9/20/1

heart muscle has different metabolism
1. different CHhys used
2. lactic acid not accumulated
3. lactates used
4. fat "

Oxygen supply
greater capillary supply
no sarcolemma sheath
more myoglobin, cytochrome & other oxidation enzymes

Refractory period
rhythmicity insured by long refractory period

different parts different periods – Purkinje has shorter period

period of hyperexcitability after relative refractory period conduction also faster in this period

compensatory pause after extrasystoly produced during relative refractory period by greater stimulus. One beat dropped out. This evidence shows stimulus is rhythmical

Tissue	absolutely R. P.	Rel. Ref. Period
Frog Sciatic (22°C)	2.0 - 2.3 msec	7.0 - 15.0 msec
mammalian A fibers (37°C)	0.4 "	3.0 "
Skeletal muscle Frog sartorius (24°C)	3.0 "	11.0 "
Smooth muscle cat uterus nictitating membrane (35°C)	1000 msec	50.0 "

* Cardiac muscle
Ventricle (75 beats/min.) 120.0 msec 200.0 "

Refractory period length may vary. (curious that vagus decreases & ~~[illegible]~~ adrenalin increases)

All or None Law - due to syncytial nature

pulsus alternans [illegible] is apparently contradiction of above law really cause is that some fibers so unnormal that beats every other time

efficiency changes under different conditions
This is a change in "All
usual efficiency - 25%
When some work greater efficiency up to certain point
Starling's Law of the Heart

when increased venous return

9/20/2

heart has intrinsic rhythmicity
(smooth muscle & skeletal m. in special media also)

myogenic & neurogenic theories
1. strips of muscle beat rhythmically
2. nonneural embryonic m. " "

Beat originates near vena cava in rt. auricle

comparative anatomy
1. frogs - watch heat by cooling
2. Stannius' exp. - ligature - isolated heat - intrinsic rhythmicity in all (?) chambers of heart

3. Eel heart - sinus venosus - auricular canal between auricle & ventricle
human embryo has S.V. which (His) persists as S.A. node

4. Some claim in embryo ventricle starts beat before sinus venosus appeared (from rt sinus venosus cf. Baird)

5. AV node (from auricular canal)
6. AV bundle (only functional connection between two)
7. Evidence of SA origin of beat in mammalian adult

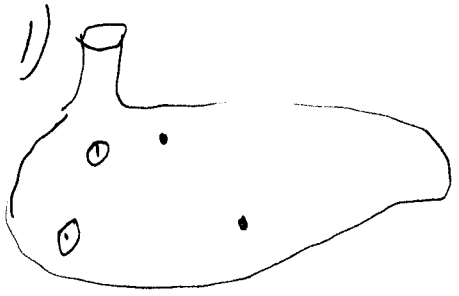
a. Erlanger cut heart into strips are found SA region beat fastest

b. isolation or removal of SA node caused heart slowing - got "nodal rhythm" starting in AV node

c. transplantation of SA node possible & still get it as pacemaker
cooling or heating of this region changes rhythm

d. electrical techniques - find pacemakers thus

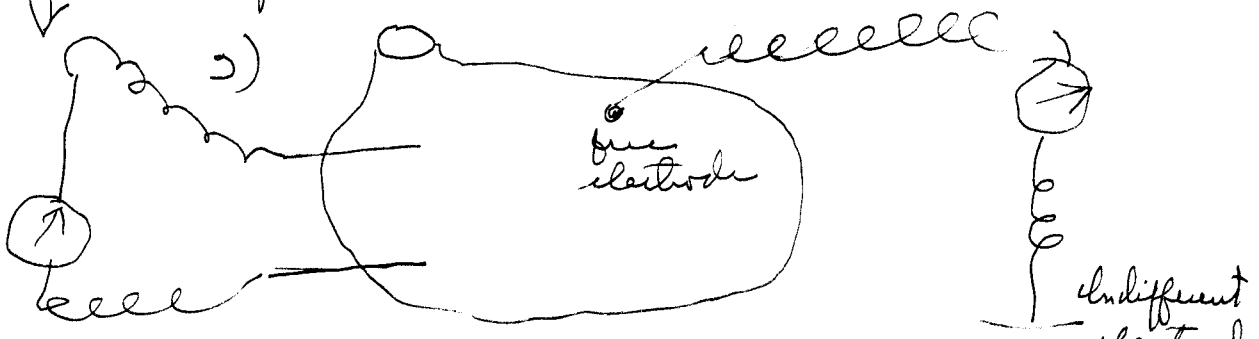
1)



Bipolar method

Reference electrogram

2)



e. normal sequence of events when SA node stimulated artificially
- subsidiary pacemakers when SA node destroyed

why originate in SA node
1. not continuous excitation with varying excitability
2. So must be rhythmical excitation
   1. build up of metabolites
   2. changes in pH
   3. hormone
   4. rhythmical electrical apparatus

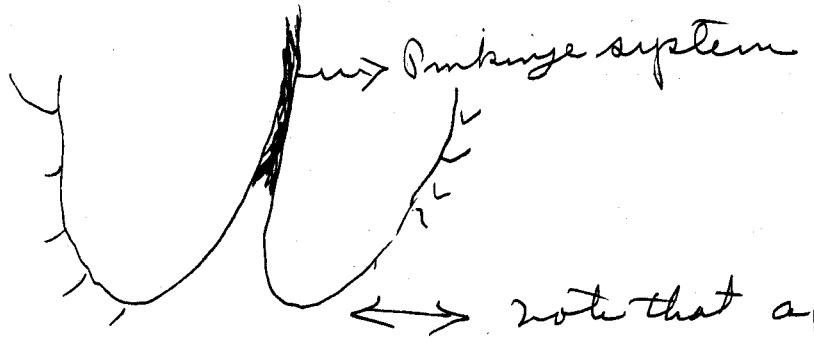
$\boxed{\text{just theories}}$

9/20/3

1. impulse spreads over auricle like wave on pond (some shorter & longer paths)
2. preferential path from SA to AV nodes
3. delay at AV node - fortuitous - "nodal delay" reason: sort of synapse - tortuous fibers also

Tissue	Speed of Conduction
Auricular muscle	600-1200 mm per sec
AV node	200-400 " " "
Purkinje System	3000-5000 " " "
Ventricular muscle (unspecialized)	400 (?) " " "
" wall	2400 " " "

Lewis originally thought endocardium-epicardium transmission very slow (cf. above) actually the Purkinje system ramifies throughout wall so that you get much faster figure above



note that apex beats first fortuitous

Electrocardiogram

String galvanometer usually used
Einthoven
silver coated wire in
electrical field
uniformity necessary

3 leads
I rt arm - lt. arm
II " " rt. leg
III lt. arm - lt. leg.
IV precordial lead - right over heart

in I rt. arm shows initial negativity

different ones emphasis different thing

less amplitude in P (obvious reason - less mass of muscle)

R negativity at apex of heart

Q & S similar - both near AV node - note anatomy

get auricular T wave under certain conditions but lost in QRS complex usually

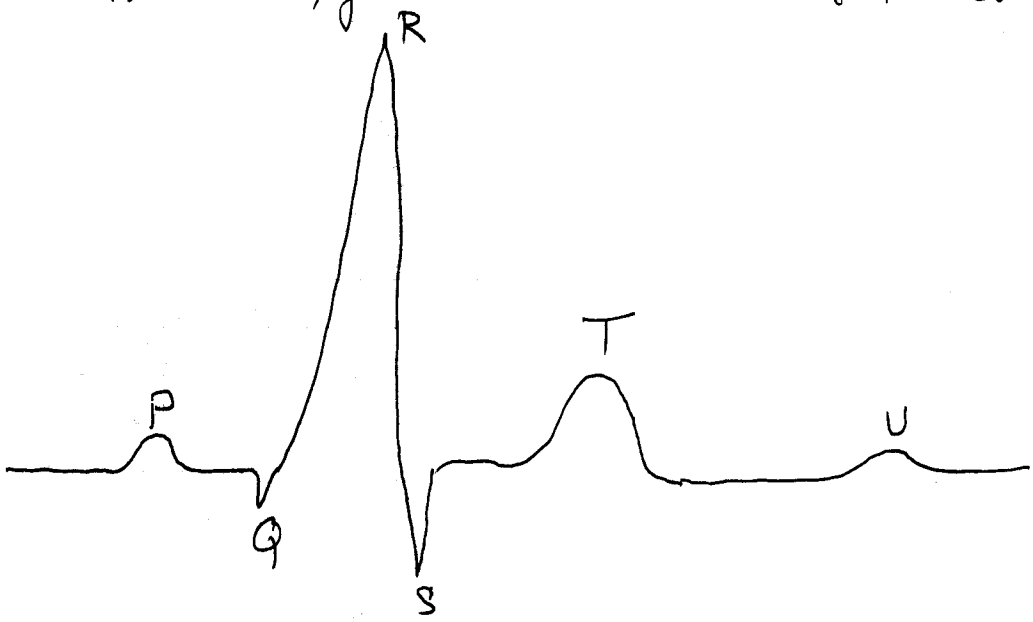
in dissociation of heart block the auricular T wave not masked

also can have patient swallow lead and thereby (close to auricle) get auricular T wave

9/20/4

Harvey, Wm "De Motu Cordis" - 1628 Trans. by Leake

Herrick, James B - "Short History of Cardiology



P = electrical activity in auricle
Q R S = ventricle
T = also ventricle recovery
u = hypersensitivity (varies in position)

Exeter and Meek

Erlanger

Slides

can have many degrees of heart block

Wave analysis

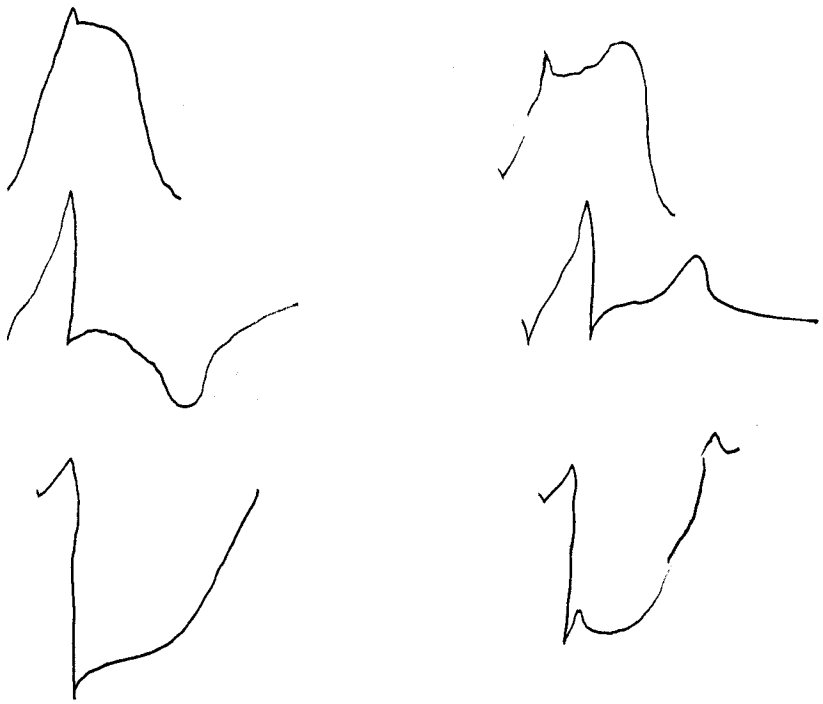
This got by cutting one bundle branch

A B

dextrocardiogram

electrocardiogram

levocardiogram



if right side of heart predominates T wave up
" left " " " " " down

9/22/1

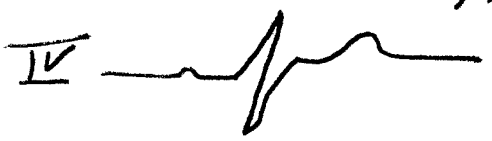
EKG = summation of electrical processes of heart

P = 0.08 sec
PR interval = 0.16 sec.
QRS = 0.08 sec
QT interval = 0.36 sec
ST interval = 0.08 sec.

T wave of P = Ta

Small R wave = weak heart

precordial wave (no Q, S large)

IV 

ST segment (level) may be above or below base line. below may be dangerous

if add lead I to lead III get lead II

extra systoles usually in region of V since hyperventilation

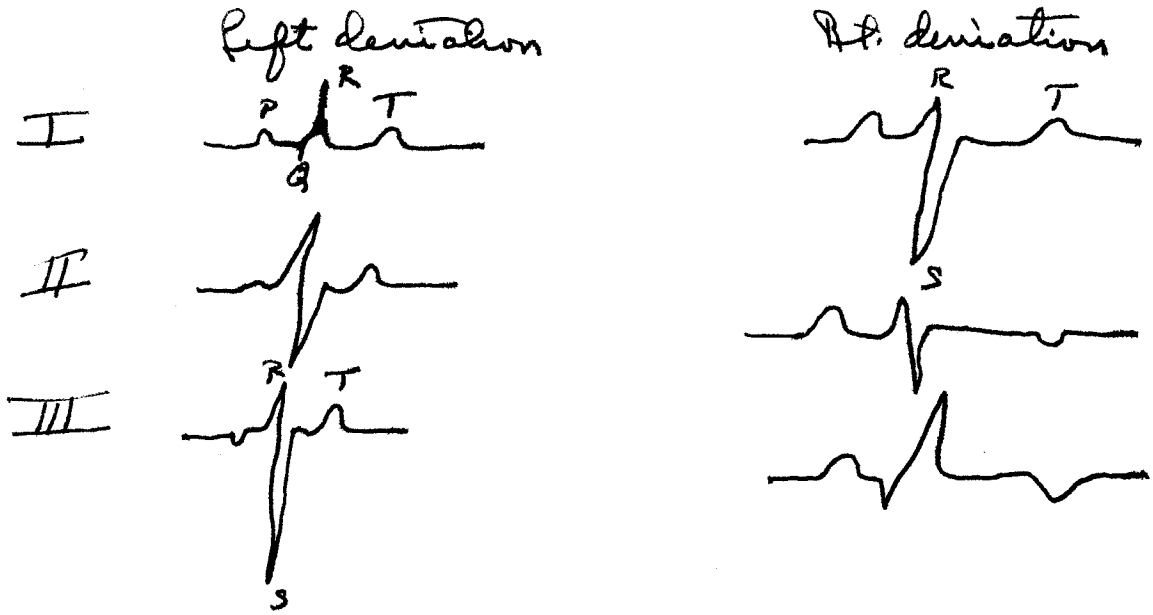
- clinical:
  1. surface injuries exaggerated by EKG
  2. not surrounded by uniform conductors
  Heart current transfers

Lateral heart surface	0%
Anterior " "	49%
Inferior " "	25%
Posterior " "	34%
Superior " "	27%

3. posture of individual gives variation since surfaces of ② changed

4. shape (contours of chest) changes position of heart

e.g. dextrorotation in narrow chests
& levorotation in broad chests



Einthoven's triangle – (mathematical analysis)
find electrical axis angle of heart in chest

also can lead three leads into machine where summated to form <u>vectocardiogram</u>

9/22/2

Easily picked up:

arrythmia or poor conduction

I Sinus arrhythmia

II Sinus tachycardia (if more extreme = paroxysmal tachycardia) slow down sensitivity of heart

PR = 0.12 sec
QRS = 0.06 "
QT = 0.24 "

III opposite of II = Bradycardia

IV Extrasystole or ectopic beat. — whether compensatory pause or not depends on position of interposition
if no pause no change in radial pulse but extra Ci in venous pulse

V AV nodal rhythm

P waves lags behind - may be behind QRS

VI Heart block
extra P's - lesion over AV bundle

VII Flutter or fibrillation of auricle

auricular flutter not too serious since auricular systole responsible for only about 8-24% of blood to ventricles dependent on conditions.

VIII Ventricular fibrillation

caused by many foci of stimulation

Serious because refractory periods don't cancel themselves out —

circus theory of fibrillation caused by slow conduction & short refractory periods

Mechanical events in Heart

Why blood not back into veins

1. ↓ of veins
2. heart originates near veins - milking action
3. pressure in ventricle less than in vein

	Systole	Rate	Diastole
auricle	0.10 sec	80/min	0.65
Ventricle	isometric cont. - 0.04 ejection phase - 0.21 0.25		isometric relaxation - 0.08 inflow 0.42 50

what closes auricular valves —

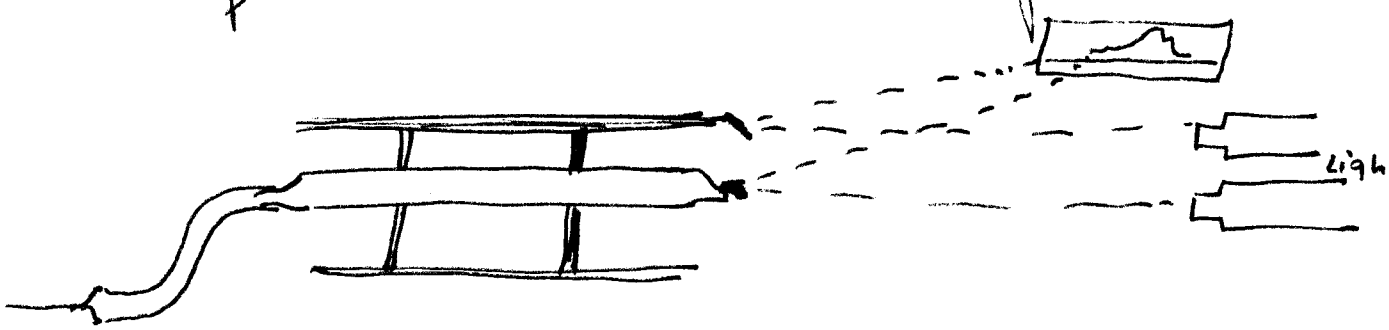
1. papillary muscles some
2. eddy currents

9/22/3

Period of isometric contraction when both valves closed & no blood ejected until 80mm of aortic diastolic pressure reached.

also period of isometric relaxation before ~~a~~ ~~H~~ valve opens when pressure attains about 10 mm.

heart sound ~~of~~ to determine ventricular pressure

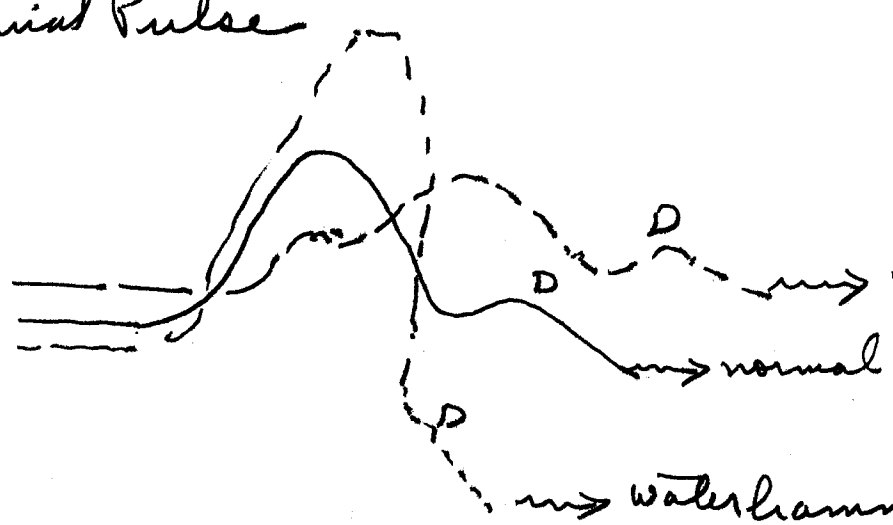


velocity of pulse in less elastic arteries is greater

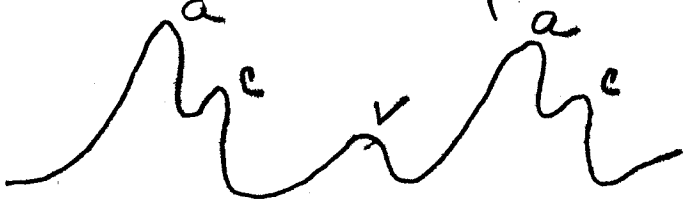
age in years	10	20	30	40	50	60	80
mean velocity (m/sec)	5.55	6.2	6.75	7.20	7.6	8.0	8.55
mean elasticity	0.41	0.33	0.28	0.24	0.22	0.22	0.17

Blood Pressure	Brachial artery - velocity in meters/sec. Inspiration	Expiration
84/45	3.4	3.8
100/55	4.3	4.6
140/65	5.3	5.4

Arterial Pulse



Venous Pulse (represents pressure changes in auricle) same as by sound in auricle



A = auricular contraction
C = ventricular contraction (pressure of blood on AV valves)
V = filling of auricle

thus By carotid and jugular pulse can tell

auricle (A)
ventricle (C)
AV valves (V)
aortic valves (Dicrotic notch) D

if numerous a's few c's - heart block -

Stethoscope
ours - amplify low frequency sounds
Cardiopean metal tubes amplifies high frequency sounds

9/22/14

heart sounds

lub dub or lub-dup

closure of AV
contraction of ventricle
eddy currents in aorta

closing of aortic valves

a third sound (20% in diseased) - flow of blood into empty ventricle as AV valves open

fourth sound (A) contraction of auricle
usually included in first sound
exaggerated in large auricle

Best information on valve conditions

AV valve leakage gives murmur
[initial insufficiency] running over into second sound
first called systolic murmur

diastolic murmur = musical aortic murmur = insufficiency of aortic valves.