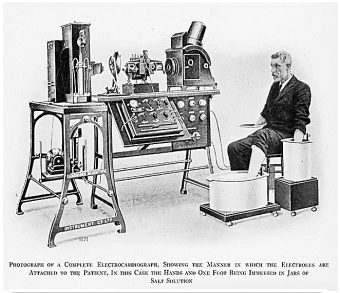
ELEKTROKARDIOGRÁFIA
ELECTROCARDIOGRAPHY
EKG (ECG)

A KEZDETEK ÉS MA



Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution

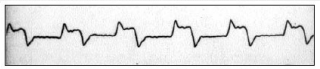
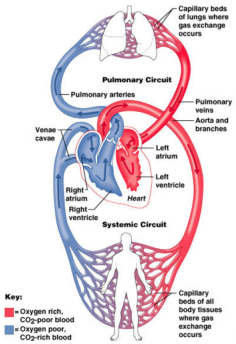


Fig. 2: An electrogram recorded by Waller using the Lippmann electrometer.

Először *Augustus Désiré Waller* angol fiziológus regisztrált emberi elektrokardiogramot 1887-ben, Lippmann féle k*apillárelektrométerrel.*
Willem Einthoven holland orvos-fiziológus kezdetben szintén kapillárelektrométert használt. Később a ma is használt elvezetés típusokat húros galvanométerrel dolgozta ki (1908). 1924-ben Nobel díjat kapott.

A SZÍVPUMPA

A KIS- ÉS NAGYVÉRKÖR

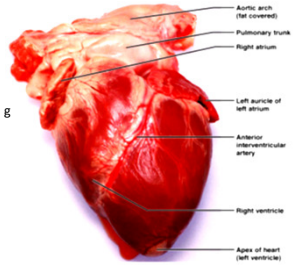


http://en.wikipedia.org/wiki/Heart

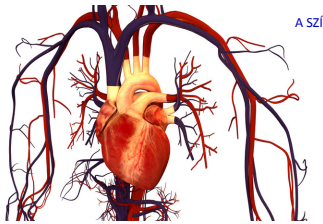
Súlya: nőknél 250-300 g
férfiaknál 300-350 g

Percenként 4,7-5,7 l vért továbbít.

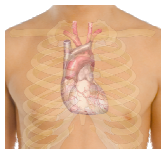
72/min pulzusszámmal számolva 75 év alatt ***3 milliárd*** összehúzódást végez.



A SZÍV ÉS A NAGYEREK

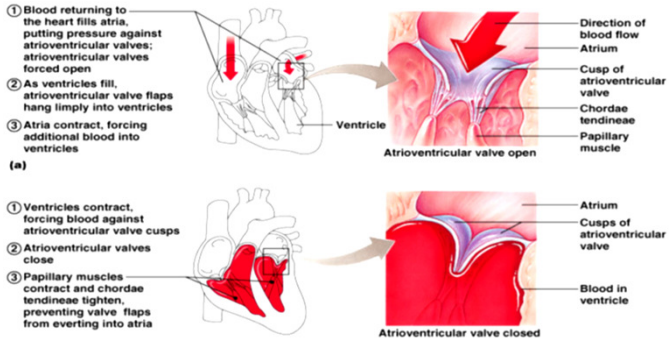


A SZÍV HELYZETE A MELLKASBAN



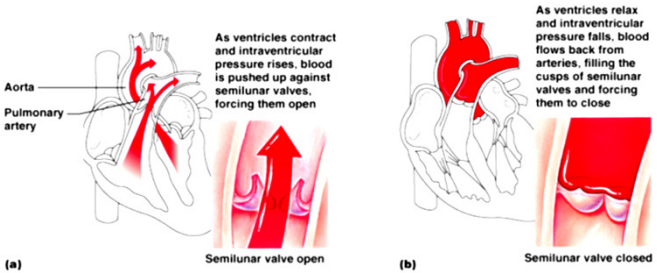
AZ ATRIOVENTRICULARIS BILLENYTŰK

A pitvarok és kamrák között. Megakadályozzák a pitvarokba történő visszafolyást.

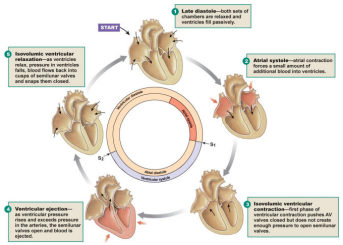


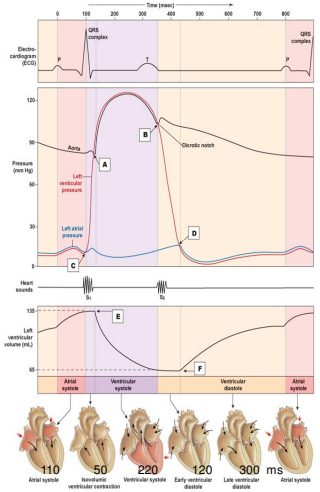
SEMILUNARIS BILLENTYŰK

A nagy erek eredésénél. Megakadályozzák, a kamrákba történő visszafolyást.



SZÍVCIKLUS





A bal kamra térfogata diasztolé végén (EDV): ~120 ml
A bal kamra térfogata szisztolé végén: (ESV) ~50 ml
A szív egy kontrakciónál ~70 ml vért továbbít az aortába (stroke volume [SV]).
Ez naponta kb. ~7,5 m³ térfogatnak felel meg.

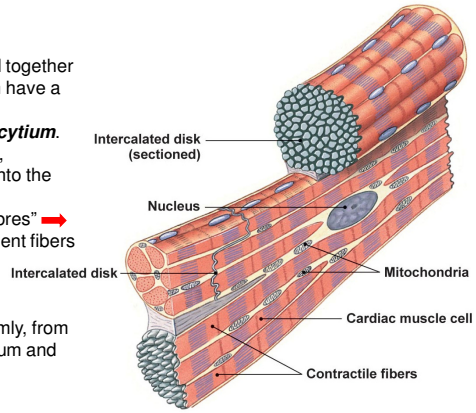
Ejekciós frakció (EF=SV/EDV): 0,5-0,7

A szív oxigénfogyasztása 25-30 ml/min, a teljes szervezet fogyasztásának 12 %-a. Terhelésre ez akár hétszeresére nőhet.

A SZÍVIZOM

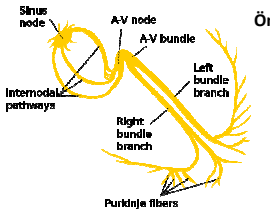
Cells of the ventricular myocardium are coupled together by ***gap junctions***, which have a very low resistance.
Heart behaves as a ***syncytium***. Excitation, once initiated, continues to propagate into the region that is still at rest.
„Uniform heart muscle fibres" ➡ Equivalent fibers

Activation wavefronts proceed relatively uniformly, from endocardium to epicardium and from apex to base.



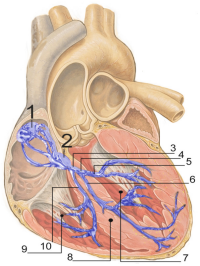
Sequence of instantaneous depolarization wavefront.

A SZÍV INGERKÉPZŐ ÉS VEZETŐ RENDSZERE



Önálló ingerképzési frekvencia:

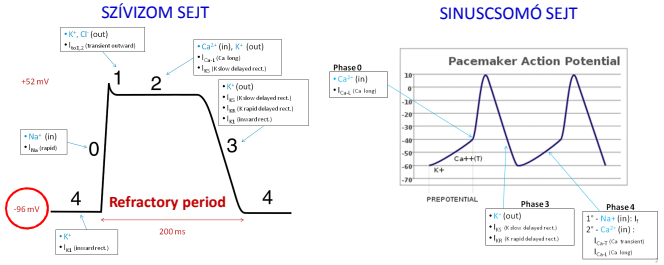
Sinuscsomó: ~ 100/min
Pitvari myociták: --
Av csomó: 40-50/min
Tawara szárak és
Purkinje-rosok: 25-40/min
Kamrai myociták: --



Terjedési sebesség:
SA csomó: 0,01 - 0,05 m/s
Pitvar izom: 1 m/s pitvari ingerület
AV csomó: 0,02 - 0,05 m/s áttevődés lassú válasz
His köteg: 1,2-2,0 m/s
Tawara szárak, Purkinje rostok: 2-4 m/s
Kamra izom: 0,3 - 1 m/s kamrai ingerület
subendocardium → subepicardium
szívcsúcs → bázis

1. Sinuscsomó -- Sinoatrial node (SA)
2. Atrioventrikularis csomó -- Atrioventricular node (AV)
3. His köteg -- Bundle of His
4. Bal Tawara szár -- Left bundle branch
5. Bal hátsó köteg -- Left posterior fascicle
6. Bal első köteg -- Left-anterior fascicle
7. Bal kamra -- Left ventricle
8. Septum -- Ventricular septum
9. Jobb kamra -- Right ventricle
10. Jobb Tawara szár -- Right bundle branch

A SZÍVIZOM ÉS AZ INGERKÉPZŐ SEJTEK AKCIÓS POTENCIÁLJAI



4 – resting membrane potential @ -90mV
0 – depolarization
Due to gap junctions or conduction fiber action
Voltage gated Na^+ channels open… close at 20mV
1 – temporary repolarization
Open K^+ channels allow some K^+ to leave the cell
2 – plateau phase
Voltage gated Ca^{++} channels are fully open (started during initial depolarization)
3 – repolarization
Ca^{++} channels close and K^+ permeability increases as slower activated K^+ channels open, causing a quick repolarization

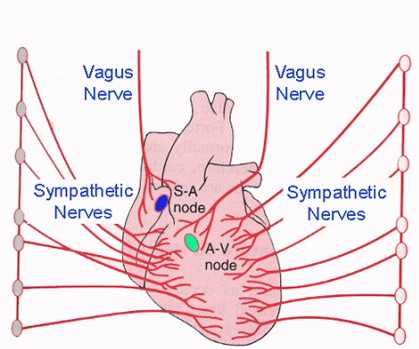
Autorhythmic Cells (pacemaker cells):

Unstable membrane potential:
"bottoms out" at -60mV,
"drifts upward" to -40mV, forming a pacemaker potential
The upward "drift" allows the membrane to reach threshold potential (-40mV) by itself

A VÁZIZOM ÉS A SZÍVIZOM AKCIÓS POTENCIÁL ÖSSZEHASONLÍTÁSA

	SKELETAL MUSCLE	CONTRACTILE MYOCARDIUM	AUTORHYTHMIC MYOCARDIUM
Membrane potential	Stable at −70 mV	Stable at −90 mV	Unstable pacemaker potential; usually starts at −60 mV
Events leading to threshold potential	Net Na^+ entry through ACh-operated channels	Depolarization enters via gap junctions	Net Na^+ entry through I_f channels; reinforced by Ca^{2+} entry
Rising phase of action potential	Na^+ entry	Na^+ entry	Ca^{2+} entry
Repolarization phase	Rapid; caused by K^+ efflux	Extended plateau caused by Ca^{2+} entry; rapid phase caused by K^+ efflux	Rapid; caused by K^+ efflux
Hyperpolarization	Due to excessive K^+ efflux at high K^+ permeability when K^+ channels close; leak of K^+ and Na^+ restores potential to resting state	None; resting potential is −90 mV, the equilibrium potential for K^+	Normally none; when repolarization hits −60 mV, the I_f channels open again. ACh can hyperpolarize the cell.
Duration of action potential	Short: 1–2 msec	Extended: 200+ msec	Variable; generally 150+ msec
Refractory period	Generally brief	Long because resetting of Na^+ channel gates delayed until end of action potential	None

A SZÍV BEIDEGZÉSE



Vegetatív idegrendszer

Hatások:

chronotrop: szívfrekvencia

inotrop: izomerő, dP/dt

dromotrop: vezetési sebesség

bathmotrop: ingerlékenység

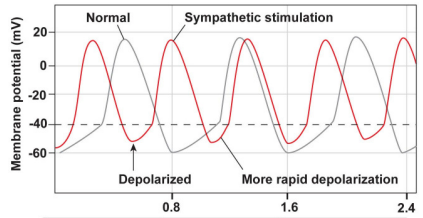
A SZÍV BEIDEGZÉSÉNEK HATÁSAI

Sympathetic Activity Summary:

increased chronotropic effects
↑heart rate

increased dromotropic effects
↑conduction of APs

increased inotropic effects
↑contractility

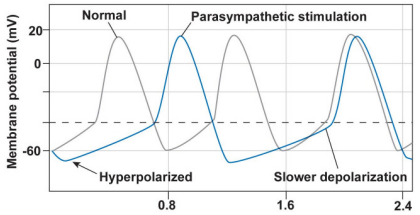


Parasympathetic Activity Summary:

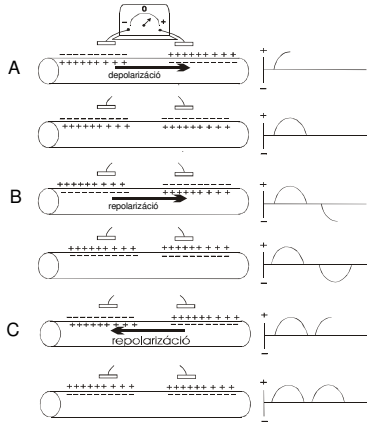
decreased chronotropic effects
↓heart rate

decreased dromotropic effects
↓ conduction of APs

decreased inotropic effects
↓ contractility

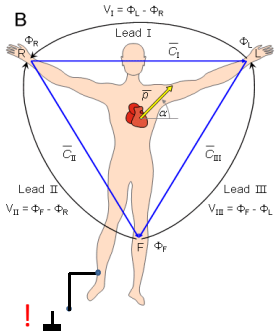


AZ EKG HULLÁMOK POLARITÁSÁNAK KIALAKULÁSA



A depolarizáció (A) alatt kialakuló felszíni potenciálváltozások mechanizmusa, amikor a repolarizáció iránya azonos (B), illetve ellentétes (C) a depolarizáció irányával.

EINTHOVEN FÉLE EKG VÉGTAG ELVEZETÉSEK AZ EINTHOVEN FÉLE HÁROMSZÖG



Einthoven limb leads and Einthoven triangle. The Einthoven triangle is an approximate description of the lead vectors associated with the limb leads. Lead I is shown as I in the above figure, etc.

Bipolar leads

The Einthoven limb leads (standard leads) are defined in the following way:

Lead I: $V_I = \Phi_L - \Phi_R$

Lead II: $V_{II} = \Phi_F - \Phi_R$

Lead III: $V_{III} = \Phi_F - \Phi_L$

where

V_I = the voltage of Lead I

V_{II} = the voltage of Lead II

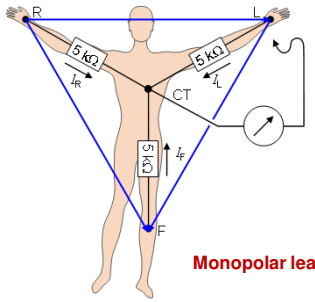
V_{III} = the voltage of Lead III

Φ_L = potential at the left arm

Φ_R = potential at the right arm

Φ_F = potential at the left foot

WILSON FÉLE MONOPOLÁRIS EKG ELVEZETÉSEK



Frank Norman Wilson (1890-1952) investigated how electrocardiographic unipolar potentials could be defined. Central terminal serves as this reference (1931-34).

Wilson *central terminal* (CT) is formed by connecting a 5 k resistance to each limb electrode and interconnecting the free wires; the CT is the common point.

Monopolar leads

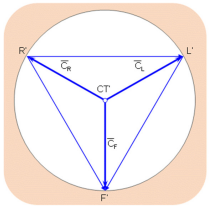
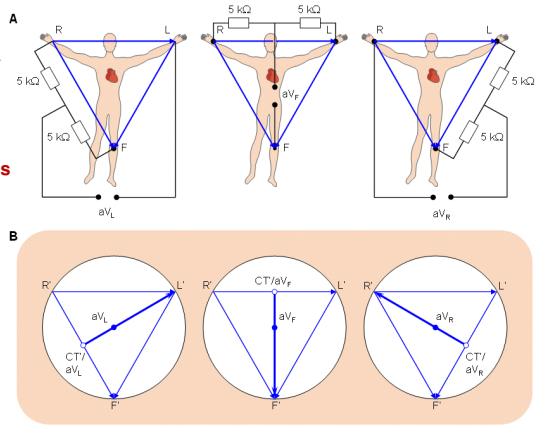


IMAGE SPACE

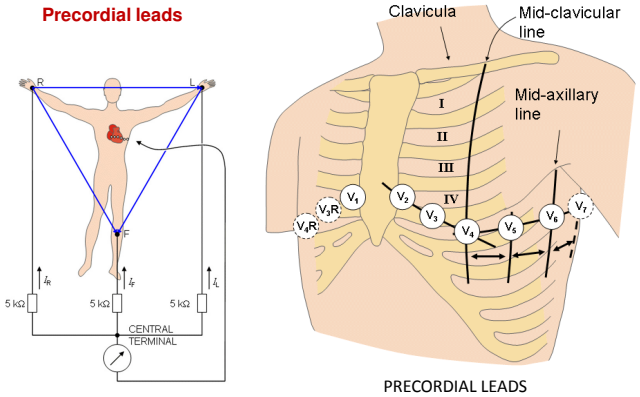
GOLDBERGER FÉLE „MEGNÖVELT" MONOPOLÁRIS ELVEZETÉS

E. Goldberger 1942

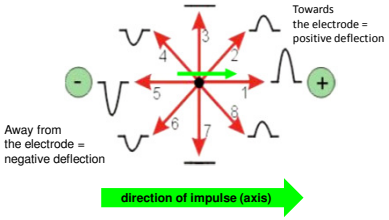
Augmented monopolar leads



MELLKASI EKG ELVEZETÉSEK

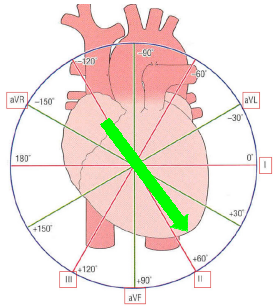


A JEL POLARITÁSA A TERJEDŐ INGERÜLETI HULLÁM FÜGGVÉNYÉBEN



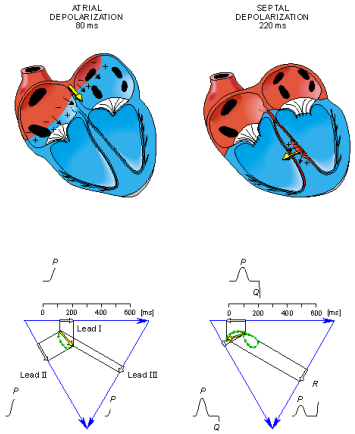
EQUIVALENT VECTOR

ELECTRODES AROUND THE HEART



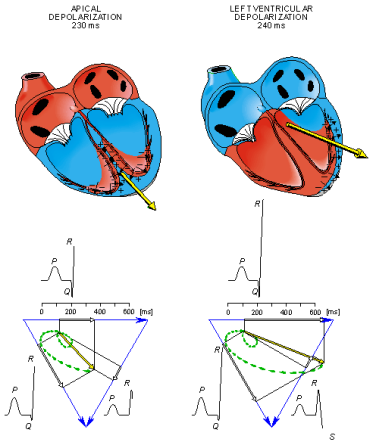
AZ EKG HULLÁM LÉTREJÖTTE A SZÍVCIKLUS SORÁN 1.

Electric activation of the heart starts at the sinus node and spreads along the atrial walls. The projections of this resultant vector on each of the three Einthoven limb leads is positive, and therefore, the measured signals are also positive.
After the depolarization has propagated over the atrial walls, it reaches the AV node. The propagation through the AV junction is very slow and involves negligible amount of tissue; it results in a delay in the progress of activation.
Activation has reached the ventricles, propagation proceeds along the Purkinje fibers to the inner walls of the ventricles. The ventricular depolarization starts first from the left side of the interventricular septum, and therefore, the resultant dipole from this septal activation points to the right. Figure shows that this causes a negative signal in leads I and II.



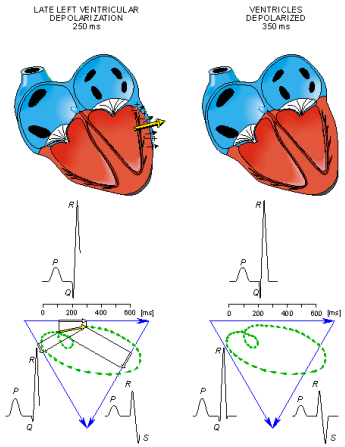
AZ EKG HULLÁM LÉTREJÖTTE A SZÍVCIKLUS SORÁN 2.

After a while the depolarization front has propagated through the wall of the right ventricle; when it first arrives at the epicardial surface of the right-ventricular free wall, the event is called *breakthrough*. Because the left ventricular wall is thicker, activation of the left ventricular free wall continues even after depolarization of a large part of the right ventricle. Because there are no compensating electric forces on the right, the resultant vector reaches its maximum in this phase, and it points leftward.



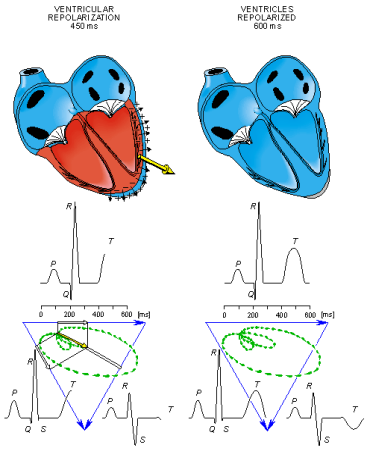
AZ EKG HULLÁM LÉTREJÖTTE A SZÍVCIKLUS SORÁN 3.

The depolarization front continues propagation along the left ventricular wall toward the back. Because its surface area now continuously decreases, the magnitude of the resultant vector also decreases until the whole ventricular muscle is depolarized. The last to depolarize are basal regions of both left and right ventricles. Because there is no longer a propagating activation front, there is no signal either.



AZ EKG HULLÁM LÉTREJÖTTE A SZÍVCIKLUS SORÁN 4.

Ventricular repolarization begins from the outer side of the ventricles and the repolarization front "propagates" inward. This seems paradoxical, but even though the epicardium is the last to depolarize, its action potential durations are relatively short, and it is the first to recover. Recovery generally does move from the epicardium toward the endocardium.
The inward spread of the repolarization front generates a signal with the same sign as the outward depolarization front. (recall that both direction of repolarization and orientation of dipole sources are opposite). Because of the diffuse form of the repolarization, the amplitude of the signal is much smaller than that of the depolarization wave and it lasts longer.

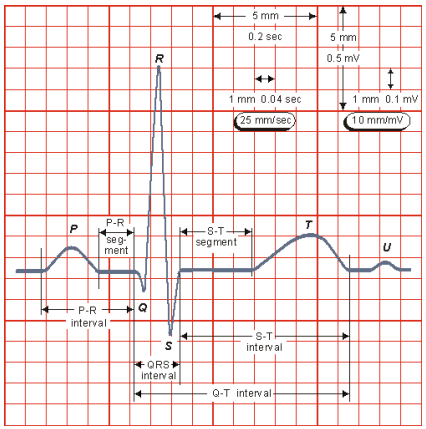


A JELLEMZŐ EKG GÖRBE

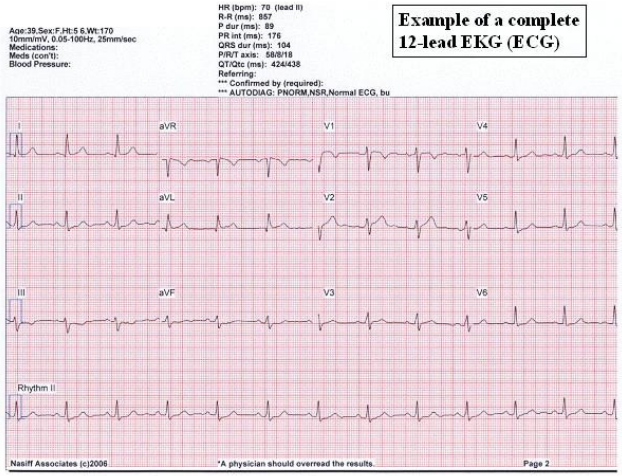
Az EKG-görbe időviszonyai:

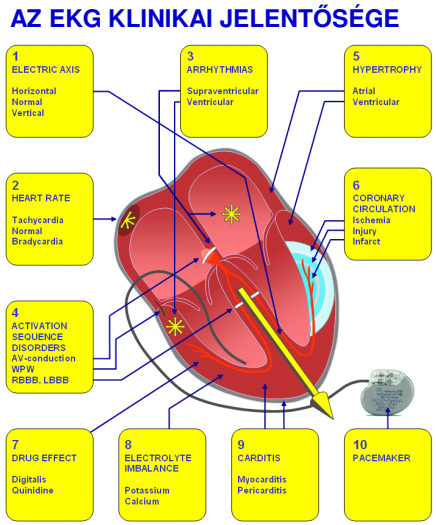
P hullám:	80-100 ms
QRS komplexum:	*60-80 ms*
P-Q intervallum:	*120-200 ms*
P-R intervallum:	120-200 ms
QT intervallum:	
40/min szívfrekv:	450 ms
80/min szívfrekv:	350 ms
120/min szívfrekv:	280 ms
180/min szívfrekv:	230 ms

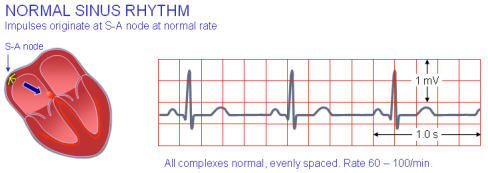
Q-T_c intervallum: $QT/\sqrt{RR}$
Q-T_c intervallum: *≥440 ms*



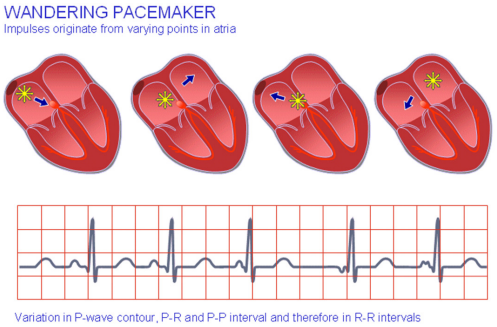
A 12 ELVEZETÉSES KLINIKAI EKG REGISZTRÁTUM



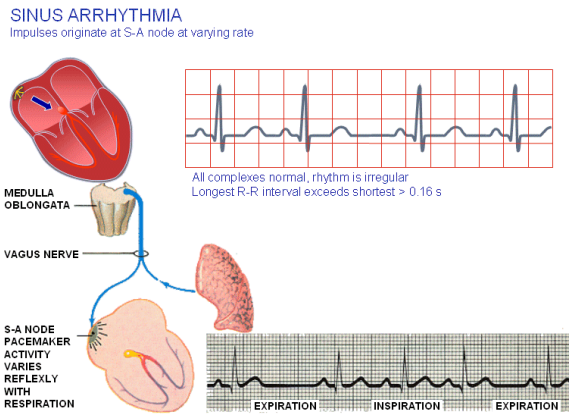


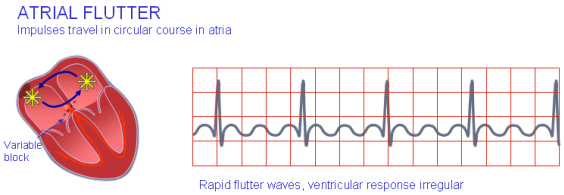


A sinus rhythm of less than 60/min is called sinus bradycardia.
A sinus rhythm of higher than 100/min is called sinus tachycardia.

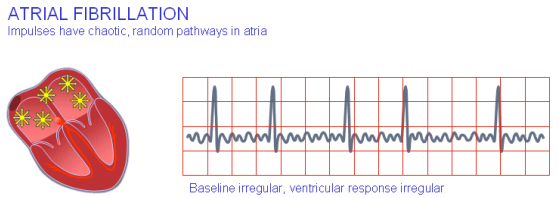


The origin of the atrial contraction may also vary or *wander*. Consequently, the P-waves will vary in polarity, and the PQ-interval will also vary.



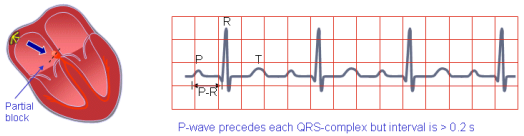


The frequency of these fluctuations is between 220 and 300/min. The AV-node and, thereafter, the ventricles are generally activated by every second or every third atrial impulse (2:1 or 3:1 heart block).



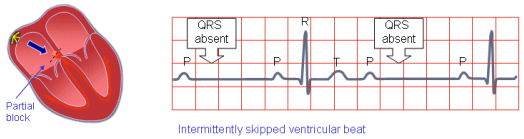
A-V BLOCK, FIRST DEGREE

Atrioventricular conduction lengthened



A-V BLOCK, SECOND DEGREE

Sudden dropped QRS-complex



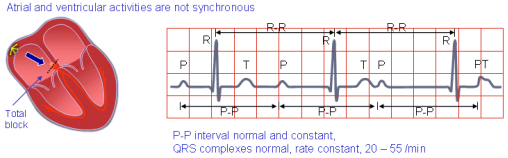
1st degree:
constant PR, >0.2 seconds

2nd degree:
type 1
(Wenckebach)
PR widens over subsequent beats then a QRS is dropped
type 2
PR is constant then a QRS is dropped

3rd degree:
No discernable relationship between p waves and QRS complexes

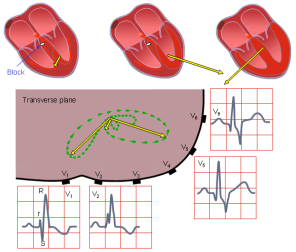
A-V BLOCK, THIRD DEGREE

Impulses originate at AV-node and proceed to ventricles
Atrial and ventricular activities are not synchronous



RIGHT BUNDLE-BRANCH BLOCK

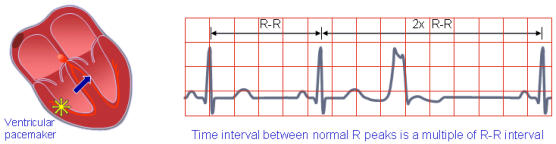
QRS duration greater than 0.12 s
Wide S wave in leads I, V5, and V6



The right bundle-branch is defective so that the electrical impulse cannot travel through it to the right ventricle, activation reaches the right ventricle by proceeding from the left ventricle.
It then travels through the septal and right ventricular muscle mass.

PREMATURE VENTRICULAR CONTRACTION

A single impulse originates at right ventricle

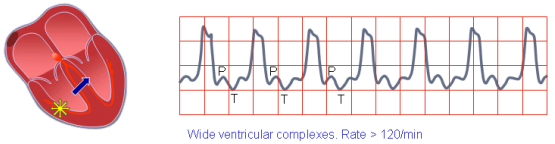


A premature ventricular contraction is one that occurs abnormally early. If its origin is in the atrium or in the AV node, it has a supraventricular origin. The complex produced by this supraventricular arrhythmia lasts less than 0.1 s. If the origin is in the ventricular muscle, the QRS-complex has a very abnormal form and lasts longer than 0.1 s. Usually the P-wave is not associated with it.

Extraszisztolé
- szupraventrikuláris
- ventrikuláris

VENTRICULAR TACHYCARDIA

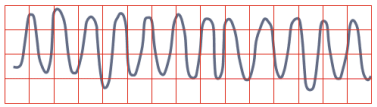
Impulses originate at ventricular pacemaker



A rhythm of ventricular origin may also be a consequence of a slower conduction in ischemic ventricular muscle that leads to circular activation (re-entry). The result is activation of the ventricular muscle at a high rate (over 120/min), causing rapid, bizarre, and wide QRS-complexes. The arrythmia is called ventricular tachycardia. As noted, ventricular tachycardia is often a consequence of ischemia and myocardial infarction.

VENTRICULAR FIBRILLATION

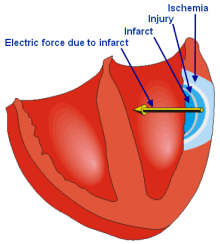
Chaotic ventricular depolarization



Rapid, wide irregular ventricular complexes

When ventricular depolarization occurs chaotically, the situation is called ventricular fibrillation. This is reflected in the ECG, which demonstrates coarse irregular undulations without QRS-complexes. The cause of fibrillation is the establishment of multiple re-entry loops usually involving diseased heart muscle. In this arrhythmia the contraction of the ventricular muscle is also irregular and is ineffective at pumping blood. The lack of blood circulation leads to almost immediate loss of consciousness and death within minutes. The ventricular fibrillation may be stopped with an external defibrillator pulse and appropriate medication.

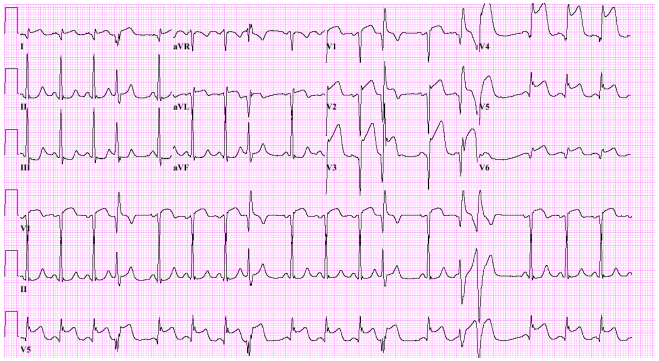
SZÍVINFARKTUS



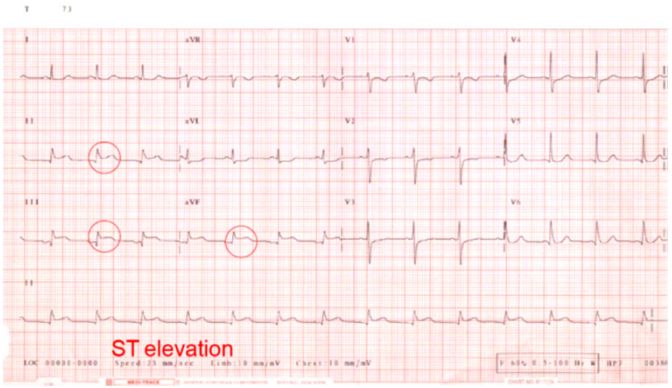
MYOCARDIAL ISCHEMIA AND INFARCTION

If a coronary artery is occluded, the transport of oxygen to the cardiac muscle is decreased, causing an oxygen debt in the muscle, which is called *ischemia.* Ischemia causes changes in the resting potential and in the repolarization of the muscle cells, which is seen as changes in the T-wave. If the oxygen transport is terminated in a certain area, the heart muscle dies in that region. This is called an *infarction.*
An infarct area is electrically silent since it has lost its excitability. According to the solid angle theorem the loss of this outward dipole is equivalent to an electrical force pointing inward. With this principle it is possible to locate the infarction.

AKUT SZÍVIZOM INFARKTUS



ALSÓ FALI SZÍVIZOM INFARKTUS



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