

These guidelines are based on the most current and comprehensive review of resuscitation science, systems, protocols, and education.

2015 Update in *Circulation*

View the new 2015 AHA Guidelines Update for CPR & ECC in the *Circulation* journal.

VIEW GUIDELINES UPDATE

NEW Web-Based Integrated Guidelines

This site blends the 2015 and 2010 AHA Guidelines for CPR & ECC into a new online interface. Explore and search all guidelines from your desktop or mobile device.

VIEW INTEGRATED GUIDELINES

ASPECTOS DESTACADOS

de la actualización de las Guías de la AHA para RCP y ACE de 2015

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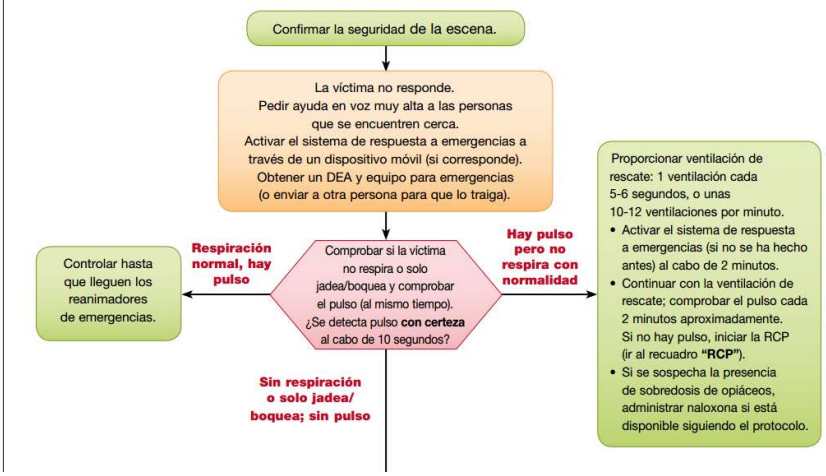
Figura 5

Algoritmo de paro cardíaco en adultos para profesionales de la salud que proporcionan SVB/BLS: actualización de 2015



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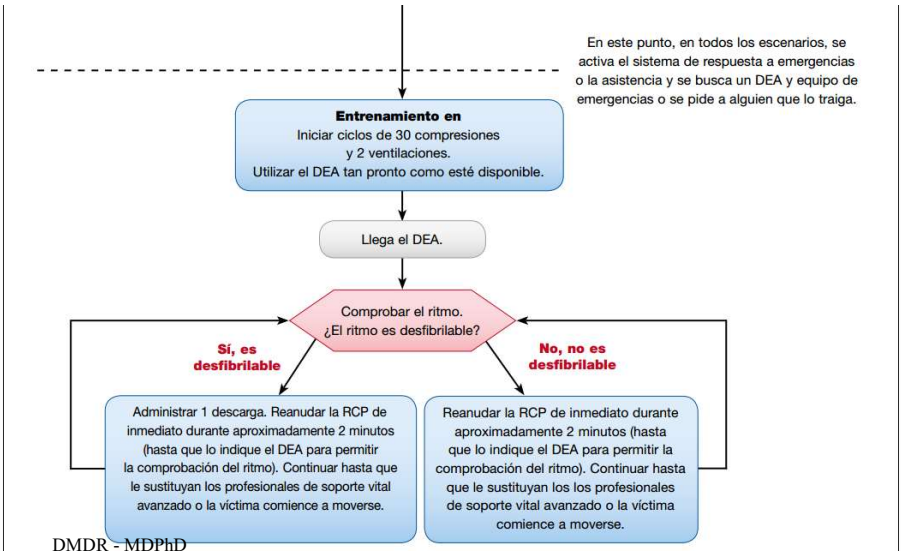

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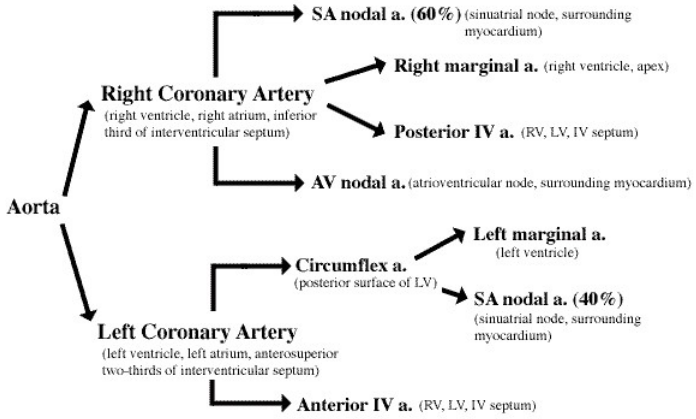

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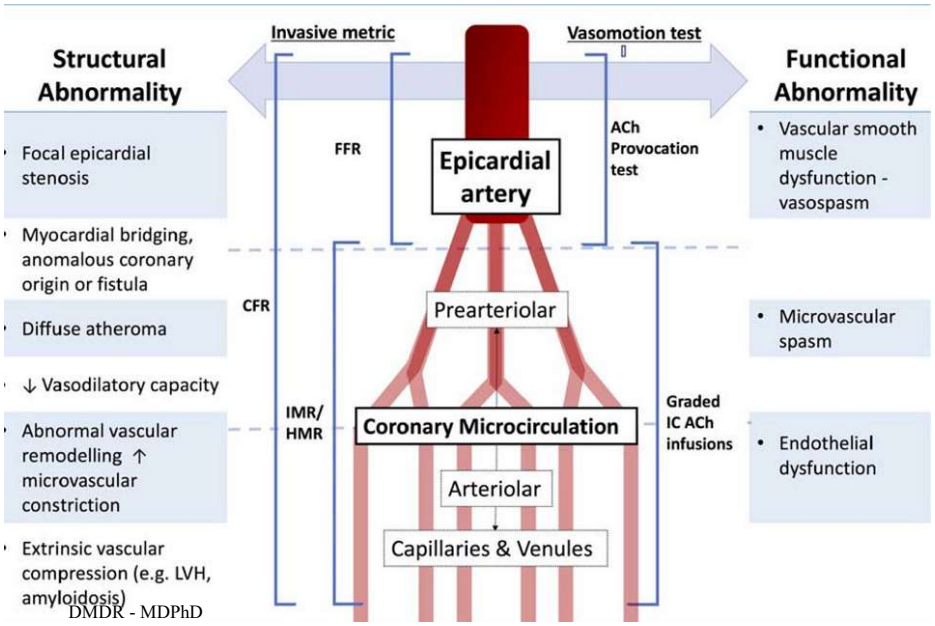

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Ciclo Cardiaco

60x' (60seg/60lat) 1000
Ciclo cardiaco = 1000mseg.
Sístole 400mseg
Diástole 600mseg

100x´ (60seg/100lat) 1000
Ciclo cardiaco = 600mseg.
Sístole 300mseg
Diástole 300mseg

140x´ (60seg/140lat) 1000
Ciclo cardiaco = 428mseg.
Sístole 256mseg
Diástole 173mseg

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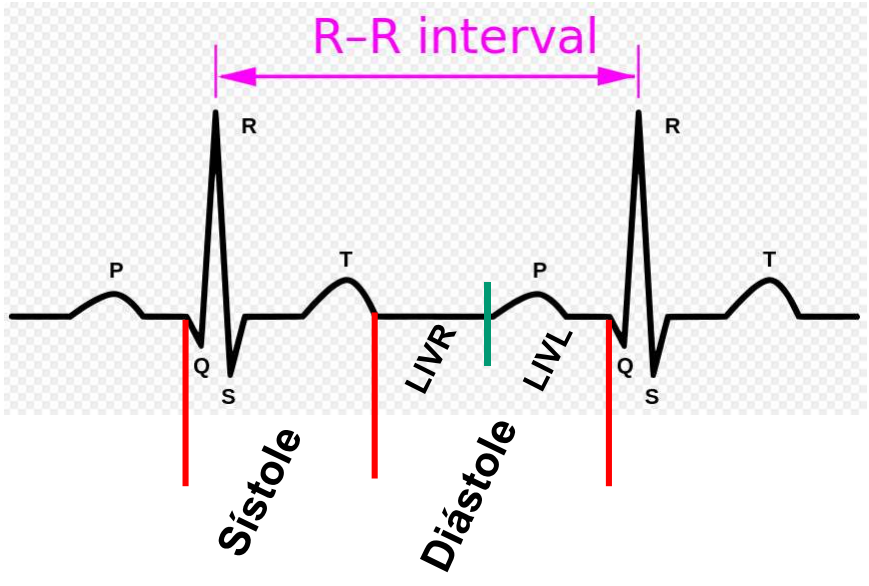

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FUNCIÓN VENTRICULAR Y MECANISMOS DE ADAPTACIÓN

- Precarga.
- Postcarga.
- Contractilidad.
- Frecuencia Cardiaca.
- Interdependencia Biventricular y Pulmonar (Ventilatoria).

FUNCION CARDIACA

Inotrópico: Influencia de la fuerza muscular sobre la contracción miocárdica.
Lusitrópico: Influencia del efecto de la relajación miocárdica.
Cronotrópico: Influencia del evento periódico de la contracción miocárdica.
Batmotrópico: Influencia del nivel de umbral de excitación.
Dromotrópico: Influencia de la velocidad de conducción de los estímulos eléctricos.

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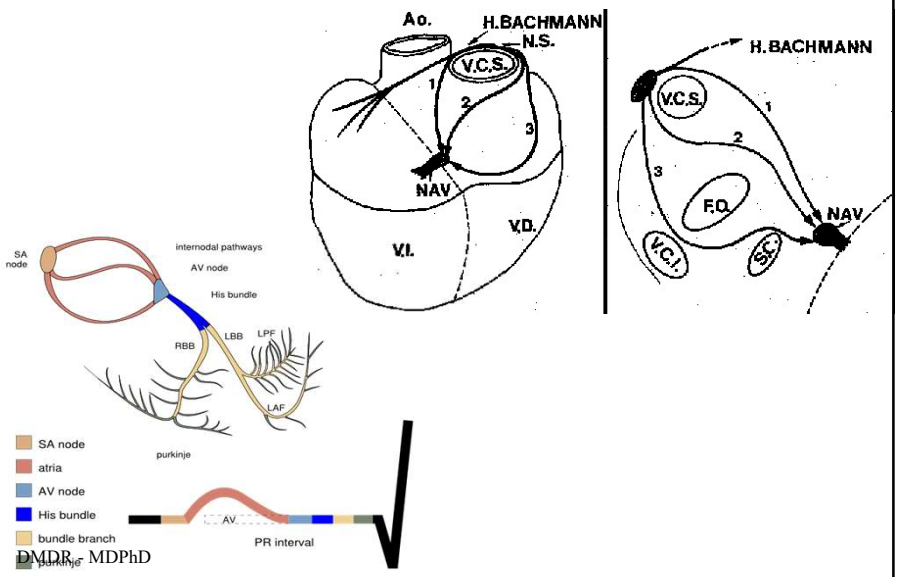
Fibras de Conducción Auriculares



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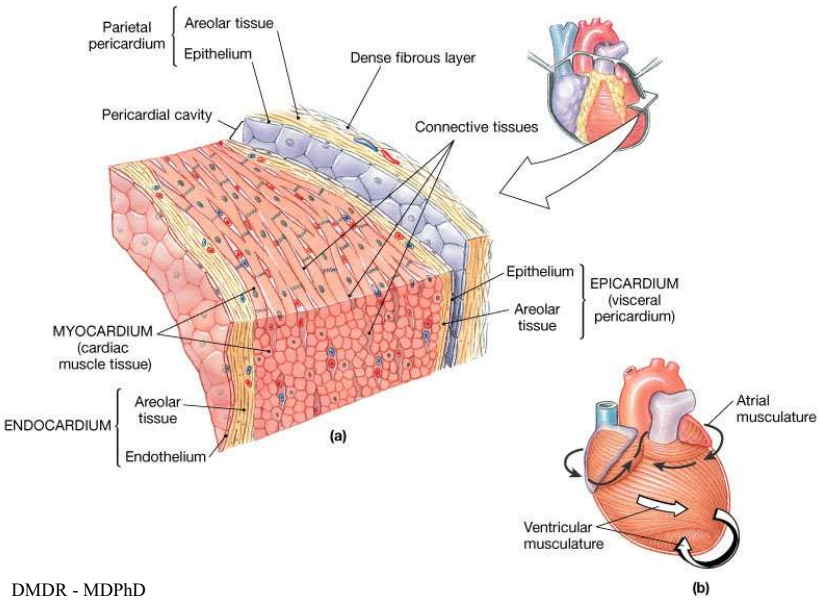

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Normal activation sequence		Time (ms)	ECG	Conduction velocity ($m \cdot s^{-1}$)	Intrinsic rate (min^{-1})
SA node					
Impulse generation		0	P wave	0.05	60–100
Arrival of impulse in distal parts of atrium	Right atrium	50		0.8–1.0 in atrium	
	Left atrium	85			
AV node			P-Q segment (delayed conduction)		40–55
Arrival of impulse		50		0.05	
Relaying of impulse		125			
His bundle activated		130		1.0–1.5	25–40
End of bundle branches activated		145		1.0–1.5	
Purkinje fibers activatedt		150		3.0–3.5	
Inner myocardium completely activated	Right ventricle	175	QRS complex	1.0 in myocardium	None
	Left ventricle	190			
Outer myocardium completely activated	Right ventricle	205			
	Left ventricle	225			

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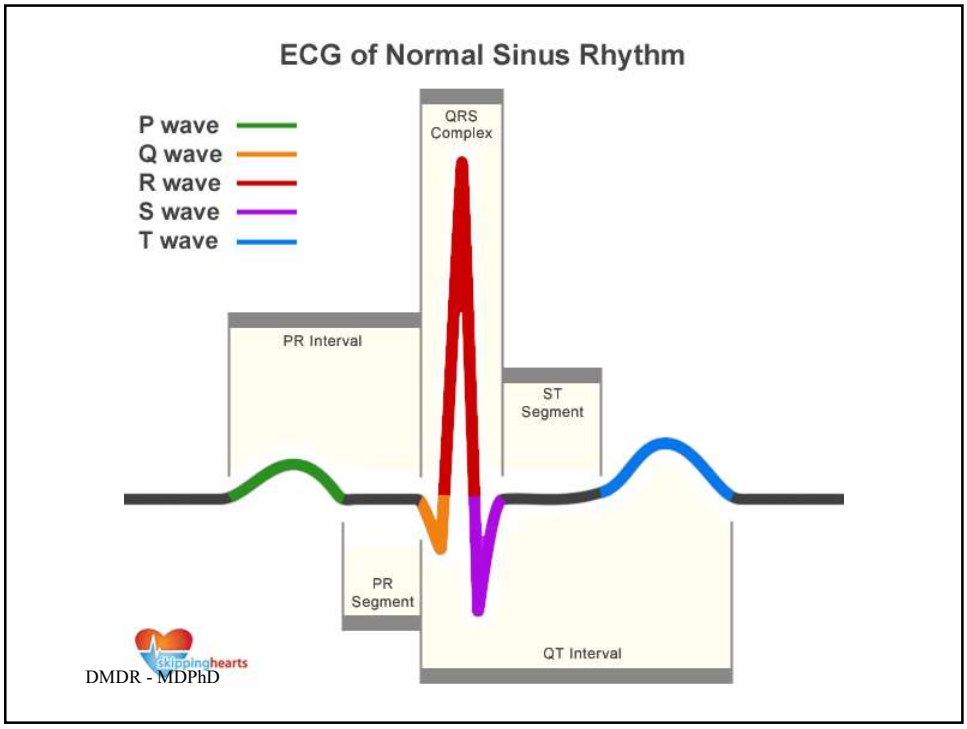

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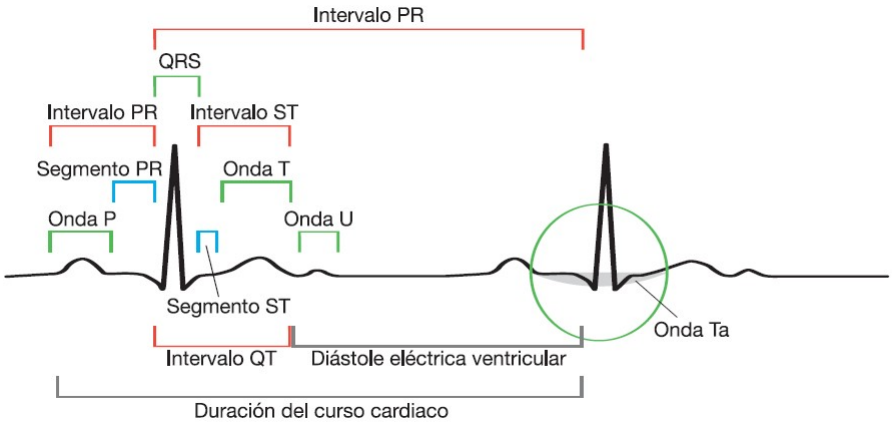

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Descartes and the ECG Lettering Series

JAMES ROBERT HENSON

Journal of the History of Medicine : April 1971

DESCARTES published his influential *La geometrie* in 1637 and became the first mathematician to adopt letters exclusively to represent numbers and points in calculations and mathematical diagrams.[1] He invented the cartesian coordinate system seen today as the familiar two-dimensional graph. He also investigated the geometry of optics. For example, he used the letter series A–B–C, G–H–I, K–L–M–N, and P–Q–R–S–T to illustrate his diagram on light refraction (Fig. 1). In other illustrations he used the letter O to designate the origin or center of a circle and often followed with the letter series P–Q–R . . to represent points on the perimeter of the curve. Most often Descartes used the series A–B–C . . to note known points or values in a diagram or calculation and used the series X–Y–Z to represent unknown values. Today we usually represent an unknown value in mathematics by calling it 'unknown x.'

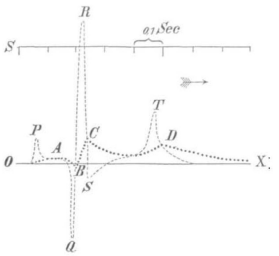

FIG. 7. From W. Einthoven, 'Ueber die Form des menschlichen Electrocardiogramms,' *Pflüg. Arch. ges. Physiol.*, 1895, 60, 101–123 (fig. 2, p. 105).

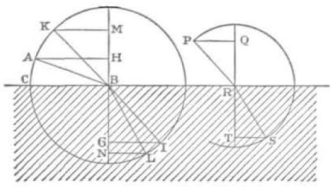

FIG. 1. Redrawn from René Descartes, *Discours de la methode . . .* and *La dioptrique . . .* (Leiden, 1637), p. 21.

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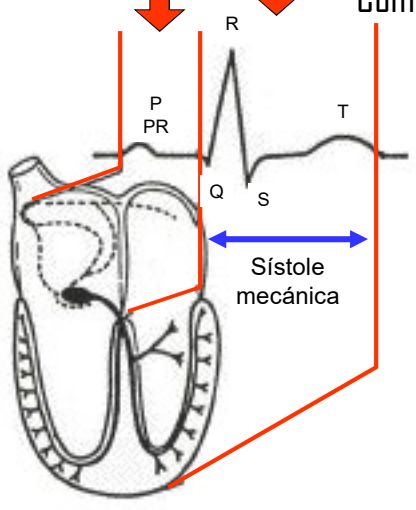
Activación auricular
Onda P
Intervalo PR

Activación
Ventricular
Complejo QRS



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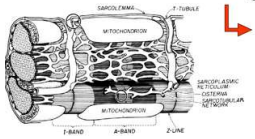
PROPIEDADES ELÉCTRICAS DEL MIOCARDIO

I. Automatismo
II. Excitabilidad
III. Conductibilidad

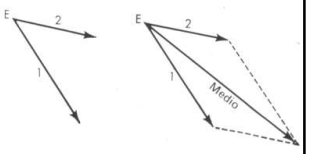


Energía bioquímica
↳ Energía eléctrica
↳ Energía mecánica

VECTORES (Del lat. *vector*, que conduce)

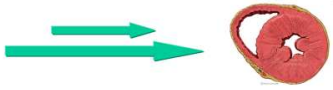
PROPIEDADES

I. Polaridad
II. Magnitud
III. Dirección



VECTORES

Magnitud



Polaridad

Dirección

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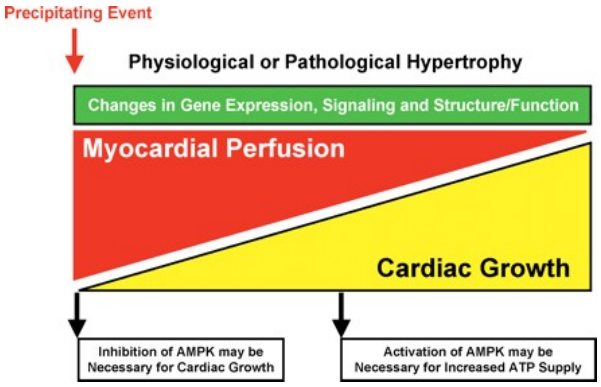
AMPK alterations in cardiac physiology and pathology: enemy or ally?



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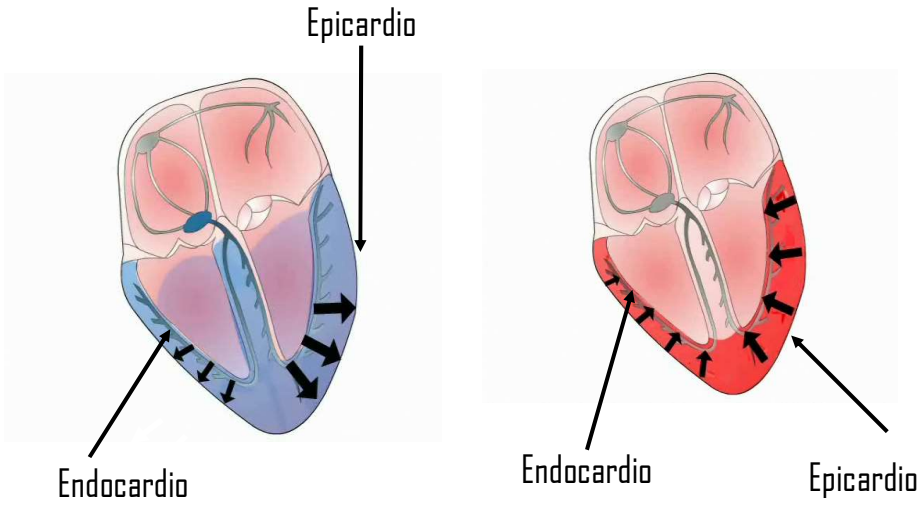
J Physiol 2006;574:95-112

Despolarización:
Subendo a Subepi

Repolarización:
Subepi a Subendo



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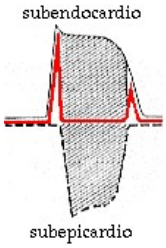

Podemos definir dos zonas desde un punto de vista eléctrico: el subepicardio y el subendocardio. Ambas están separadas por lo que se denomina endocardio eléctrico.

La zona subendocárdica es la primera que se despolariza y la última que se repolariza, y de esta manera el PAT del subendocardio se inicia antes y finaliza más tarde que el PAT del subepicardio.

El ECG de superficie es la resultante de las dos curvas.

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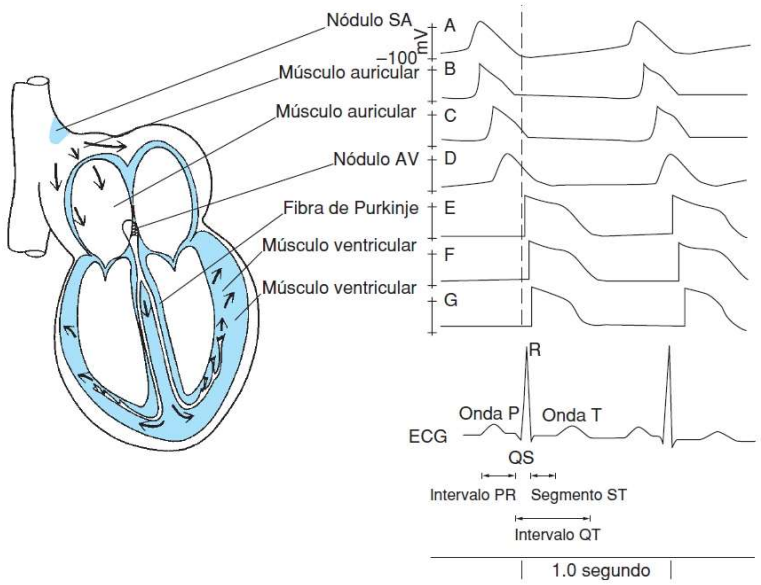

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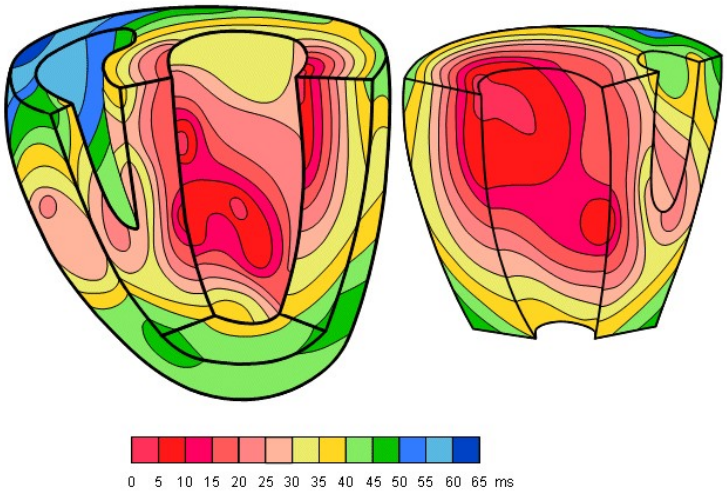
Isochronic surfaces of the ventricular activation



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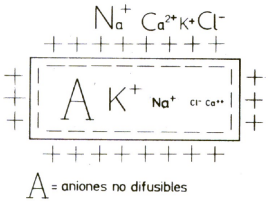

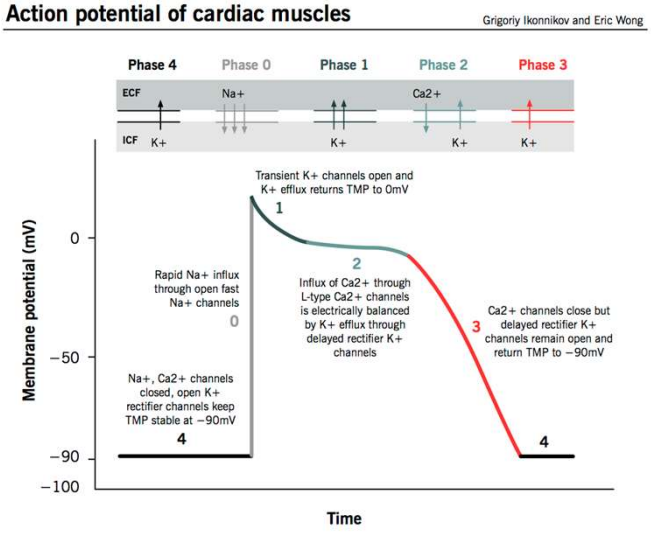

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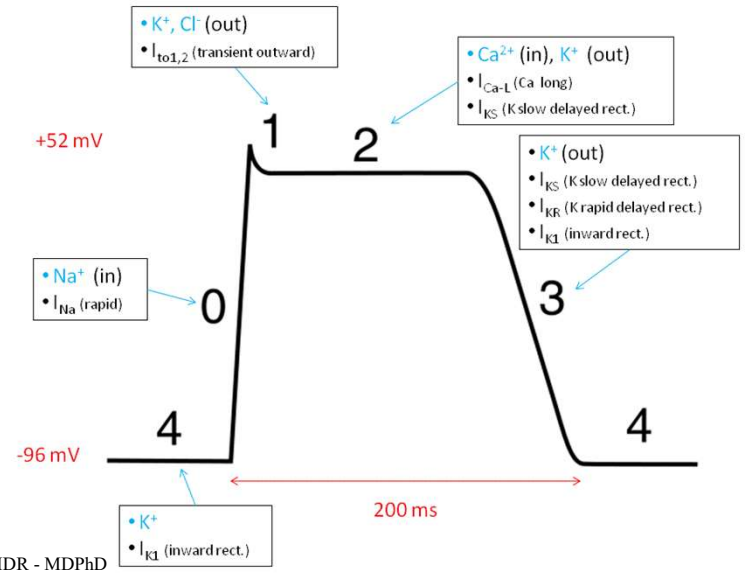

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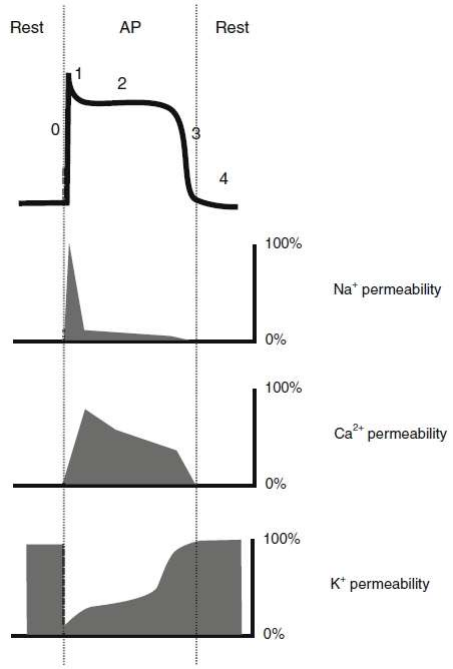

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POTENCIAL DE ACCION TRANSMEMBRANA FASES

- FASE O:

Entrada de Na^+ y Ca^{++} al espacio intracelular.
< permeabilidad al K^+.
Complejo QRS (-90mv a +20mv).

- FASE I:

Inicia repolarización de la membrana celular. Cierre de los canales rápidos de Na^+ (+20mv a 0mv).
Sale poco K^+ e ingresa Na^+ y Ca^{++} (canales lentos) Punto J.

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POTENCIAL DE ACCION TRANSMEMBRANA FASES

FASE II:
Repolarización lenta o meseta. Ingreso de Ca^{++} y Na^{+} y salida de K^{+}. Al equilibrarse las cargas se inscribe el segmento ST.
("equilibrio eléctrico")
Comienza a salir K^{+} para compensar la positividad perdida por la entrada de Na^{+} y Ca^{++} hacia el espacio intracelular.

FASE III:
Se inhibe la entrada de Na^{+} y Ca^{++}. >> salida de K^{+} 0mv (Onda T).
La célula ha ganada Na^{+} y Ca^{++} y perdido K^{+}

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POTENCIAL DE ACCION TRANSMEMBRANA FASES

FASE IV:
El desequilibrio iónico se corrige por medio de un mecanismo activo (Bomba Iónica).
Ingresa K^{+} y sale Na^{+} y Ca^{++} por acción de la hidrolización del ATP.
Consumo de energía: Inscripción Onda U.

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PERIODOS REFRACTARIOS

Todas las células miocárdicas después de haberse excitado tardan un cierto tiempo en recuperar su excitabilidad (fase o periodo refractario), en condiciones normales.

Absoluto (PRA): Ningún estímulo sin importar su fuerza, puede provocar otro estímulo (ocupa gran parte de la sístole).
Efectivo (PRE): Respuestas locales: Muy breve, la célula forma potenciales locales que no son, sin embargo capaces de propagarse (a). Respuestas locales supra-umbrales (b).

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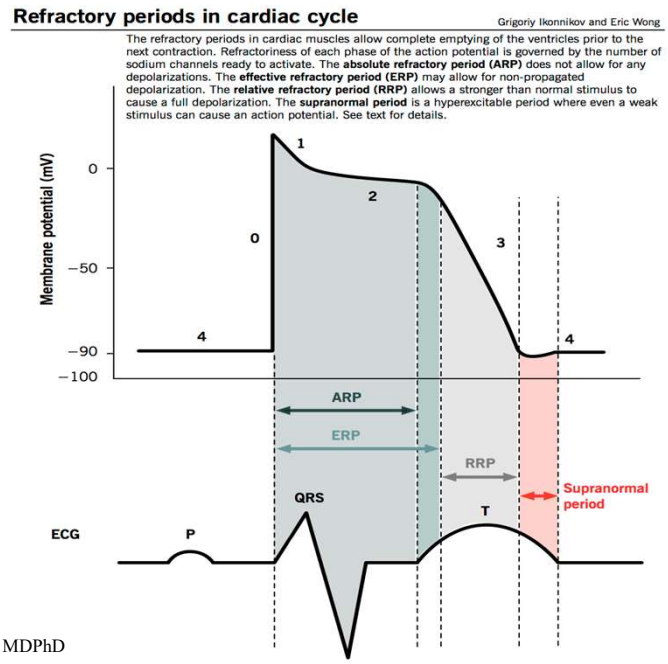

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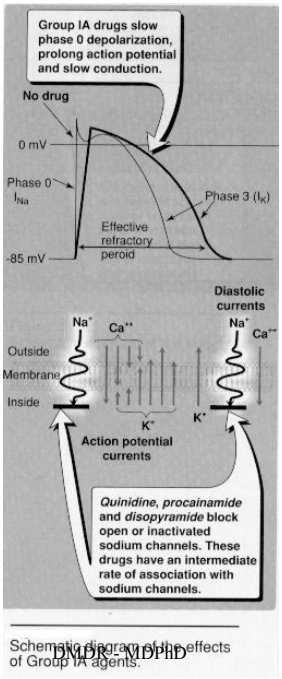
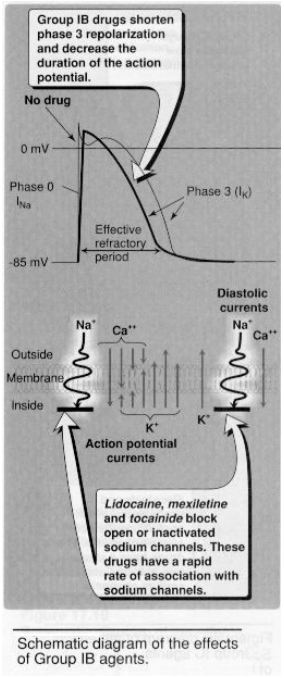
CLASIFICACIÓN DE LOS FÁRMACOS ANTIARRÍTMICOS

Vaughan Williams EM. Clasification of antiarrhytmical drugs. Symposium on Cardiac Arrhytmias. Elsimore, Denmark. Sandoe E., Flensted-Jensen E, Olsen KH. Ed. Asta. Sweden, 1970: 449-472.

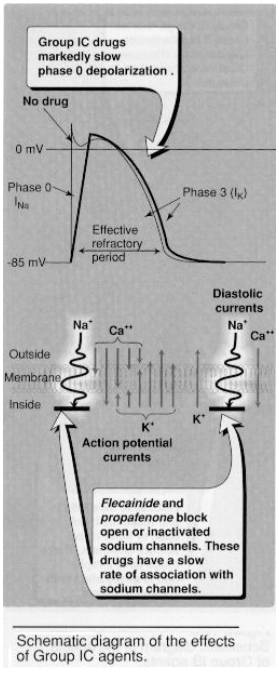
CLASE	ACCIONES	FÁRMACOS
IA Bloqueantes de los canales del Na.	Prolongan la repolarización. Anticolinérgicos. Cinética intermedia. Prolongan la duración del potencial de acción.	Procainamida Disopiramida Quinidina
IB Bloqueantes de los canales del Na.	Cinética rápida. Reducen o acortan el potencial de acción.	Lidocaina Mexiletina Tocainida Morizacina
IC Bloqueantes de los canales del Na	Cinética lenta. Prolongan ligeramente el potencial de acción.	Propafenona Flecainida Encainida
II Betabloqueantes.		Propranolol Metoprolol Nadolol Atenolol Sotalol
III Bloqueantes de los canales del K.	Prolongan la repolarización. Antiadrenérgicos. Prolongan la repolarización	Bretilio Amiodarona Sotalol Azimilida
IV Bloqueantes de los canales del Ca.		Verapamilo Diltiazem Bepridil



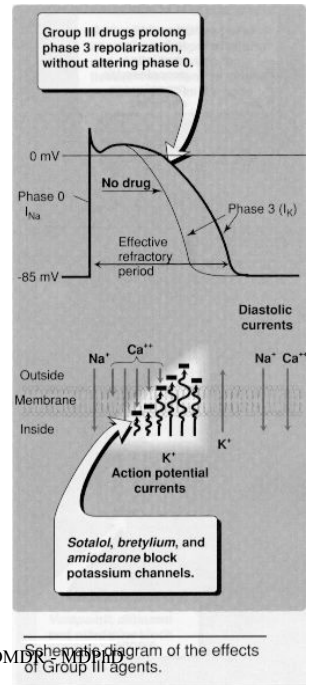
Schematic diagram of the effects of Group IA agents.



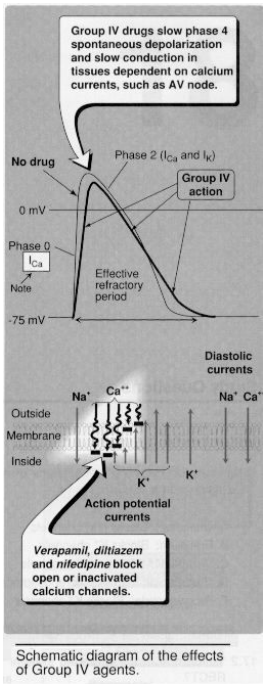
Schematic diagram of the effects of Group IB agents.



Schematic diagram of the effects of Group IC agents.



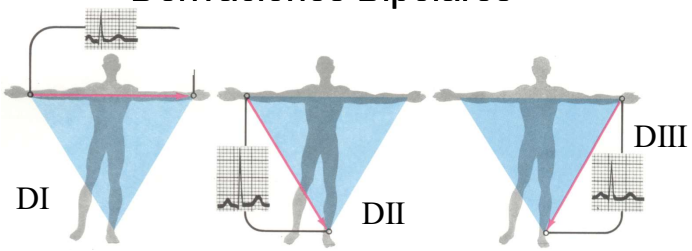
Schematic diagram of the effects of Group III agents.



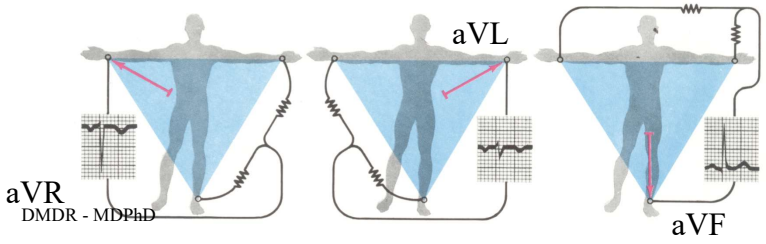
Schematic diagram of the effects of Group IV agents.

Triángulo de Einthoven y Sistema Hexaxial de Bailey Derivaciones y Correlación ECG-Anatómica

Triángulo de Willem Einthoven
Derivaciones Bipolares



Triángulo de Einthoven
Derivaciones Unipolares



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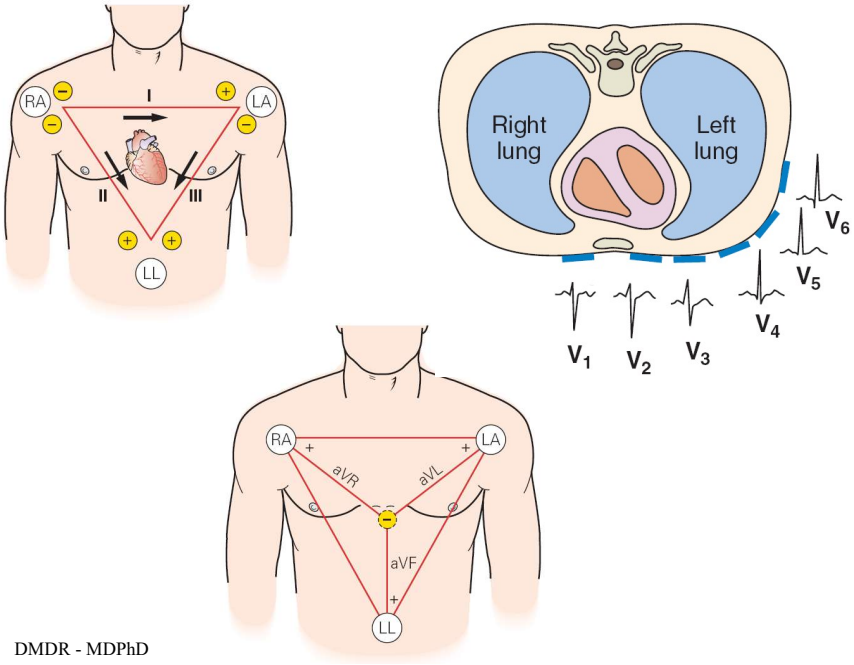

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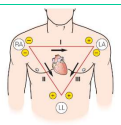
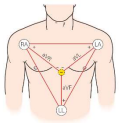
Un ECG normal está compuesto por doce derivaciones diferentes.
Se dividen en tres grupos:



I.- Derivaciones Bipolares de las Extremidades:
Registran la diferencia de potencial eléctrico entre dos puntos:



Derivación I: entre brazo izquierdo (+) y brazo derecho (-).
Derivación II: entre pierna izquierda (+) y brazo derecho (-).
Derivación III: entre pierna izquierda (+) y brazo izquierdo (-).

II.- Derivaciones Monopolares de los Miembros:
Registran las variaciones de potencial de un punto con respecto a otro que se considera con actividad eléctrica 0. Se denominan aVR, aVL y aVF, por:

a: significa aumento y se obtiene al eliminar el electrodo negativo dentro del propio aparato de registro.
V: Vector.
R (right), **L** (left) y **F** (foot): según el lugar donde se coloque el electrodo positivo, brazo derecho, brazo izquierdo o pierna izquierda.

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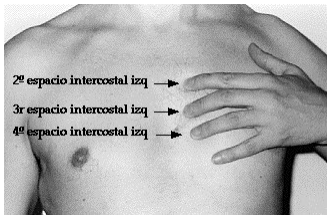
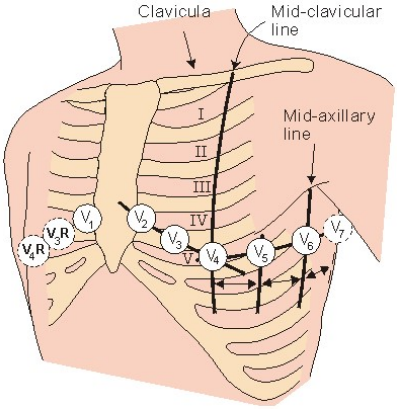
Un ECG normal está compuesto por doce derivaciones diferentes.
Se dividen en tres grupos:





III.- Derivaciones precordiales (de Wilson):

El electrodo se coloca en:
V1: 4º espacio intercostal derecho, línea paraesternal derecha.
V2: 4º espacio intercostal izquierdo, línea paraesternal izquierda.
V3: simétrico entre V2 y V4.
V4: 5º espacio intercostal izquierdo, línea medioclavicular.
V5: 5º espacio intercostal izquierdo, línea anterior axilar.
V6: 5º espacio intercostal izquierdo, línea axilar media.

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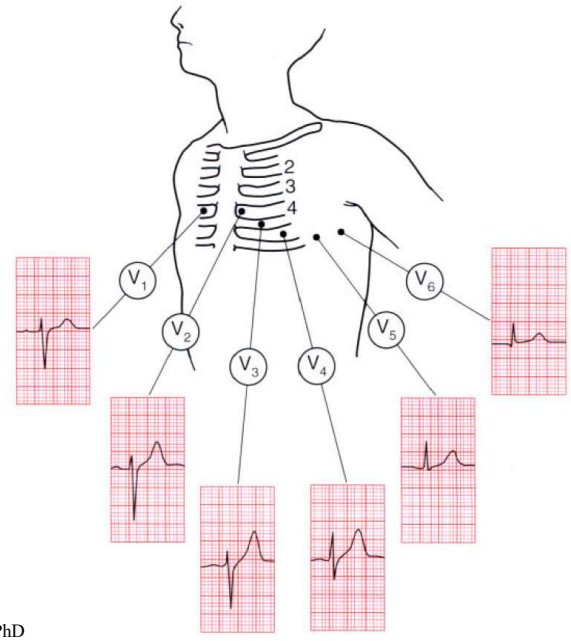

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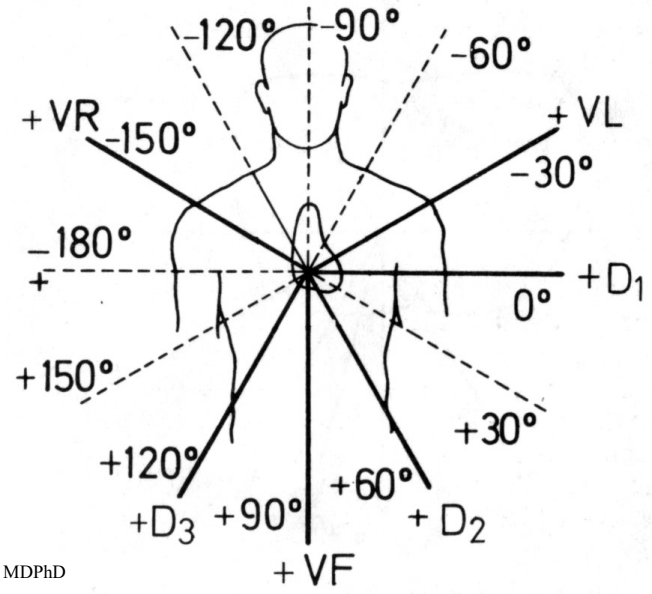
Sistema hexaxial de Bailey



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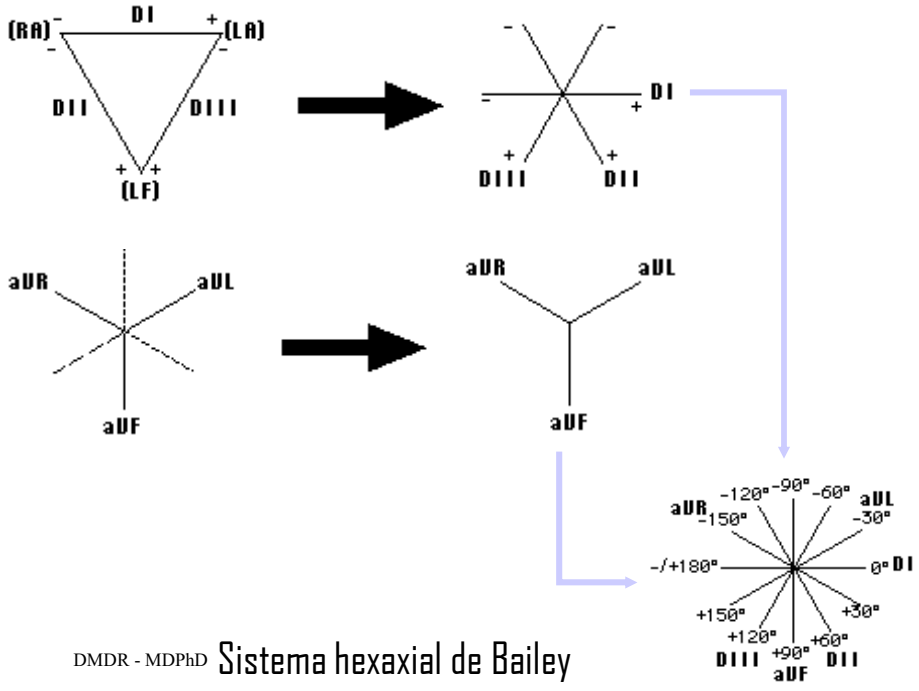
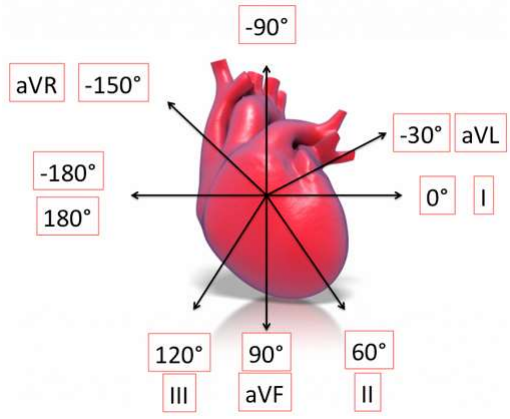


DMDR - MDPhD Sistema hexaxial de Bailey



Cálculo del Eje Cardiaco

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Secuencia de Activación Ventricular

http://www.bem.fi/book/00/co.htm

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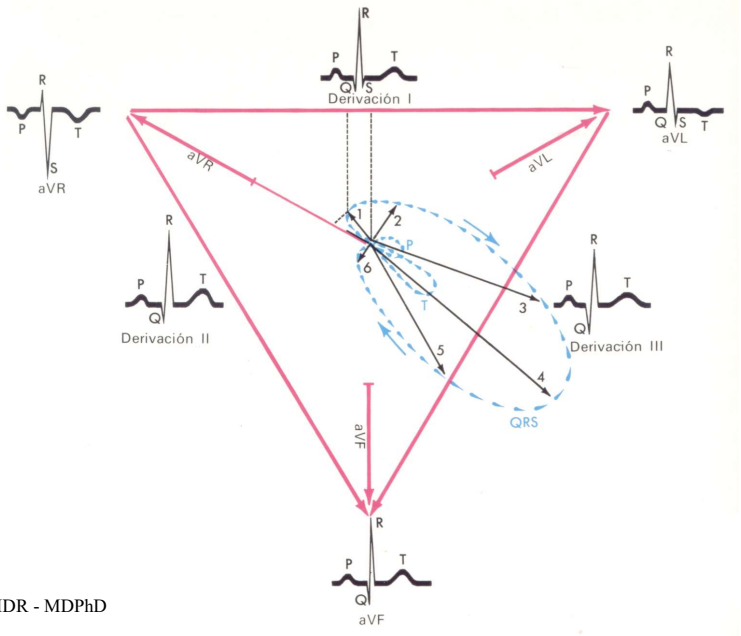
QRS – Plano Frontal



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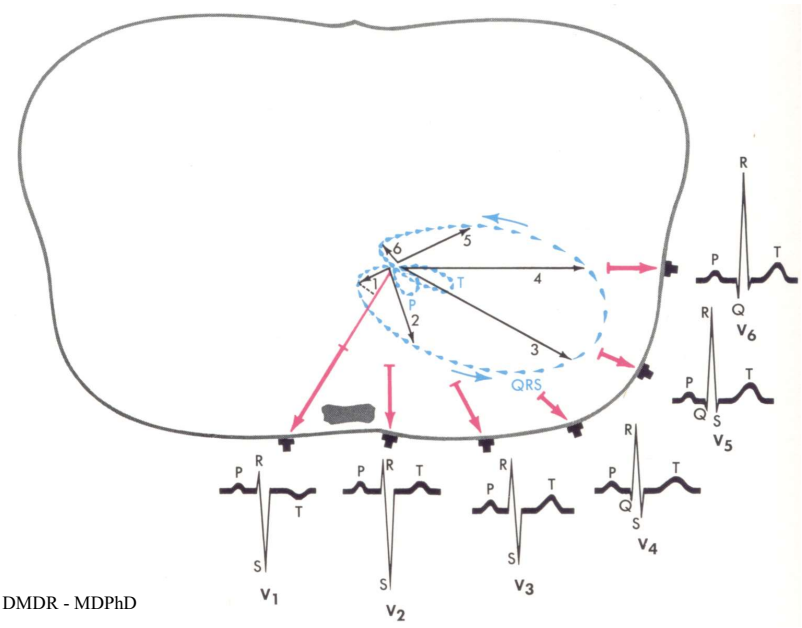
QRS – Plano Horizontal



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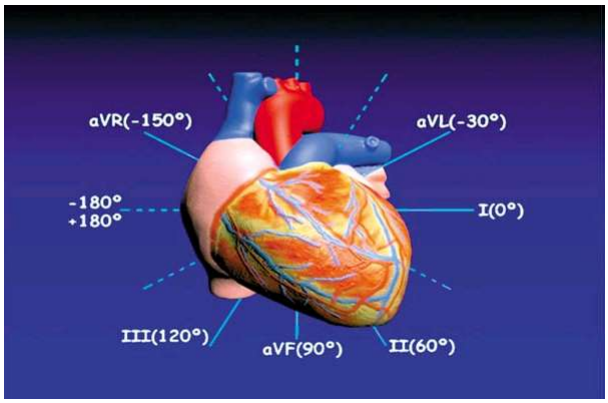

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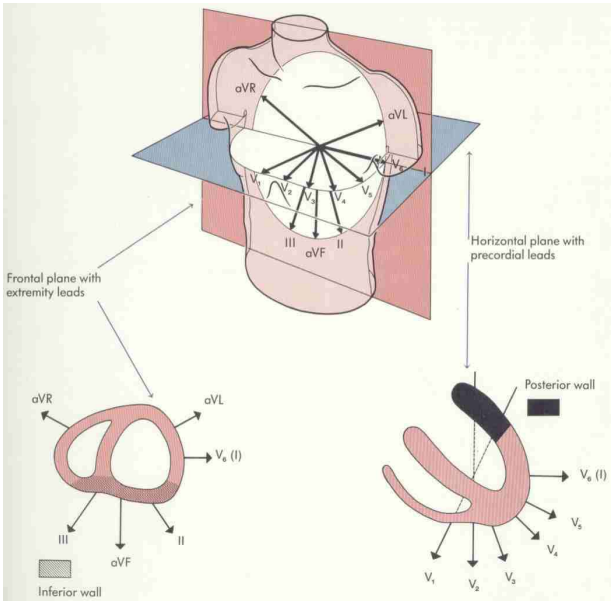

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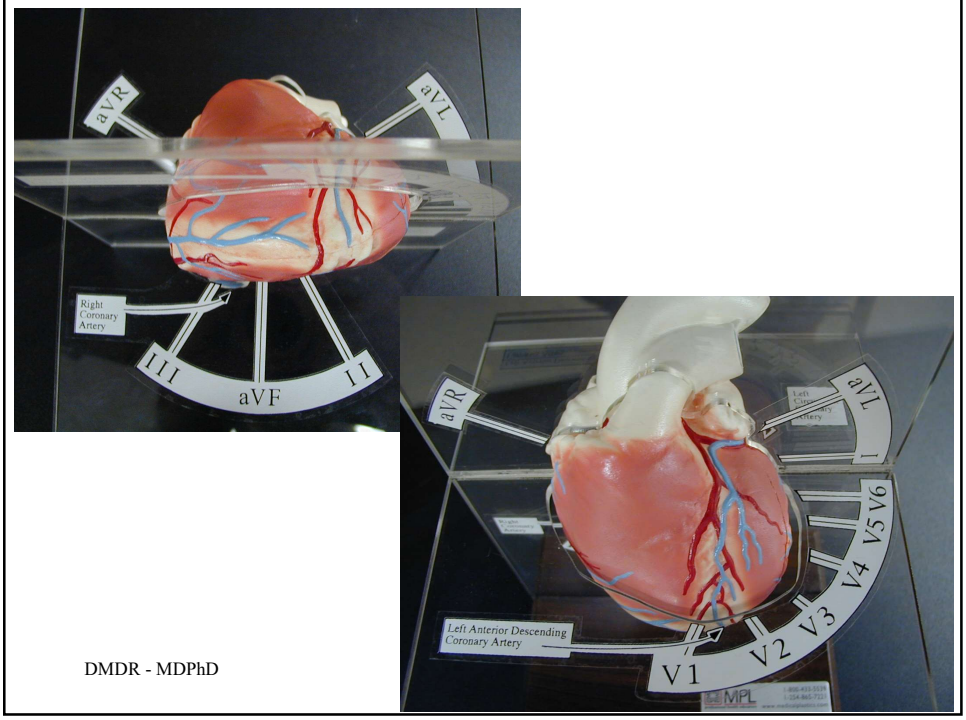

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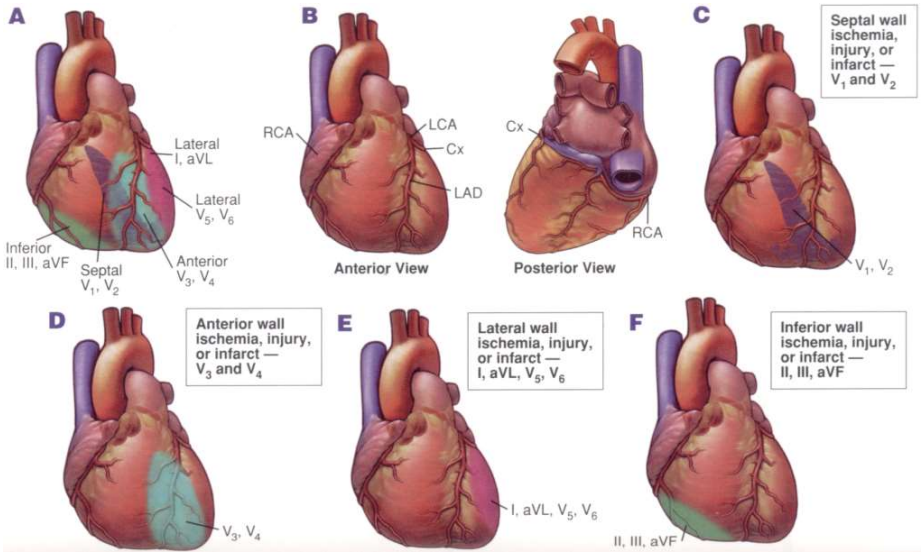

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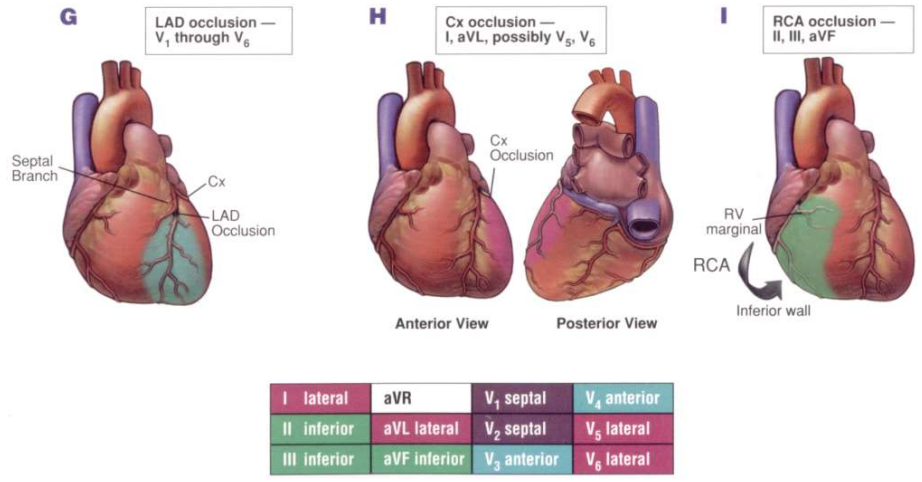

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Rutina de Interpretación

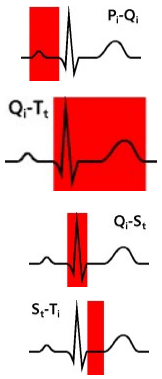
Secuencia de Activación Miocárdica

i. Nodo sinusal (sinoauricular)
ii. Miocardio auricular
iii. Nodo auriculoventricular
iv. Haz de His
v. Fibras de Purkinje
vi. Miocardio ventricular

600mseg.

Rutina de Interpretación

i. Ritmo
ii. Frecuencia cardiaca
iii. Intervalo PR
iv. Eje eléctrico (plano frontal)
v. Intervalo QT
vi. Onda P
vii. Complejo QRS
viii. Segmento ST
ix. Onda T
x. Onda U



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Variantes a considerar

- Hábito constitucional
- Malformaciones torácicas
- Raza, edad, sexo, etc.
- Hiperventilación
- Hipotermia
- Taquicardia
- Ingesta de alcohol
- Alteraciones iónicas
- Acción de fármacos
- Artefactos

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Ritmo Sinusal

- **Ondas P + en DII y aVF y - en aVR**
- **Cada onda P va seguida de un QRS**
- **El intervalo PR es constante (120 a 200mseg)**
- **Intervalos RR regulares ¿?. (arritmia respiratoria)**

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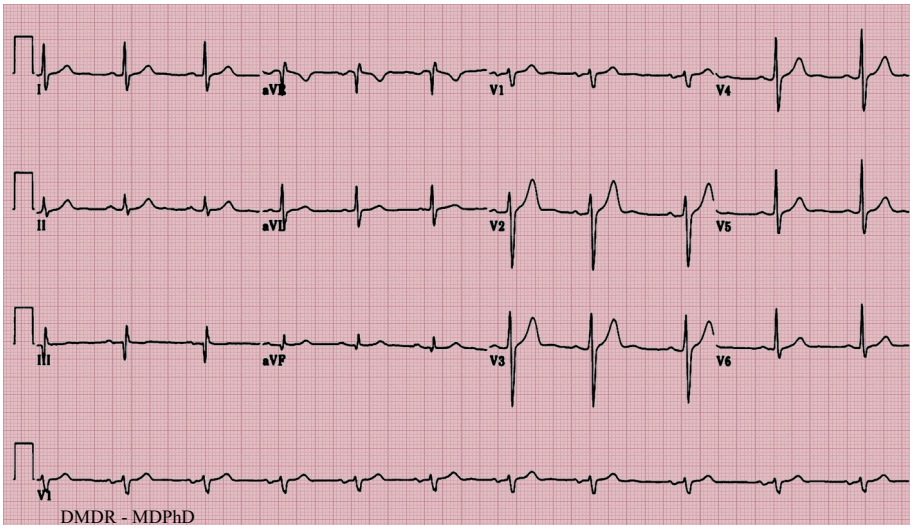
Ritmo Sinusal



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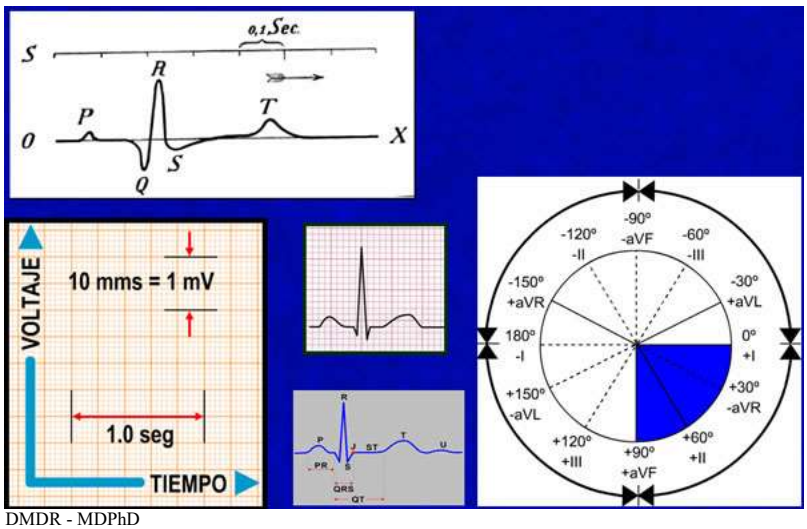

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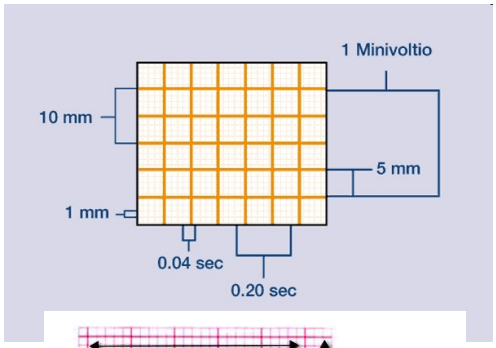

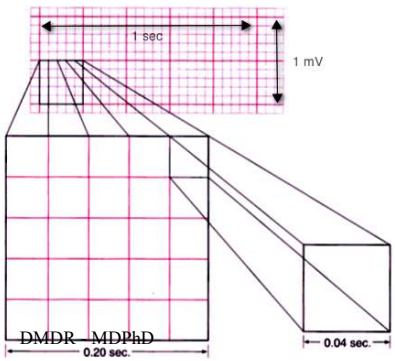

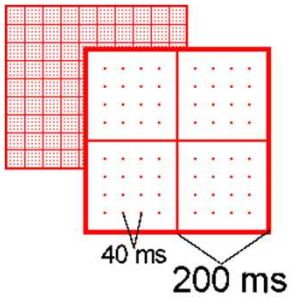

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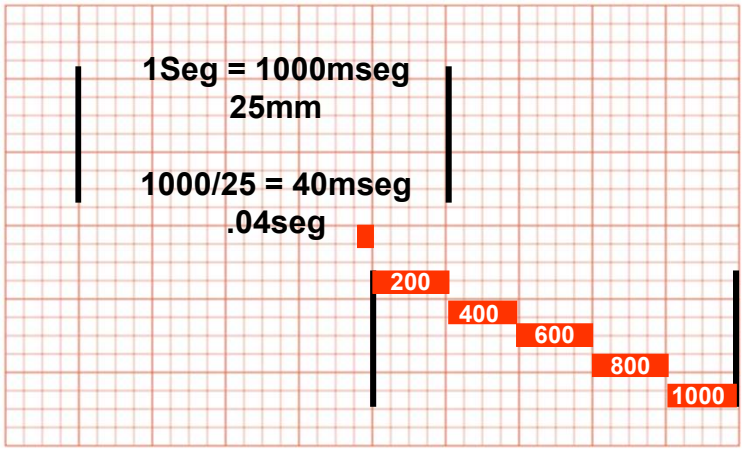
Frecuencia cardiaca



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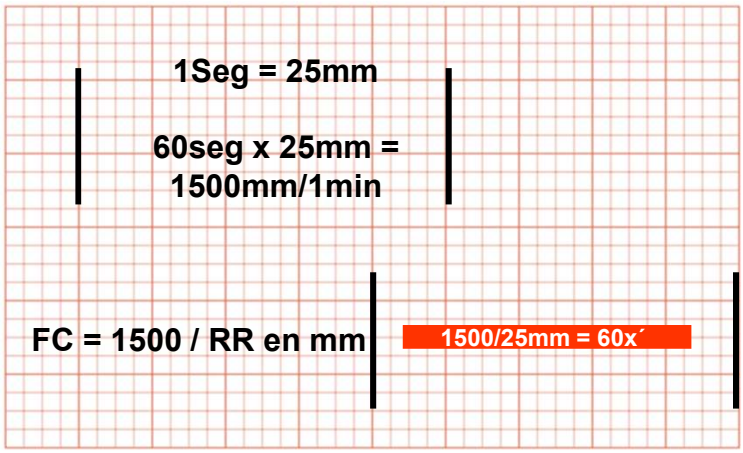
Frecuencia cardiaca



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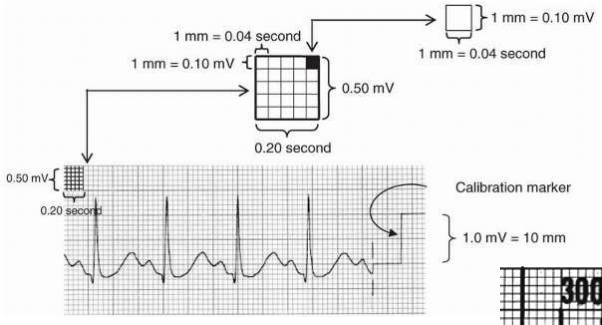

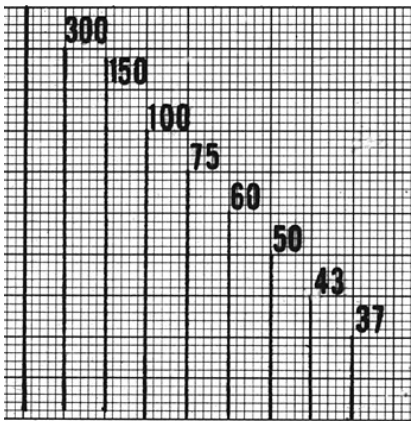

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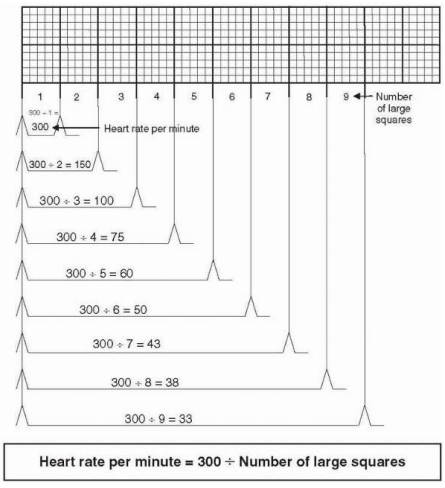

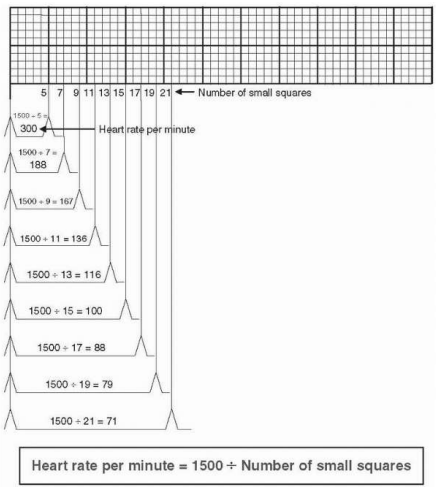

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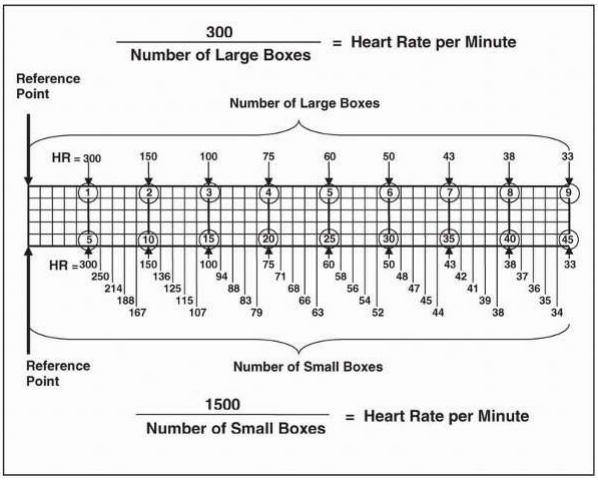

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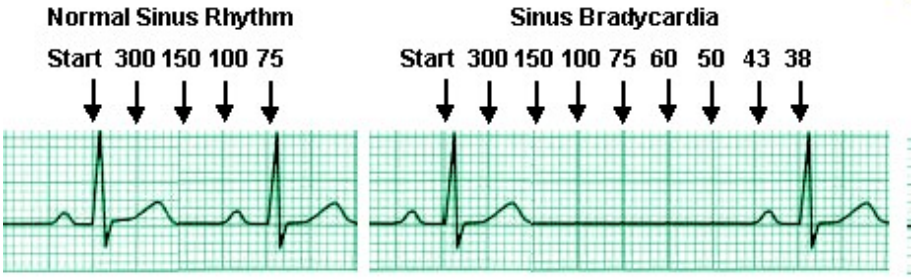

$$\text{Heart Rate} = \frac{1500}{\text{No. small boxes}}$$

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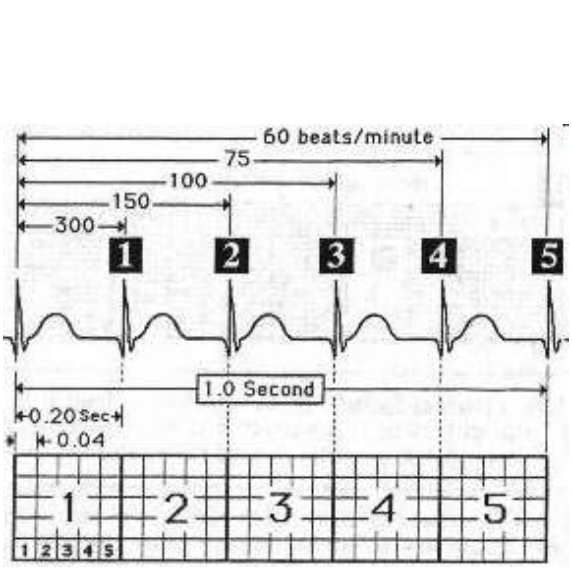

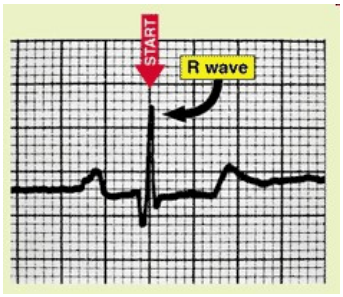

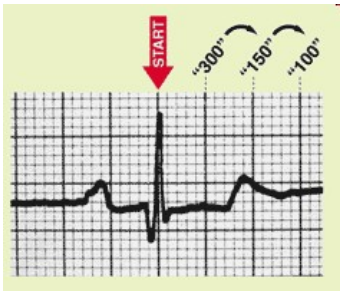

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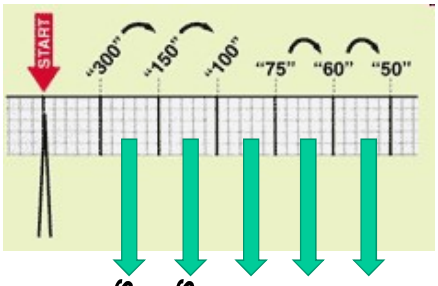


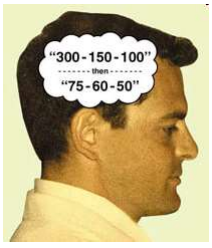
c/mm = 30 latidos

c/mm = 10 latidos

c/mm = 5 latidos

c/mm = 3 latidos

c/mm = 2 latidos



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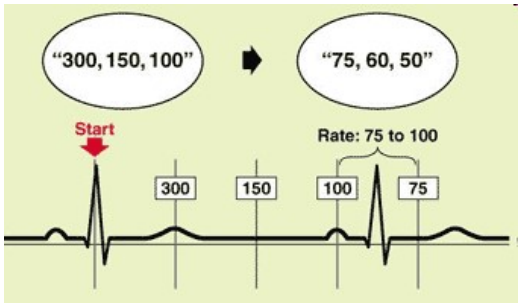

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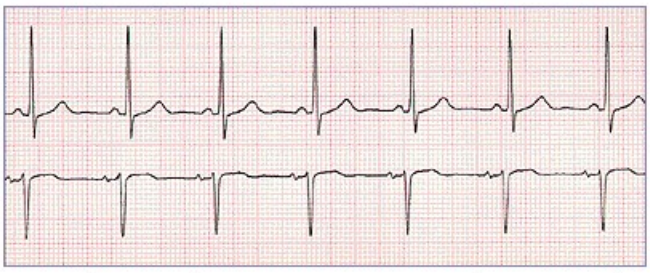
Normal Sinus Rhythm



Heart Rate	Rhythm	P Wave	PR Interval (in seconds)	QRS (in seconds)
60 - 100 bpm	Regular	Before each QRS, identical	.12 to .20	< .12

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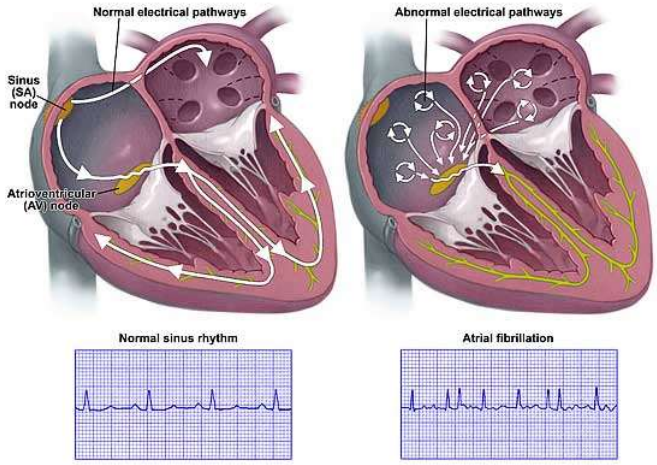

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Variaciones ECG en el Anciano

- **Relativa bradicardia**
- **Intervalo PR mas largo (hasta 220mseg)**
- **Eje QRS desviado a la izquierda**
- **Disminución de fuerzas iniciales (septales)**
- **Desnivel del segmento ST (>0.05mm)**
- **Ondas S hasta V6**

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Onda P

- **Duración <0.12seg. ó 120mseg.**
- **Amplitud <2.5mm**
- **Eje de Plano frontal: 0º a +75º**
- **Pueden ser melladas en el plano frontal**

Crecimientos Auriculares
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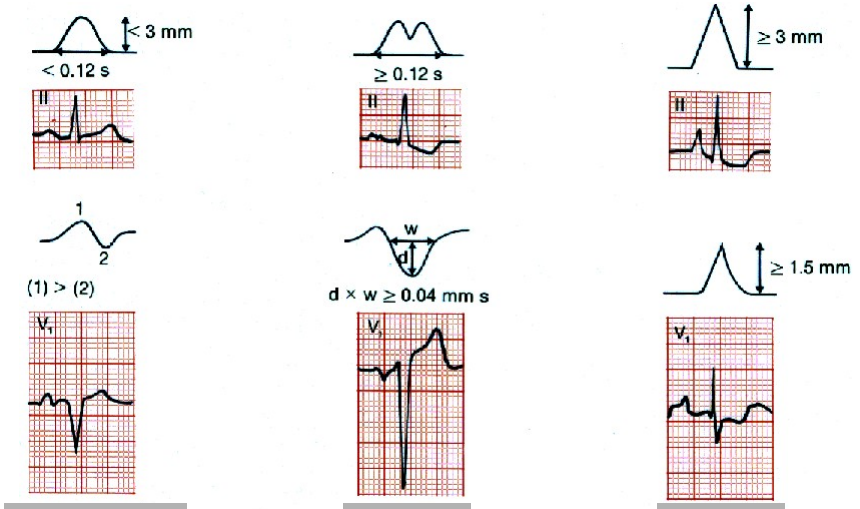
Onda P



Auricula normal

Hipertrofia Auricular Izquierda

Hipertrofia Auricular Derecha

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Crecimiento Auricular Derecho

- Onda P alta y picuda D1 y D2.
- Onda P aplanada en D3.
- P pulmonale (>2.5mm).
- Hipoxemia, simpaticotonía, enfisema.
- Bloqueo intraauricular derecho.

Crecimiento Auricular Izquierdo

- Onda P bimodal (>0.12seg) D2.
- Morfología en V_1.
- Negatividad evidente e inscripción lenta.
- P mitrale (estenosis mitral).

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Crecimiento Biauricular

- Onda P en D2 más ancha y alta de lo normal.
- Onda P ancha en derivaciones de extremidades y/o V3-V6 y picuda + en precordiales derechas.
- Signos de CAIzq con eje de P derecho
- Onda P bifásica en V1.
- AcxFa, más alteraciones del QRS.

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Criterios Diagnósticos de Crecimiento Auricular

Crecimiento Auricular Derecho:

1.- Morfología qR en V1 en ausencia de infarto (especificidad del 100%)
2.- Voltaje del QRS en V1 ≤ 4mV y voltaje del QRS en V2 / voltaje QRS en V1 ≥ 5 (sensibilidad 46%, especificidad 90%)
3.- Criterios basados en alteraciones de la onda P (>2.5mm en DII y/o ≥ 1.5mm en V1). Son poco sensibles y específicos.

Crecimiento Auricular Izquierdo:

1.- Onda P ≥0.12seg (120mseg) + P(-) terminal, con duración en V1 ≥ a 0.04seg (40mseg) (sensibiliadad 51%, especificidad 87%).
2.- P ± en DII, DIII, aVF (alta especificidad, baja sensibilidad).

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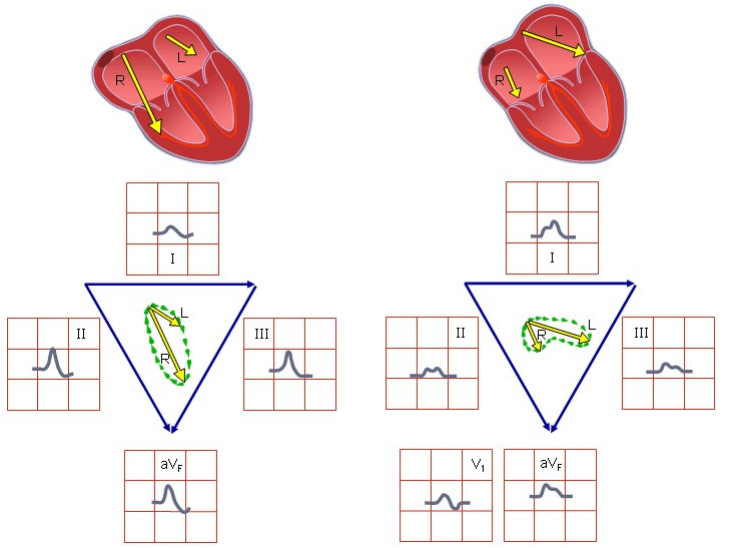
RIGHT ATRIAL HYPERTROPHY
Tall, peaked P wave in leads I and II

LEFT ATRIAL HYPERTROPHY
Wide, notched P wave in lead II. Diphasic P wave in V1



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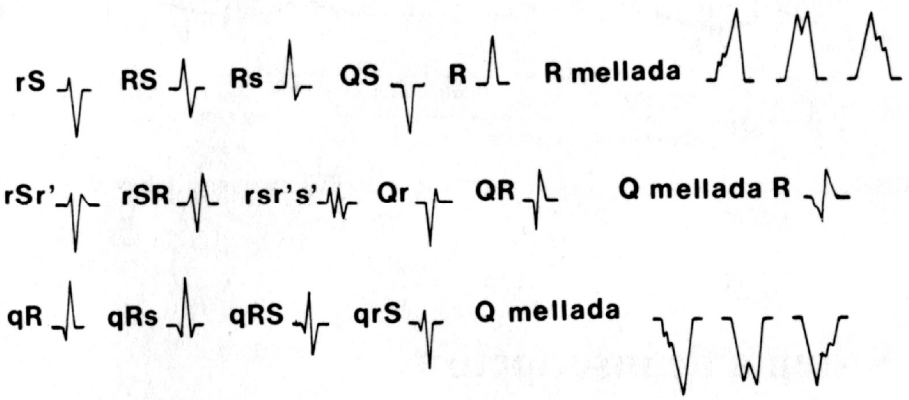
http://www.bem.fi/book/19/19.htm#03

Nomenclatura Complejo QRS



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INTERVALO QT

- **Duración total de la sístole eléctrica ventricular**
- **Varía con la FC**
- **QTc = QT/√RR (Fórmula de Bazett)**
  Hombres: Hasta 0.37seg.
  Mujeres: Hasta 0.44seg.

Bazett HC. An analysis of the time-relations of electrocardiograms. Heart. 1920;7:353-70.

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«Waller, Bazzet Taran, Szilagyi»

Sístole mecánica = K × RR

donde K tiene un valor de 0.343

En 1920, Bazett adaptó esta fórmula a la duración de la sístole eléctrica del corazón, el intervalo QT, y propuso que el valor normal del QT para una determinada frecuencia cardiaca es K × RR, donde K es 0.37 para el varón y 0.4 para la mujer.

$QTc = QT/\sqrt{RR}$

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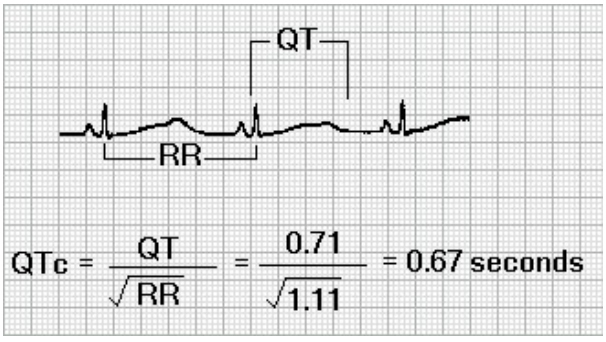
Besterman E, Creese R. Waller - pioneer of electrocardiography. Br Heart J. 1979;42:61-4.
The duration of the electrical systole (QT) in acute rheumatic carditis in children. Am Heart J. 1947;33:14-26.

INTERVALO QT



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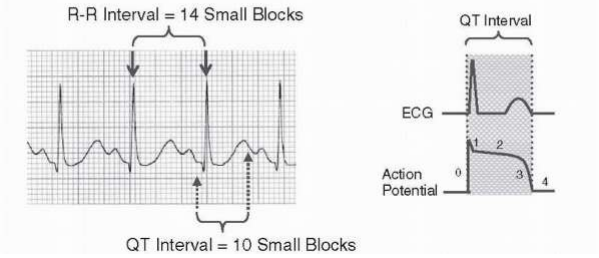
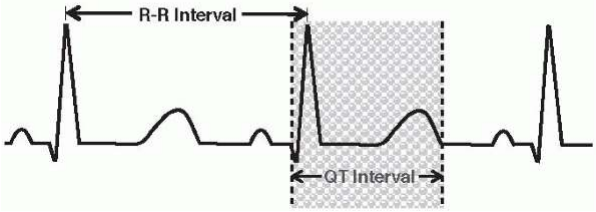
$$QTc = \frac{\text{QT interval (in seconds)}}{\sqrt{\text{R-R interval (in seconds)}}} = \frac{0.40}{0.75} = 0.53 \text{ sec}$$



$$QTc = \frac{\text{QT interval (in seconds)}}{\sqrt{\text{R-R interval (in seconds)}}}$$



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Fórmula de Bazett

$$QTc = QT / \sqrt{RR}$$

RR = 0.96seg (24mm) √.979
QT = 0.40seg (10mm)
0.40/.979 = 0.40seg

RR = 0.9seg √.948 (22.5mm)
QT = 0.36seg (9mm)
0.36/.948 = 0.37seg

RR = 0.8seg √.894 (20mm)
QT = 0.42seg (10.5mm)
0.42/.894 = 0.46seg

RR = 0.68seg √.824 (17mm)
QT = 0.36seg (9mm)
0.42/.894 = 0.43seg

Bazett HC. An analysis of the time-relations of electrocardiograms.
Heart 1920;7:353.

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PROLONGACION DEL INTERVALO QTc

Causas de prolongación marcada del QTc (>25%):

1.- Congénita (Síndrome de Jervell-Lange-Nielsen y Síndrome de Romano-Ward).
2.- Neurógena, incluyendo intoxicación por organofosforados.
3.- Hipotermia severa.
4.- Hipocalcemia severa.
5.- Regímenes dietéticos inapropiados.
6.- Inyecciones de contraste en las arterias coronarias.
7.- Algunos antiarrítmicos (amiodarona, etc.).
8.- Bradicardia severa, BAV, isquemia miocárdica, post RCP.

Causas de prolongación moderada del QTc (15 - 25%):

1.- Postisquemia por IAM transmural y no transmural.
2.- Varias miocardiopatías y postcirugía cardíaca.
3.- Hipocalcemia moderada.
4.- Fármacos antiarrítmicos clase I, tranquilizantes.
5.- Hipotiroidismo e insuficiencia hipofisaria.
6.- Neurógena o desconocida.

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INTERVALO QT

Medido del inicio del QRS al final de la onda T, en el plano frontal.
Normal: Dependiente de la FC.
(QTcorregido = QTc = QT medido / raíz^2 RR en segundos. Límite normal QTc = 0.44seg.)
Síndrome del QT largo - “SQTL" (basado en límites altos normales para la FC; QTc >0.47seg en hombres y >0.48seg. en mujeres, es diagnóstico de SQTL hereditario en ausencia de otras causas que lo provoquen).

Incrementa la vulnerabilidad de arritmias ventriculares malignas, síncope y muerte súbita. La arritmia prototipo es la *Torsade de pointes*, una TV polimórfica caracterizada por morfologías y amplitud cambiante del QRS sobre la línea basal isoeléctrica.

Causas:
Drogas (muchos antiarrítmicos, tricíclicos, fenotiazinas y otros).
Desequilibrios electrolíticos (K^+, Ca^{++}, Mg^{++}).
Enfermedad vascular cerebral (hemorragia subaracnoidea, EVC, trauma).
Prolongación del QT hereditario (Síndrome de Romano-Ward).
Cardiopatía isquémica (post-IAM).

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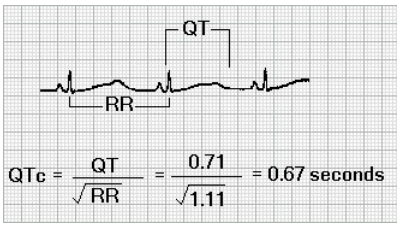
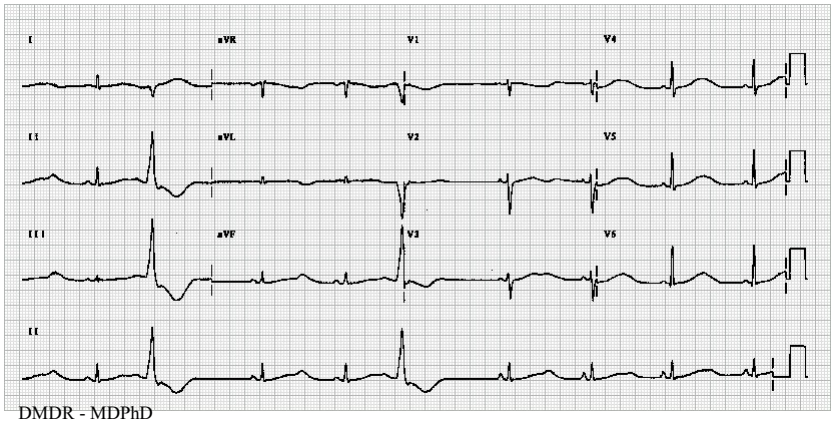
Romano-Ward syndrome





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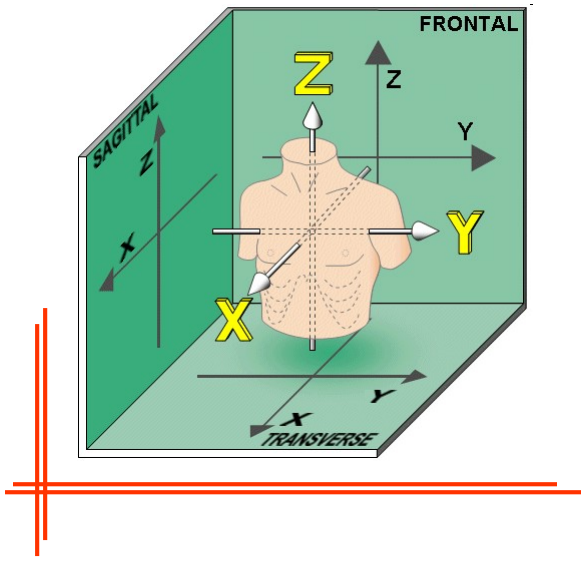

Eje Eléctrico Medio
Bloqueos AV y de Rama
Cardiopatía Isquémica

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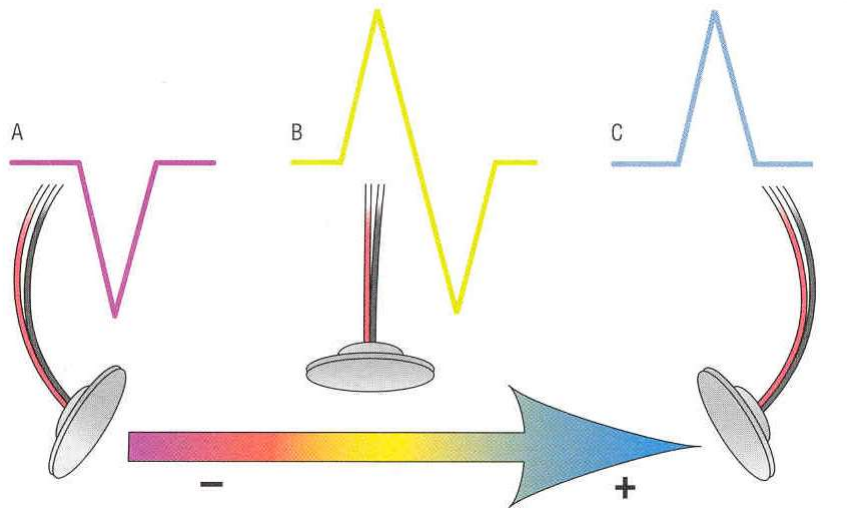
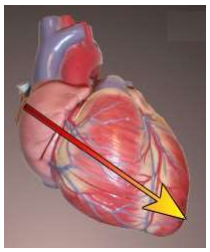
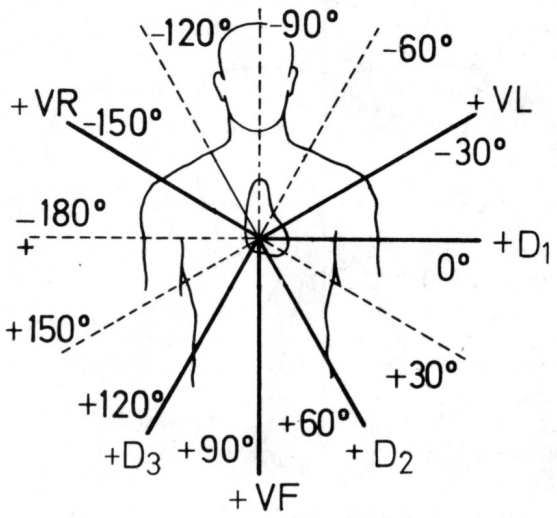
Cálculo del Eje Cardiaco





Sistema hexaxial de Bailey



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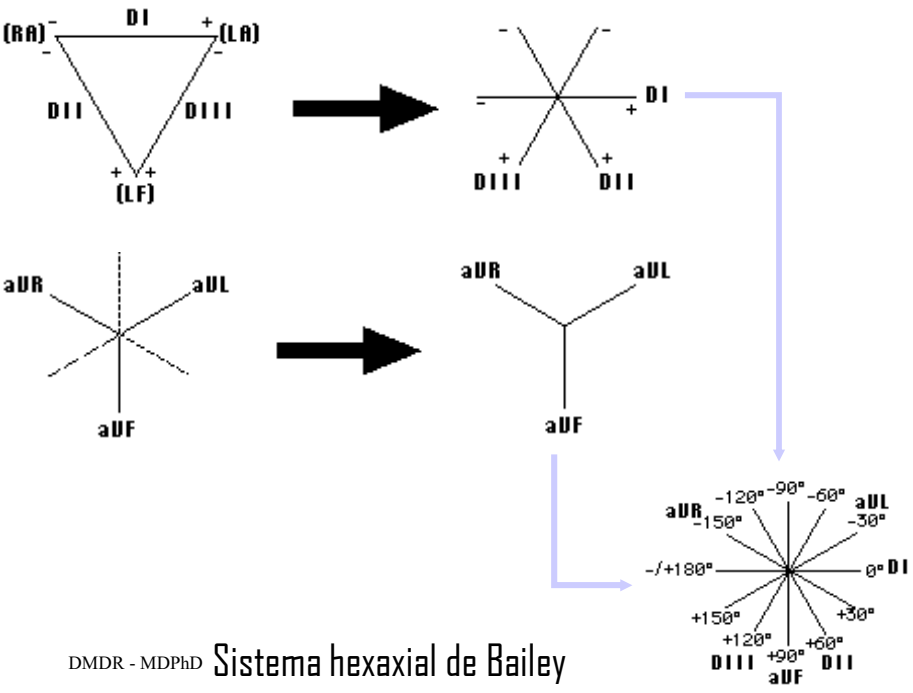
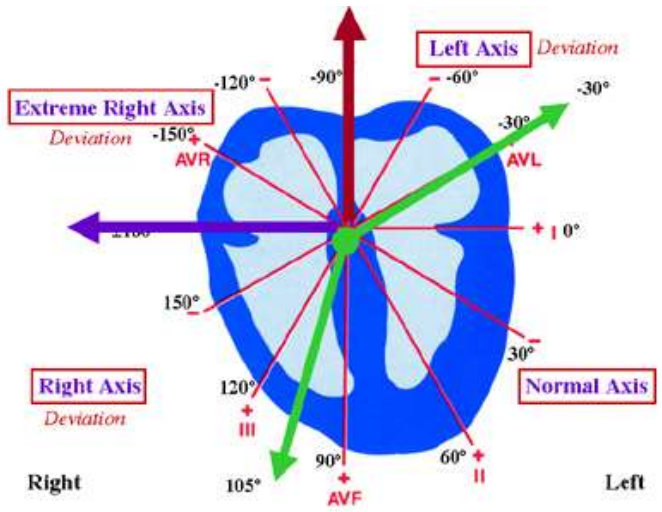


DMDR - MDPhD Sistema hexaxial de Bailey



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HEMICAMPOS

- Una línea perpendicular a cualquier derivación, que pasa por el centro del corazón, divide el cuerpo en dos hemicampos, uno + y otro -.
- En una derivación dada, un vector origina una deflexión + o – según que la cabeza de dicho vector se enfrente con el polo + o – de dicha derivación, o según que dicho vector caiga en el hemicampo + o – de la misma.

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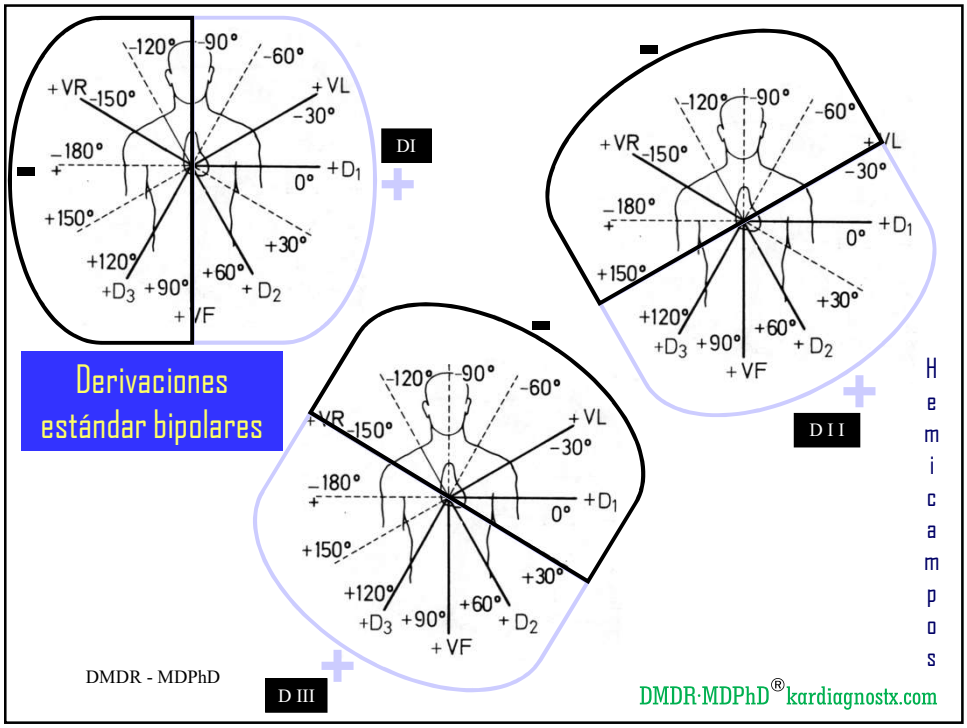
Derivaciones estándar bipolares
DI
D II
D III
Hemicampos
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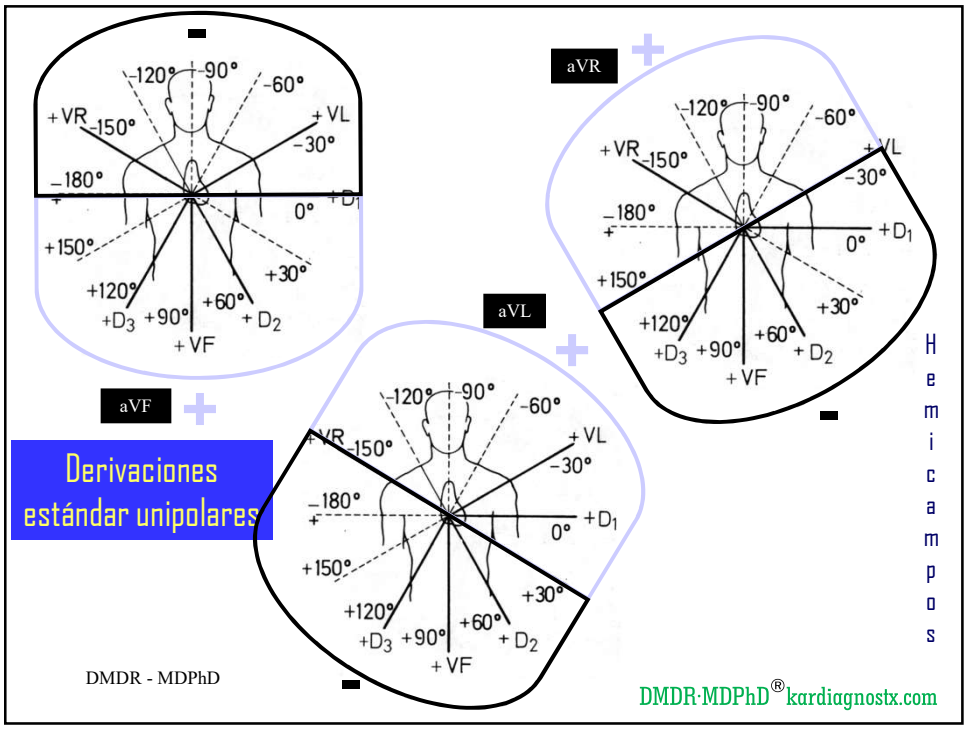
Derivaciones estándar unipolares
aVR
aVL
aVF
Hemicampos
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EJE QRS			
DI	DII	DIII	EJE
+	+	+	60°
+	+	±	30°
+	+	-	0°
+	±	-	-30°
+	-	-	-60°
±	-	-	-90°
-	-	-	-120
±	+	+	90°
-	+	+	120°
-	±	+	150°

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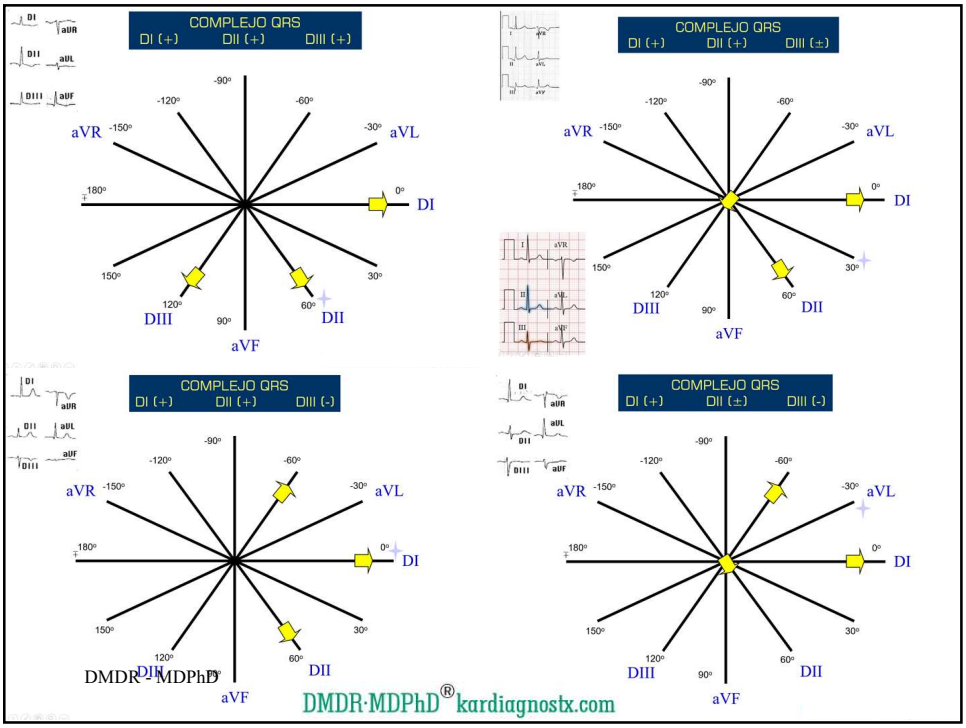

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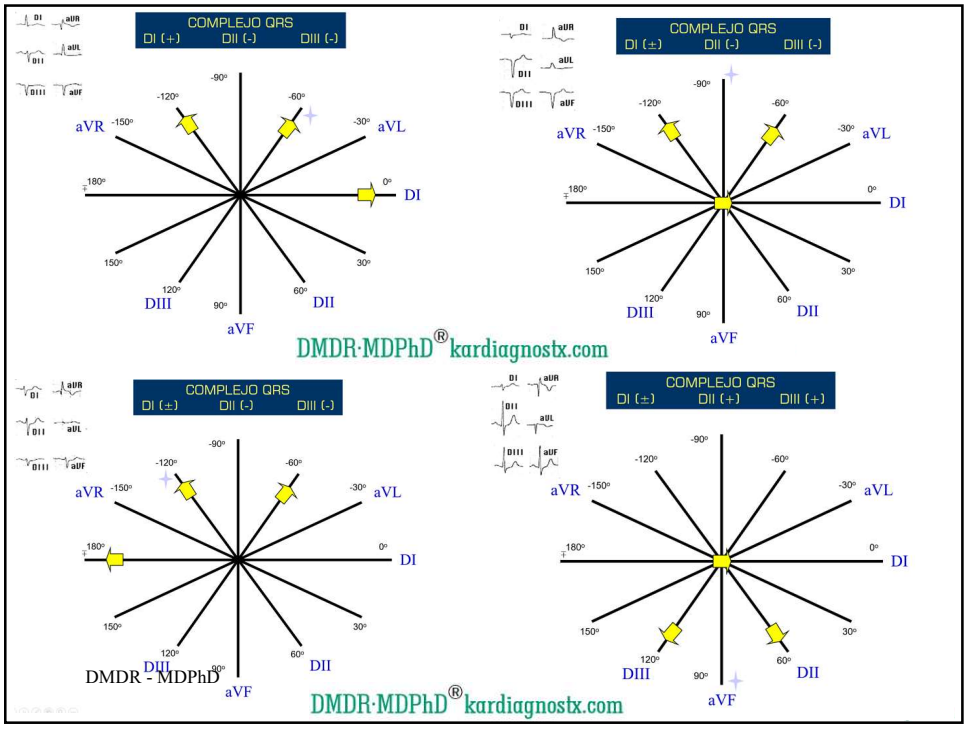
COMPLEJO QRS
DI (+) DII (-) DIII (-)
COMPLEJO QRS
DI (±) DII (-) DIII (-)
COMPLEJO QRS
DI (±) DII (-) DIII (-)
COMPLEJO QRS
DI (±) DII (+) DIII (+)
aVR
aVL
DI
DIII
DII
aVF
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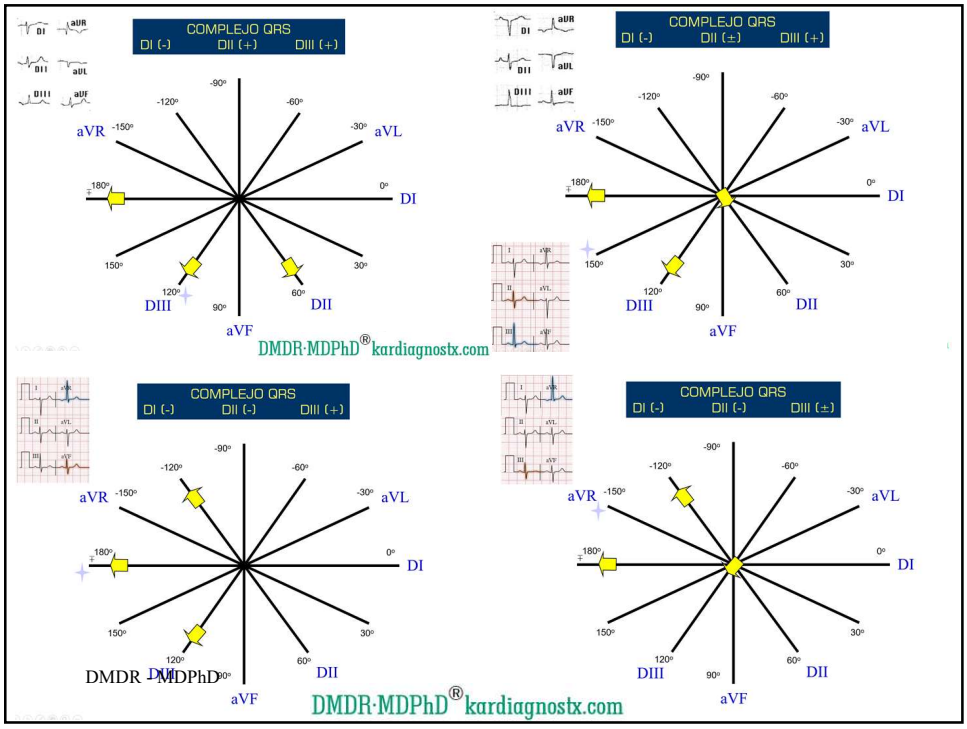
COMPLEJO QRS
DI (-) DII (+) DIII (+)
COMPLEJO QRS
DI (-) DII (±) DIII (+)
COMPLEJO QRS
DI (-) DII (-) DIII (+)
COMPLEJO QRS
DI (-) DII (-) DIII (±)
aVR
aVL
DI
DIII
DII
aVF
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Bloqueos AV y de Rama Ventriculares

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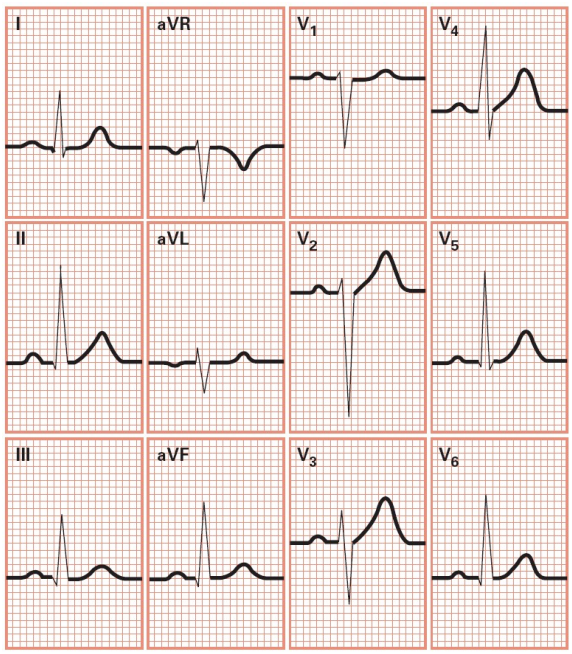
normal



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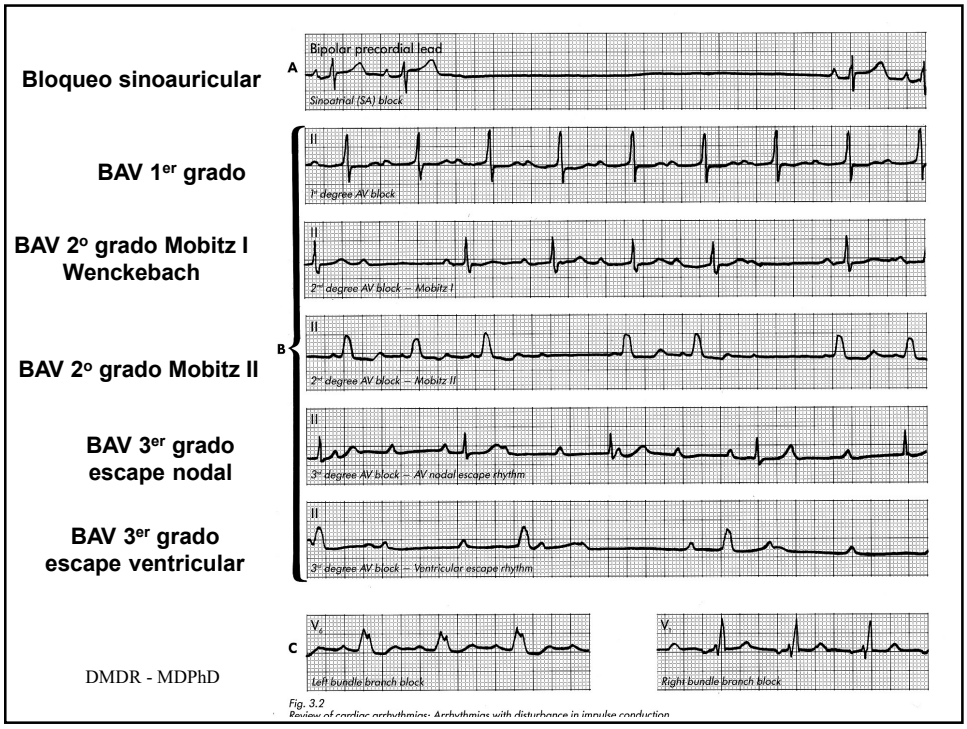
Bloqueo sinoauricular
BAV 1er grado
BAV 2o grado Mobitz I Wenckebach
BAV 2o grado Mobitz II
BAV 3er grado escape nodal
BAV 3er grado escape ventricular
A
B
C
Bipolar precordial lead
Sinoatrial (SA) block
1st degree AV block
2nd degree AV block – Mobitz I
2nd degree AV block – Mobitz II
3rd degree AV block – AV nodal escape rhythm
3rd degree AV block – Ventricular escape rhythm
Left bundle branch block
Right bundle branch block
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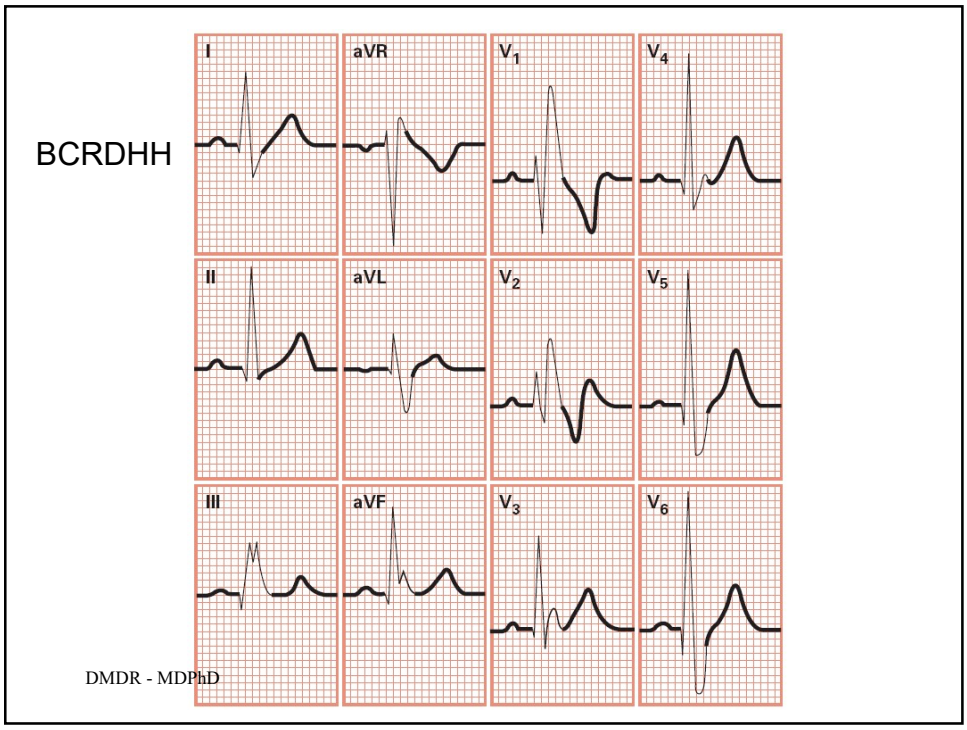
Fig. 3.2


BCRDHH
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
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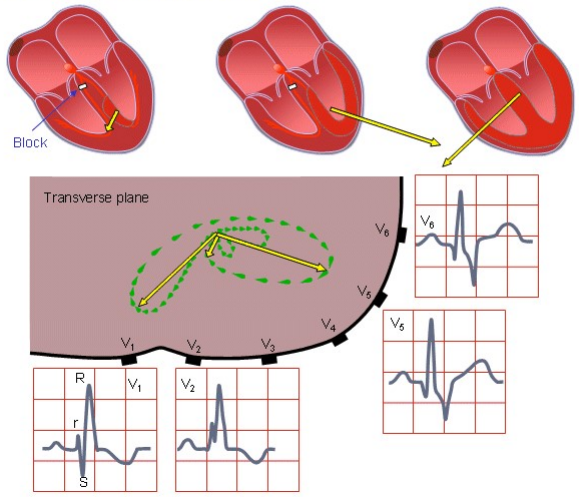
RIGHT BUNDLE-BRANCH BLOCK

QRS duration greater than 0.12 s
Wide S wave in leads I, V5, and V6



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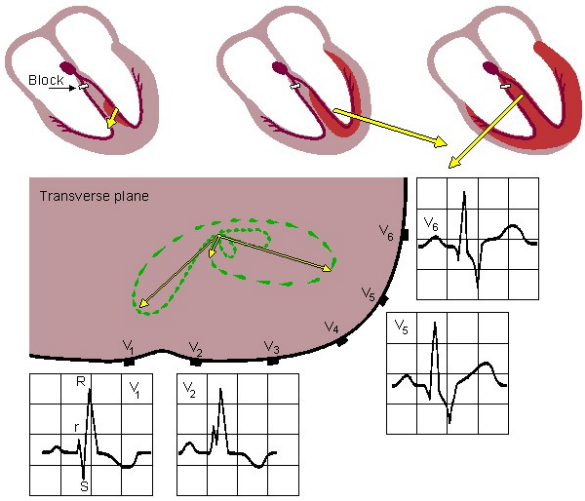
BLOQUEO DE RAMA DERCHA

QRS >120mseg.

Ondas S anchas en DI, V5 y V6.



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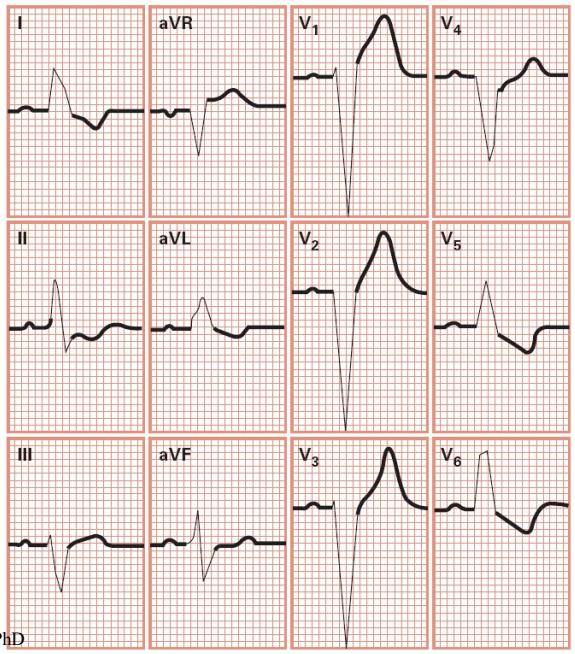
BCRIHH



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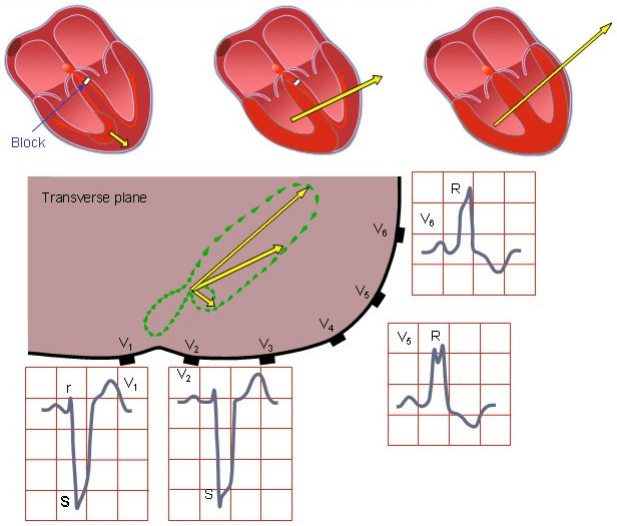
LEFT BUNDLE-BRANCH BLOCK

QRS duration greater than 0.12 s
Wide S wave in leads V1 and V2, wide R wave in V5 and V6



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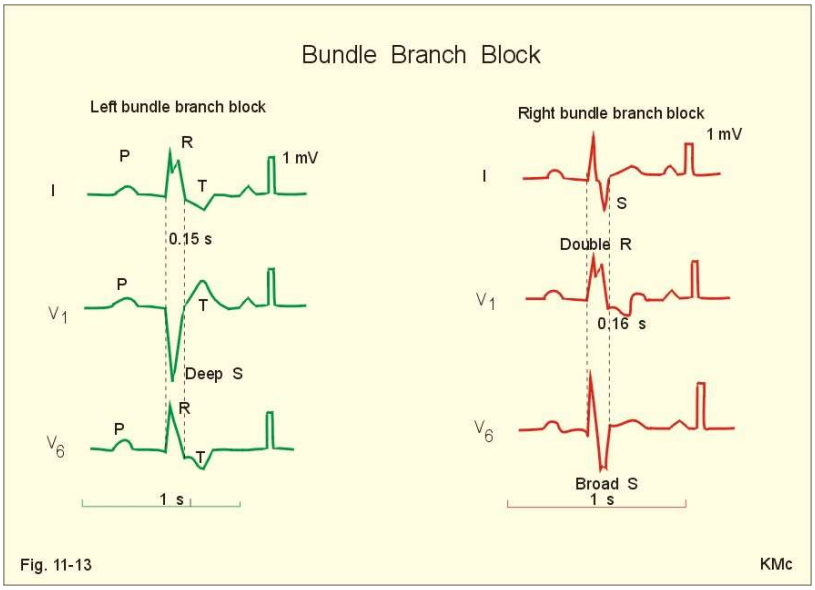

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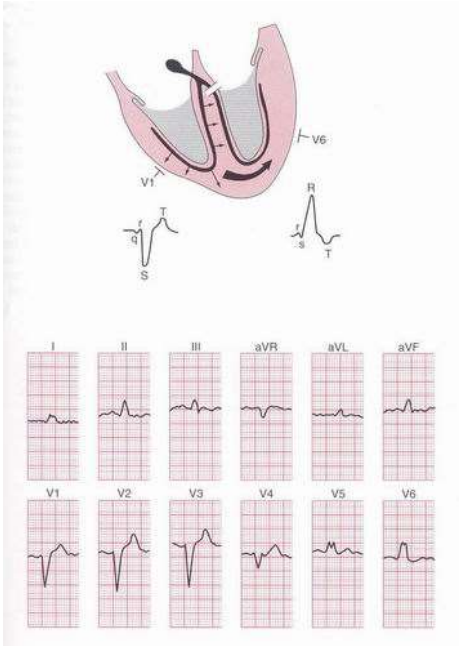

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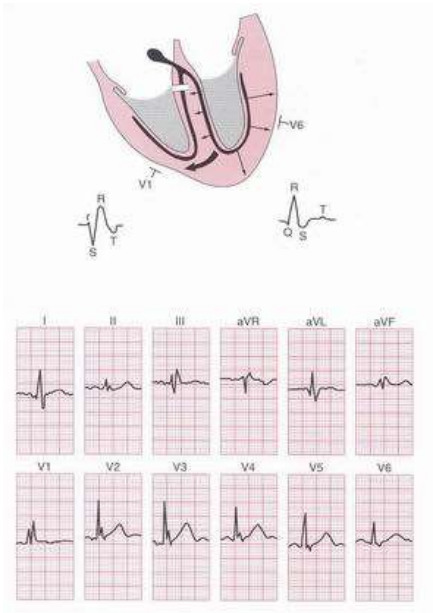

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El **fenómeno de Ashman** se debe a la dependencia de la longitud de ciclo que tiene la refractariedad de las células del tejido especializado de conducción pues el periodo refractario se alarga considerablemente cuando la amplitud del ciclo hace lo mismo y se acorta cuando el ciclo es corto.

Si el latido prematuro está precedido de un ciclo largo (que prolonga el período refractario de las células para el siguiente ciclo), tiene alta probabilidad de presentar aberrancia.

Como normalmente la rama derecha del haz de His tiene un período refractario mas largo que la izquierda, la aberrancia asociada al famoso “ciclo largo – latido corto” tiende a producirse en este sitio.

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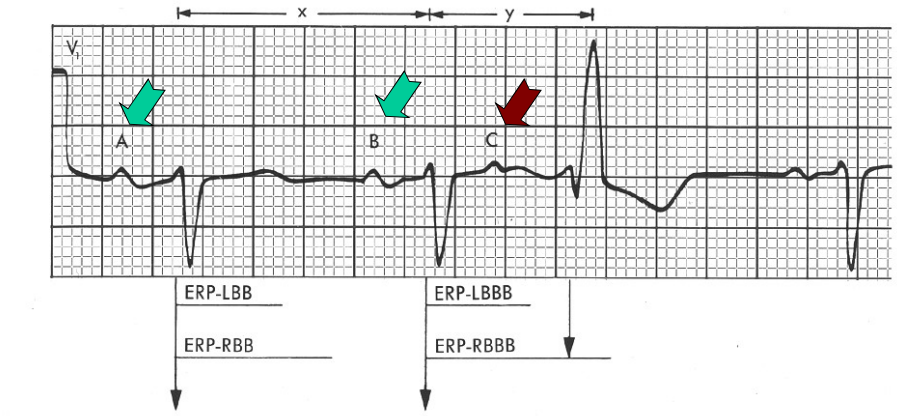
(Am Heart J 1947;34:366. Circulation 1969;39:345)

FENOMENO DE ASHMAN
(CSVP con Conducción Ventricular Aberrante)

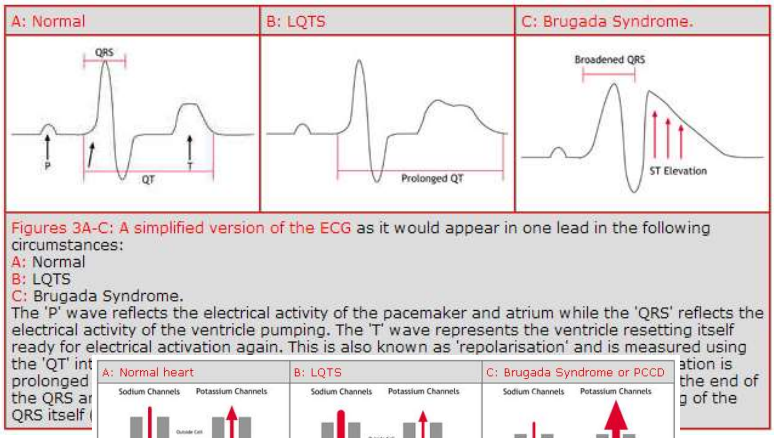


ERP-LBB: Effective refractory period of left bundle branch

ERP-RBB: Effective refractory period of right bundle branch

A y B: RS normal C: CSVP aislado

Síndrome de Brugada



Figures 3A-C: A simplified version of the ECG as it would appear in one lead in the following circumstances:
A: Normal
B: LQTS
C: Brugada Syndrome.
The 'P' wave reflects the electrical activity of the pacemaker and atrium while the 'QRS' reflects the electrical activity of the ventricle pumping. The 'T' wave represents the ventricle resetting itself ready for electrical activation again. This is also known as 'repolarisation' and is measured using the 'QT' int ation is prolonged the end of the QRS ar g of the QRS itself

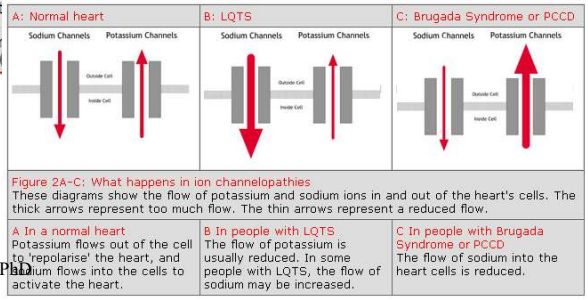


Figure 2A-C: What happens in ion channelopathies
These diagrams show the flow of potassium and sodium ions in and out of the heart's cells. The thick arrows represent too much flow. The thin arrows represent a reduced flow.

A In a normal heart	B In people with LQTS	C In people with Brugada Syndrome or PCCD
Potassium flows out of the cell to 'repolarise' the heart, and sodium flows into the cells to activate the heart.	The flow of potassium is usually reduced. In some people with LQTS, the flow of sodium may be increased.	The flow of sodium into the heart cells is reduced.

Isquemia, Lesión, Necrosis

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Isquemia Miocárdica

Proceso mediante el cual se reduce la presión de perfusión sanguínea en un área del músculo cardíaco que condiciona una deprivación de oxígeno tisular y evita la remoción de sus productos catabólicos

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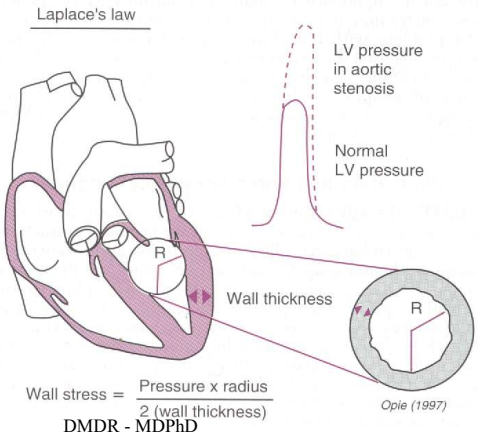
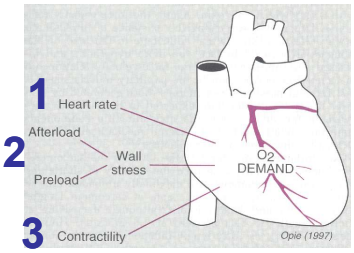
Determinantes del Consumo de O_2 miocárdico

Braunwald, 5th Ed. Heart Disease. 1997.





HIPOXIA: Condición en la que el aporte de oxígeno esta reducido aún en presencia de una perfusión adecuada.

ANOXIA: Ausencia de aporte de oxígeno con la presencia de una perfusión adecuada.

ISQUEMIA: Desequilibrio entre el aporte de oxígeno y la demanda metabólica tisular del mismo.

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SECUENCIA TEMPORAL DE LOS DIVERSOS EVENTOS CONSECUTIVOS A LA ISQUEMIA MIOCARDICA

Isquemia Miocárdica
(desequilibrio entre el aporte de 0_2 y la demanda metabólica del mismo)
⇩ $P0_2$ regional
Glicólisis anaeróbica
⇦ Lactato
⇦ Ph intracelular
Alteraciones del transporte de Ca^{++} mitocondrial
Alteraciones de la relajación (⇦ dp/dt-)
Depresión de la contractilidad (⇦ dp/dt+)
⇦ Engrosamiento parietal sistólico
⇧ Volumen telediastólico del VIzq. y del volumen telesistólico con ⇩ de la fracción de eyección
⇧ Presión telediastólica del VIzq.
Reducción del flujo subendocárdico
Alteraciones del ECG de superficie
Arritmias ventriculares, angor, IAM, ruptura parietal, etc.

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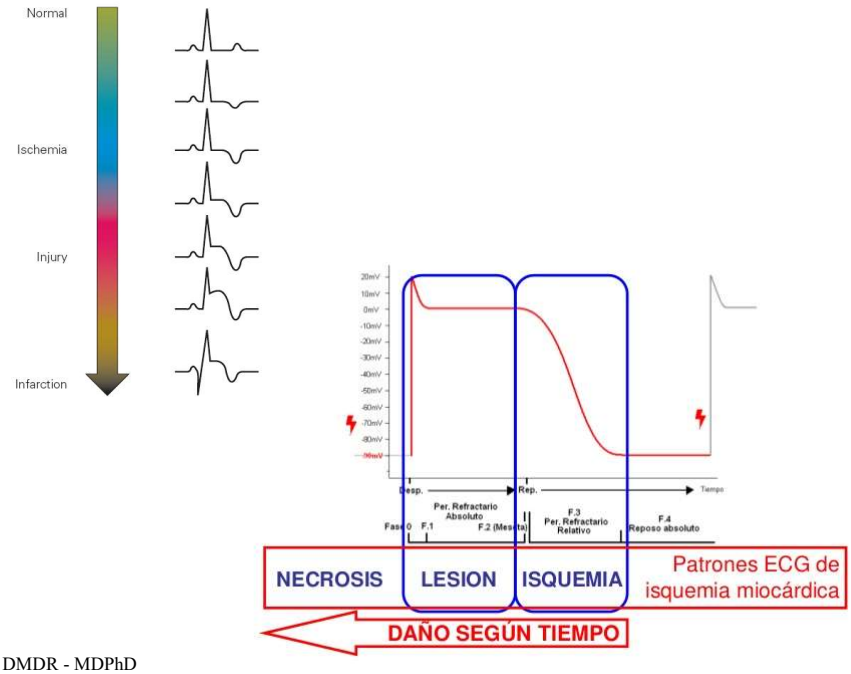

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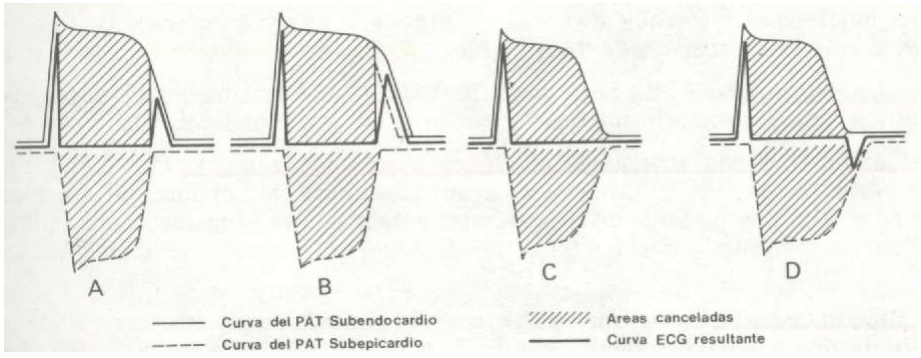
PAT Subendocárdico



PAT Subepicárdico

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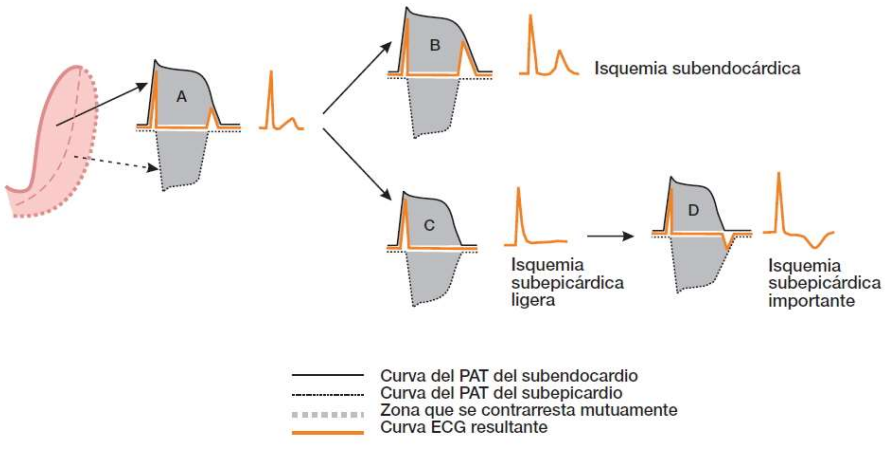

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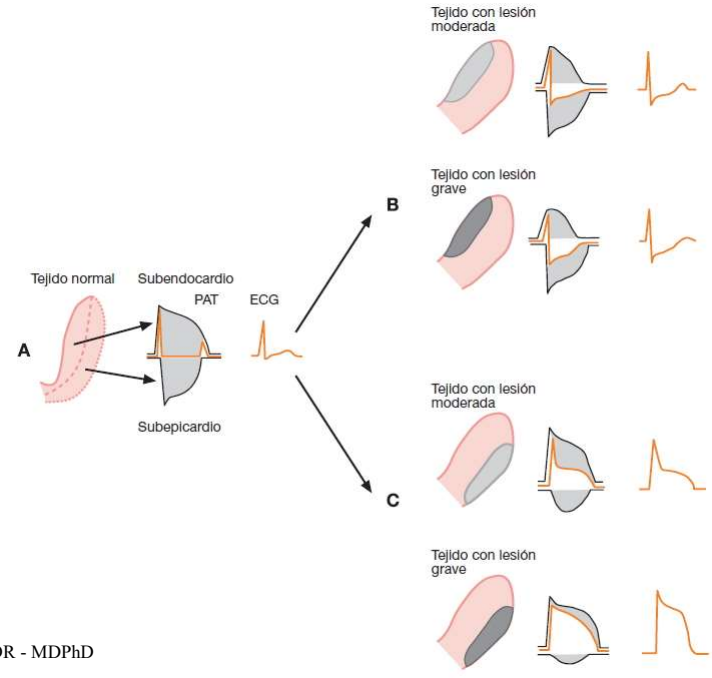

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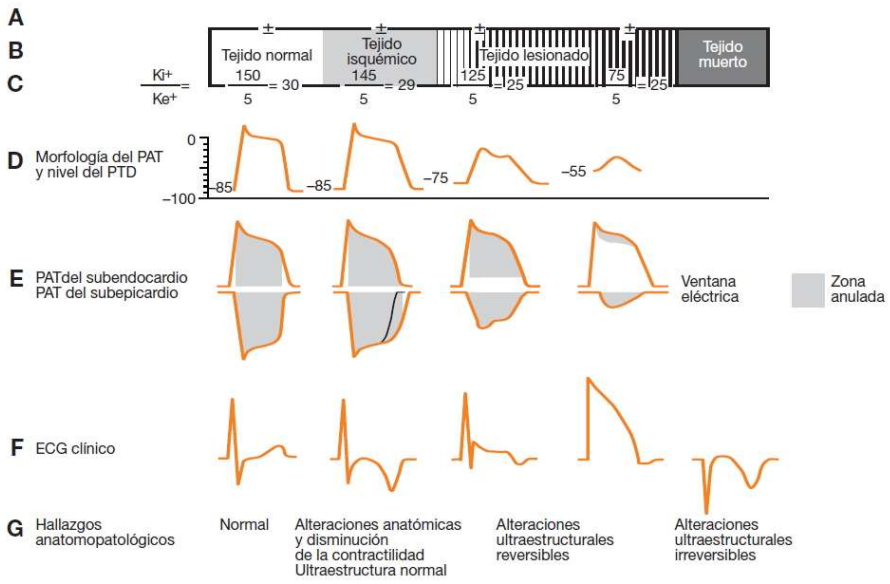

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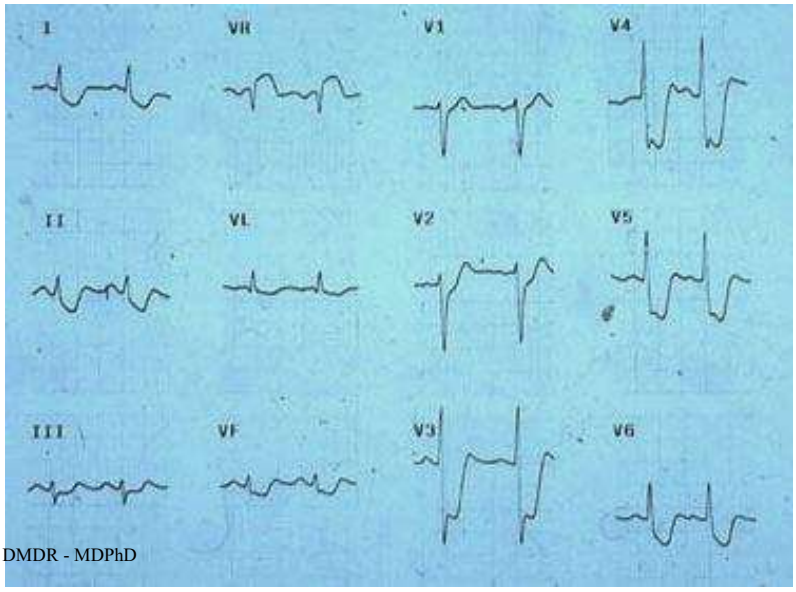
Lesión Subendocárdica



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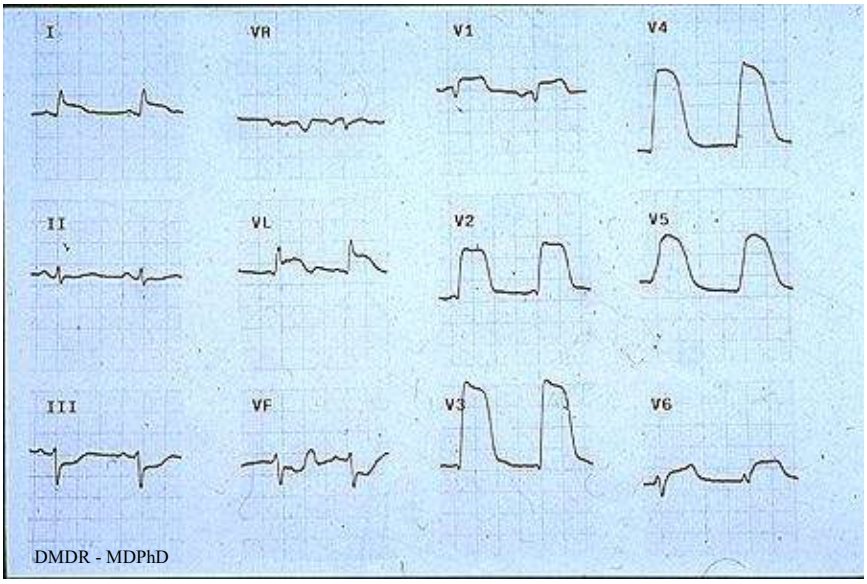
Lesión Subepicárdica



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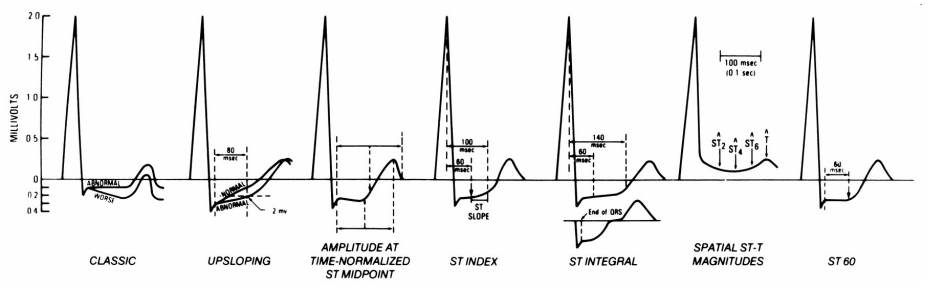
Abnormal ST responses. From Froelicher VF. Exercise and the Heart: Clinical Concepts. Chicago, Ill: Yearbook Medical Publishers, Inc; 1987:46.



(Circulation. 1995;91:580.)

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Marcador Molecular de Daño Miocárdico

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Troponina T
Peso molecular: 33
Detección: 2-4hrs.
Sensiblidad: 100% / 8-12hrs.
Pico: 10-24hrs.
Duración: 5-14 días.