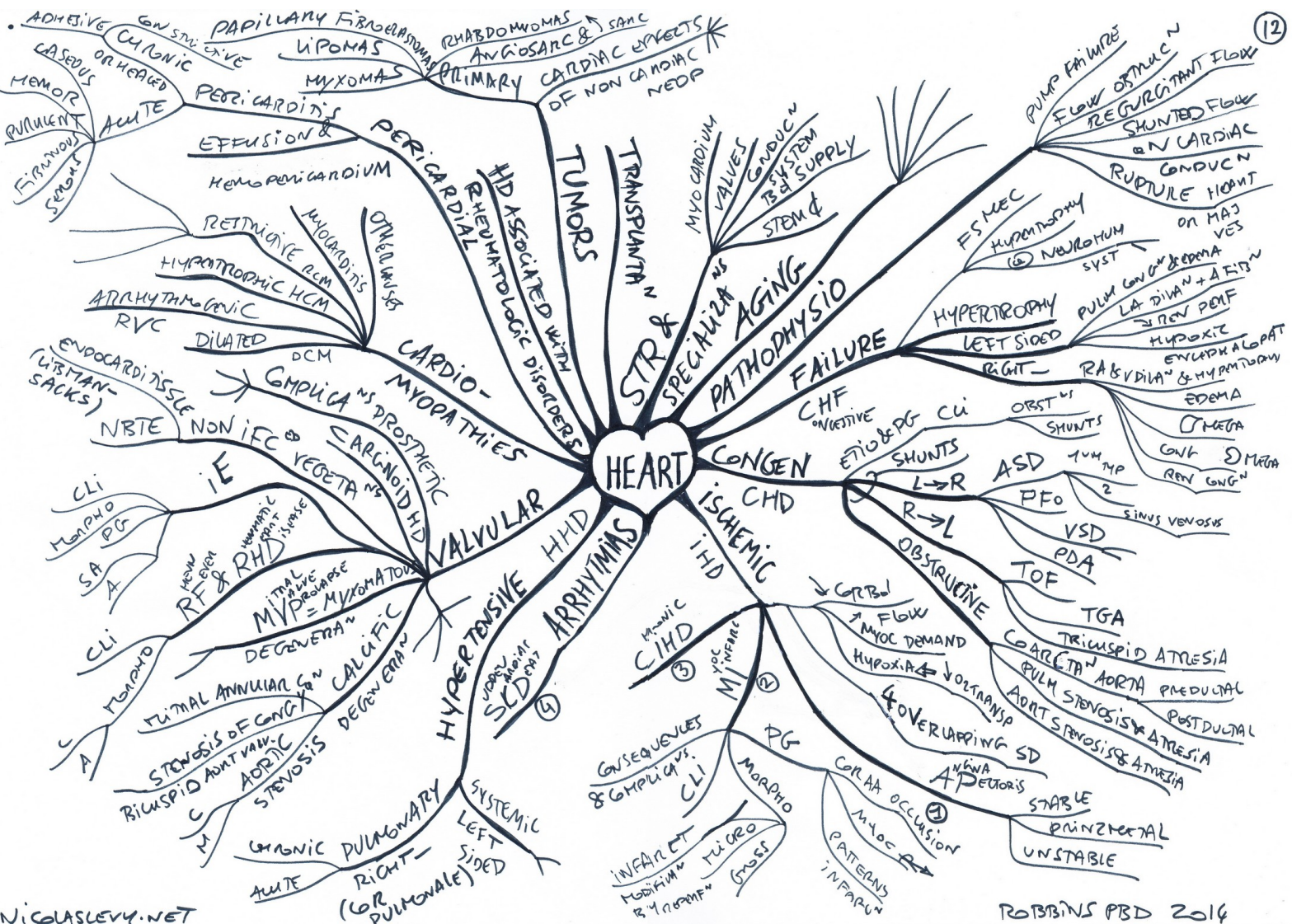
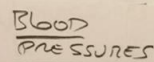
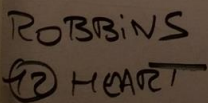
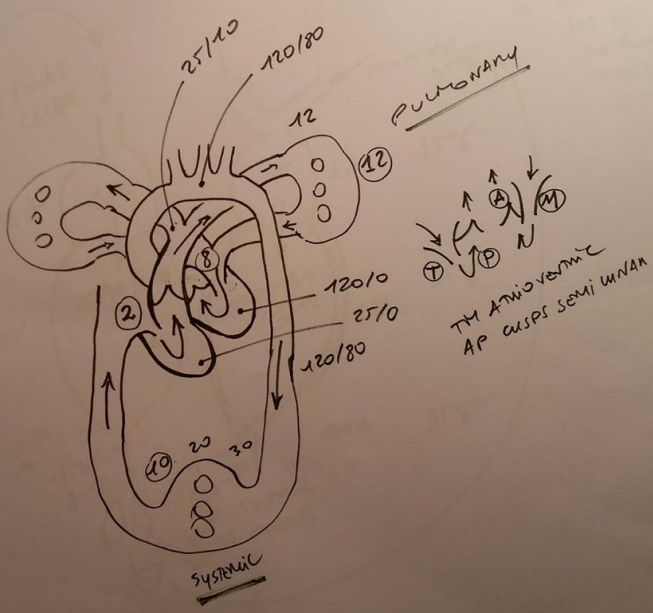
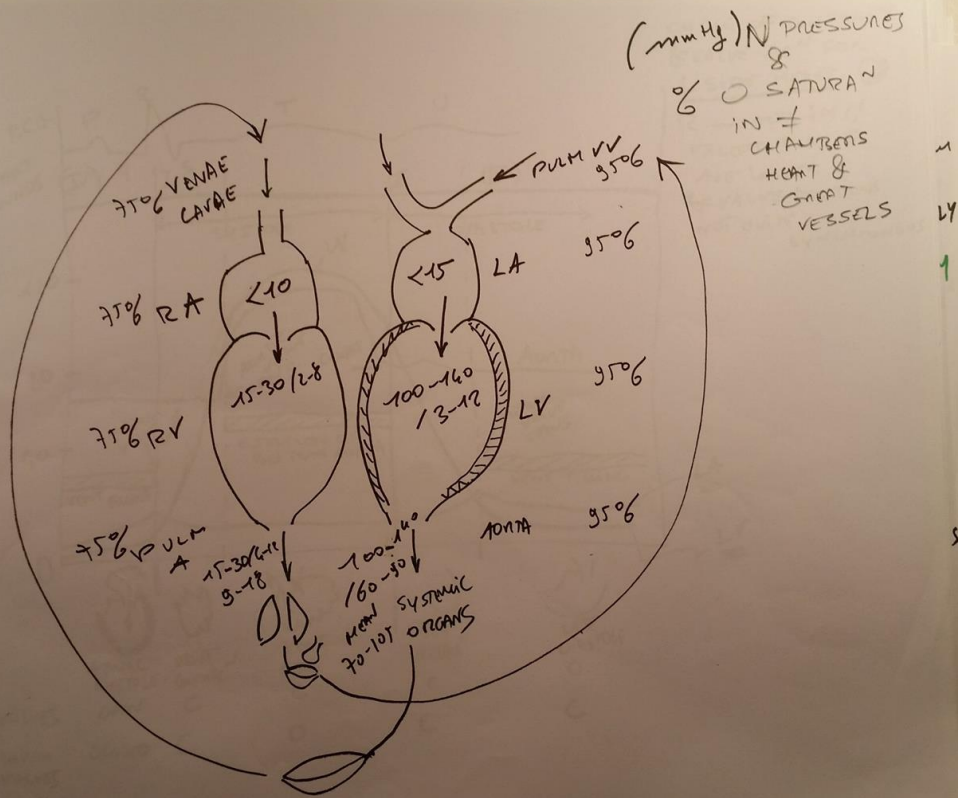


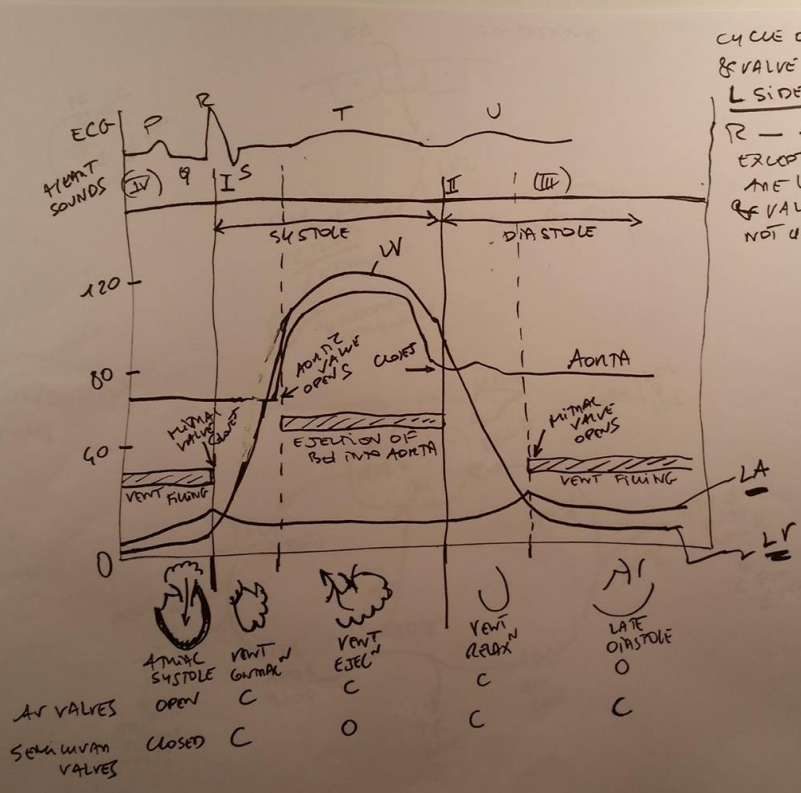




Blood Pressures

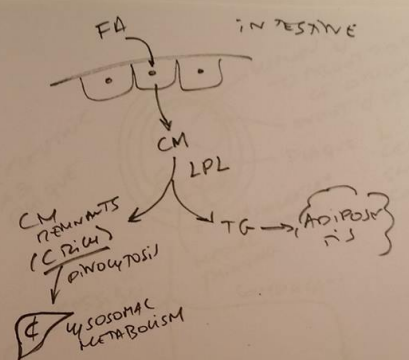
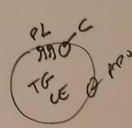




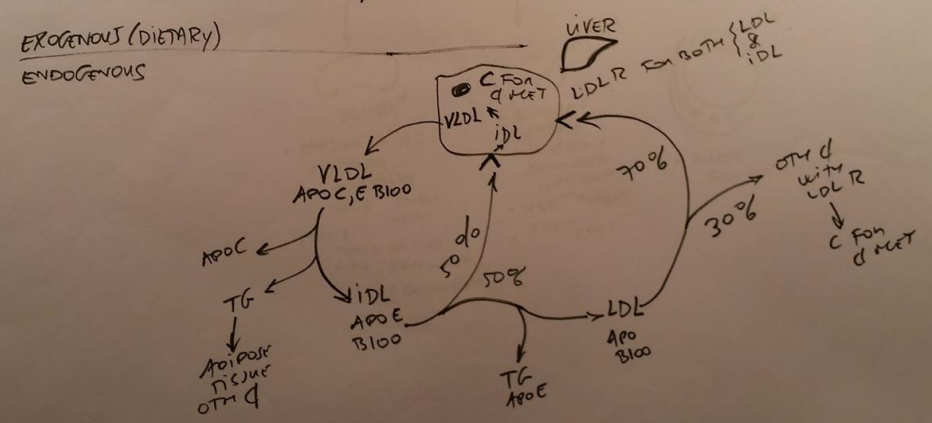


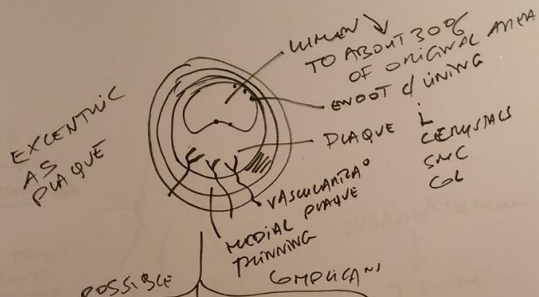
CYCLE OF CONTRACTION & VALVE ACTION FOR L SIDE HEART.

R - ACTS IN // EXCEPT THAT P ARE LOWER & VALVE MOTIONS NOT QUITE SYNCHRONOUS

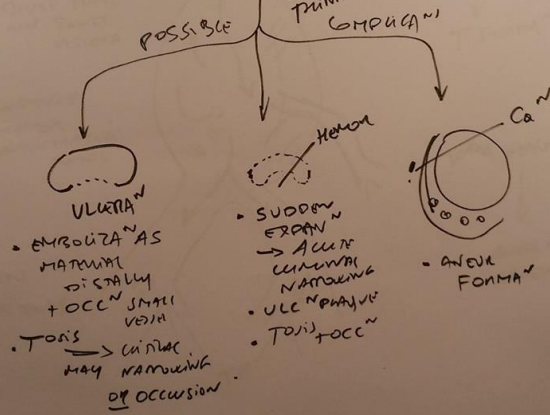


NORMAL
METABOLIC
PATHWAYS
LIPID





COMPLICATIONS AS

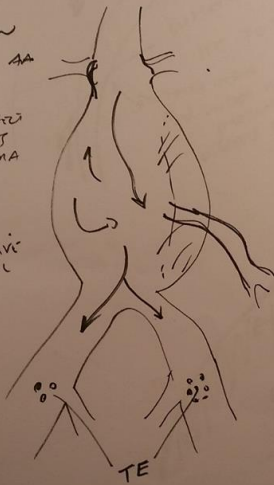


• NARROWING
ON OCCLUSION
RENAL & MESENT AA

• PRESSURE EFFECTS
ON NN BOWEL
VISCERA

• RUPTURE
HEMORR
OFTEN MASSIVE
FATAL

• TE IN
LEG ARTERIES



TURBULENT Bld FLOW

↓
T FORMATION

WALL WEAKENED

↓
ANOMALY
RARELY RUPTURE

← SAME
AS
USUALY ABD
AORTA

CLINICAL
EFFECTS
OF AAAA
 $\approx 8.0 \text{ cm}$
 $\approx 5 \text{ mm}$

Atheros

MC TYPE

AORTIC

ANEUR

↓

SYMPHIC

THORACIC

AORTIC
DISSEC^N

UNION ADATA
70%

HT 70%

INTIMAL TEAR
BD UNDER HIGH
PRESSURE
DISSECTS WARD

AC TEAM
UNDER HIGH
PRESSURE
DISSEMINATING MEDIA

OBSTRUCTIVE

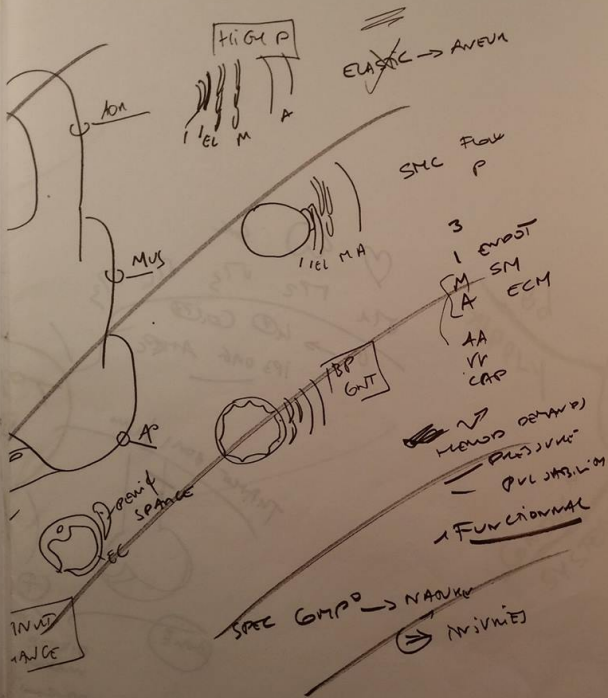
1. GROW
REVAL &
MUTANT
ICC

→

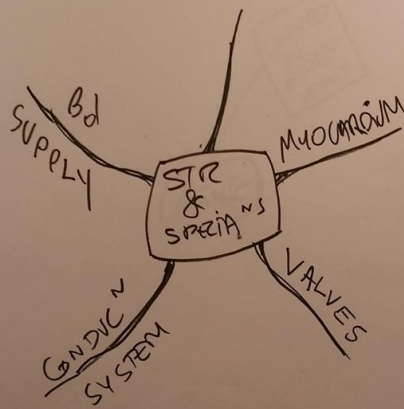
RIP INTO
HYPHAST
ON RETROPERIT
(FATAL)

rupt
into
penic
→ tamponade

NOT BANK
INT UNDER
(DOUBLE BANNER
ADDTA)



KEY CONCEPTS



173
TO
④
Gm
dino?
mimo?
nemo?
mover

my
aly
ing

s
n

2019 5/8 3/4

TO
i

⊕
Gm
direct
indirect
parent
never

AGING

Changes
in
AGING
HEPOT

my
aly
my

S

11

2014 50132014

TO

⊕

direct
indirect
referred
muscle

PATHO
PHYSIO

run

ALY

ing

S

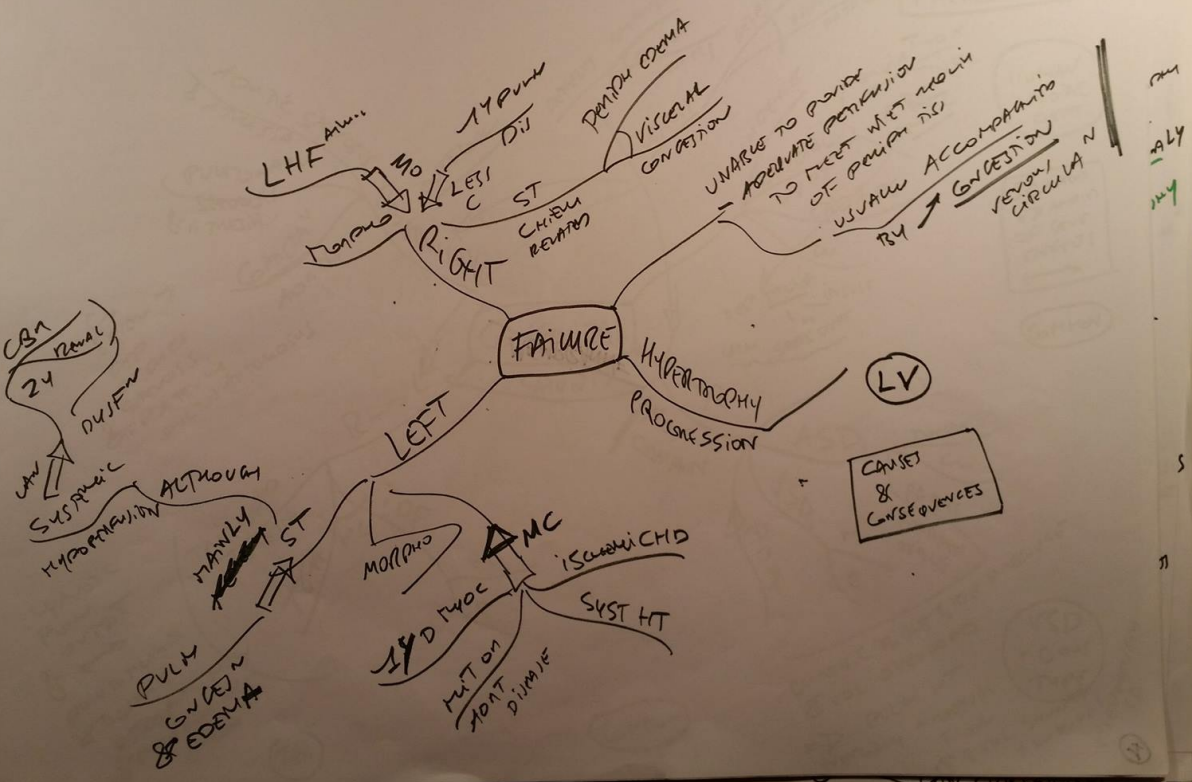
n

LOW SUBSCAP

W. CONZELMANN

Heart Failure

TO
④
Gm
directly
indirectly
rarely
never



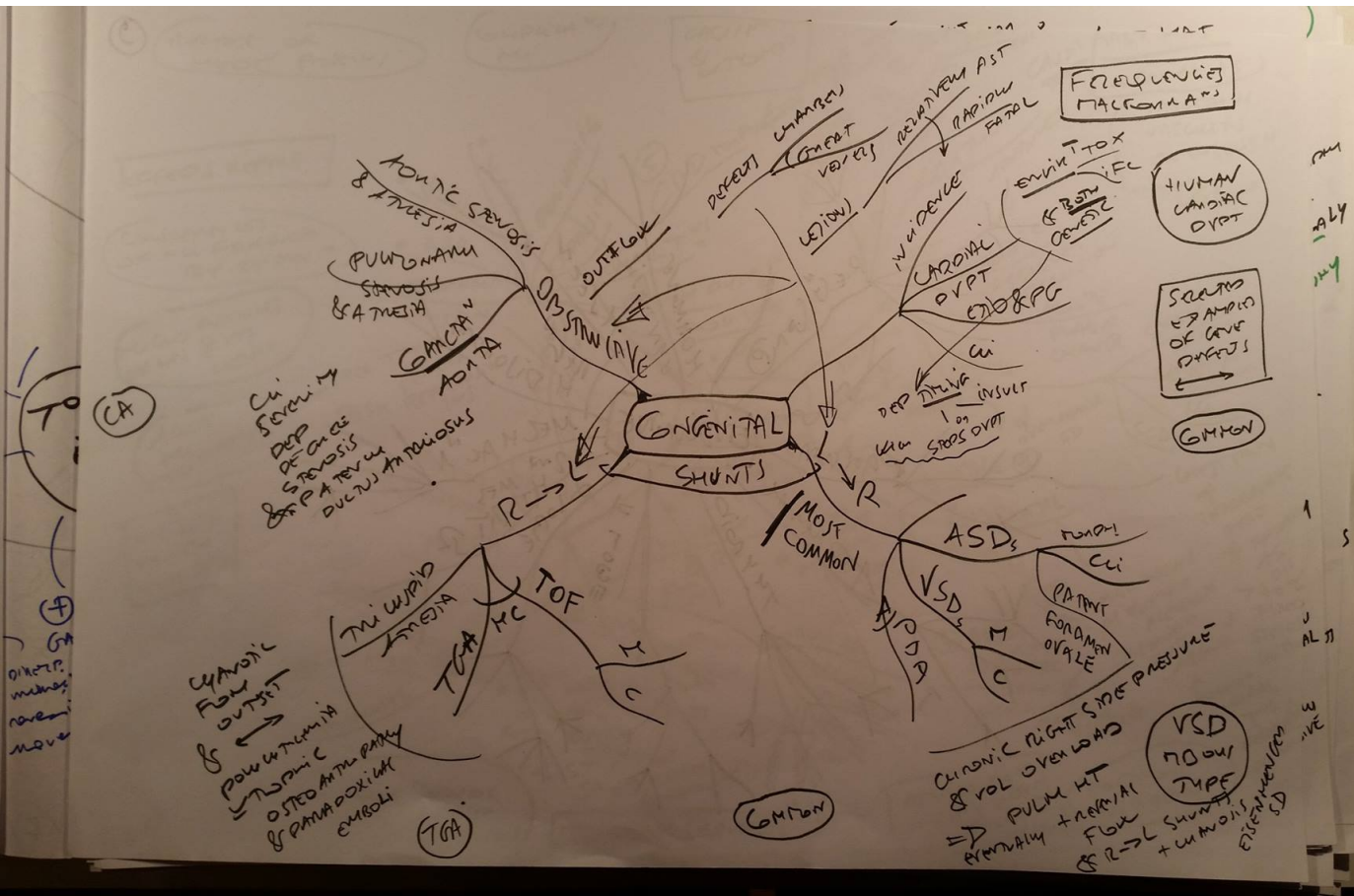
may
may
may

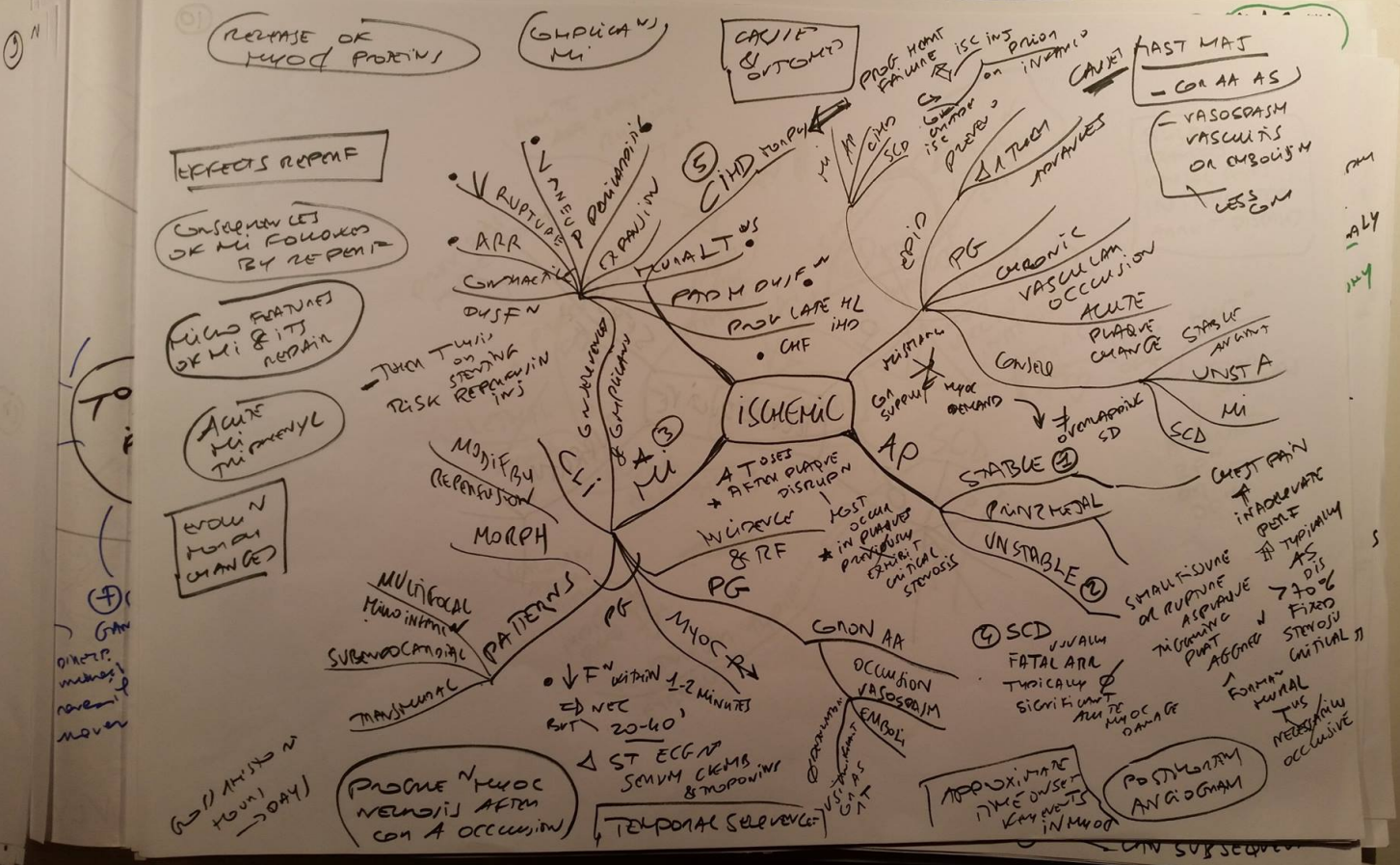
S

11

③

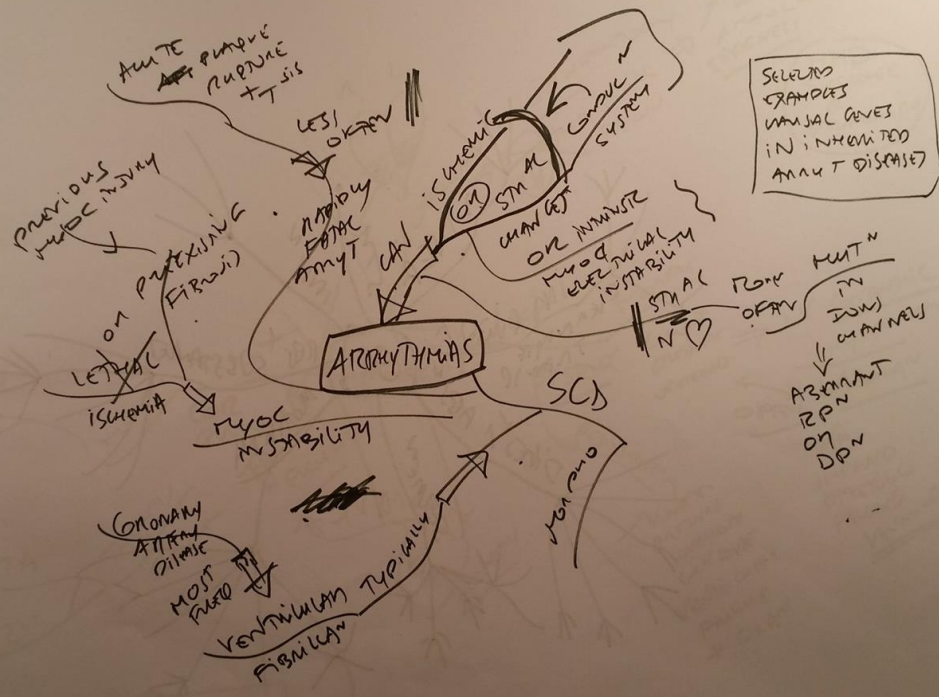
ON SUBSEQUENT



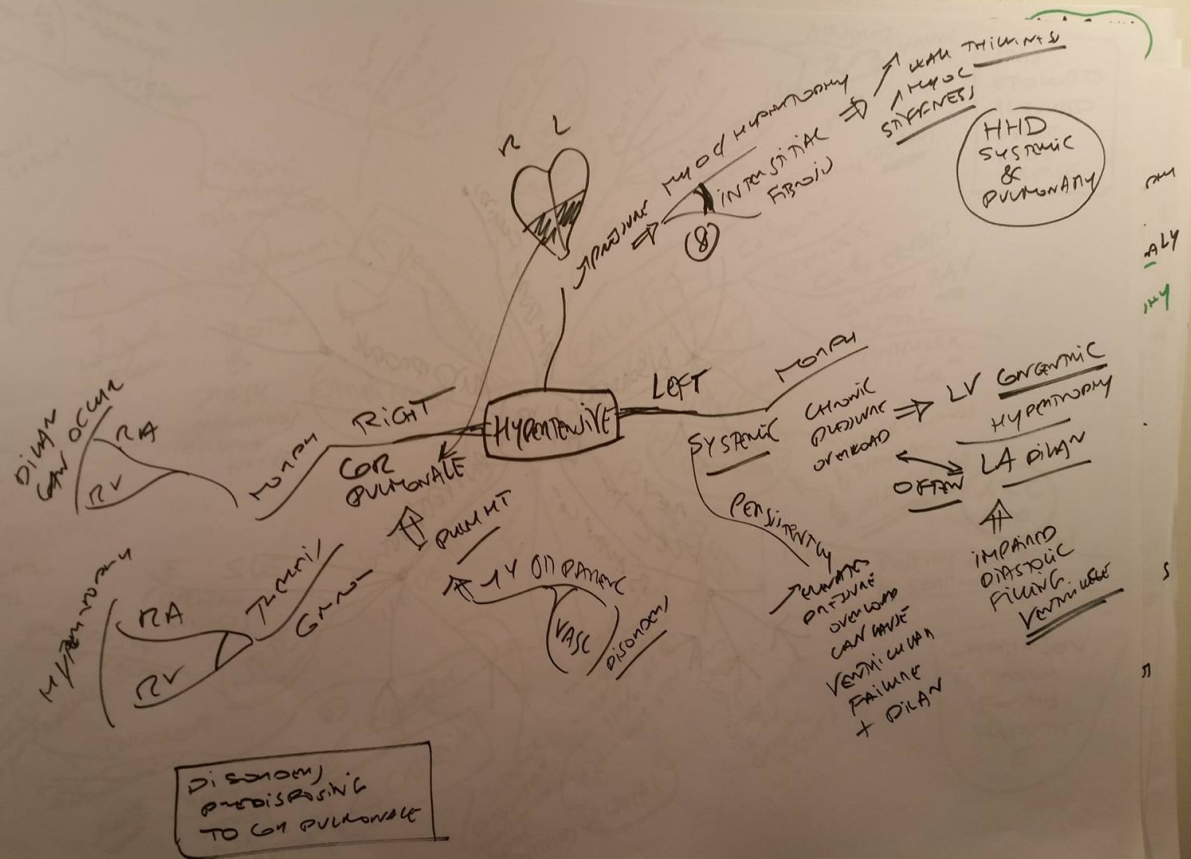


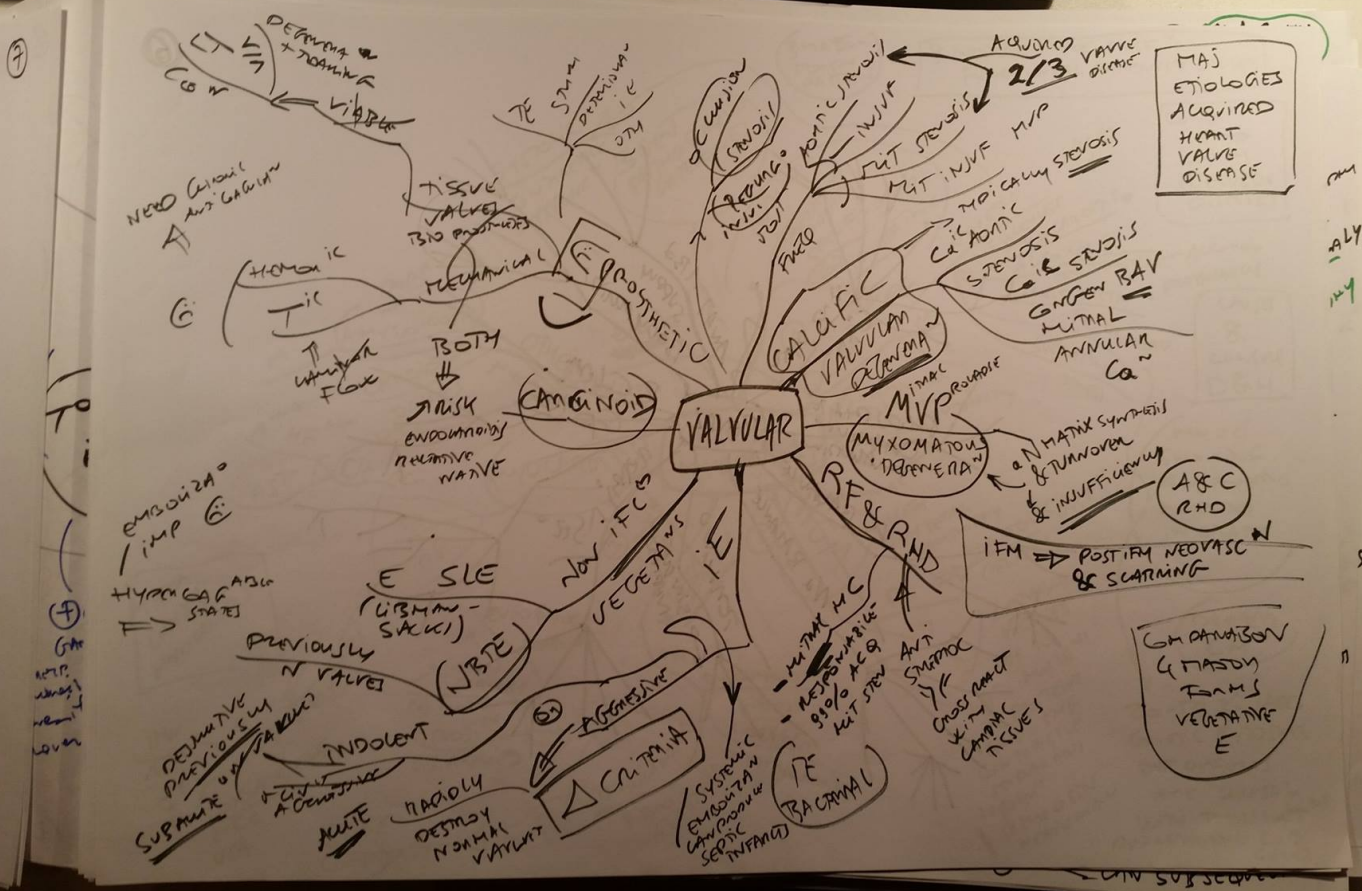
10

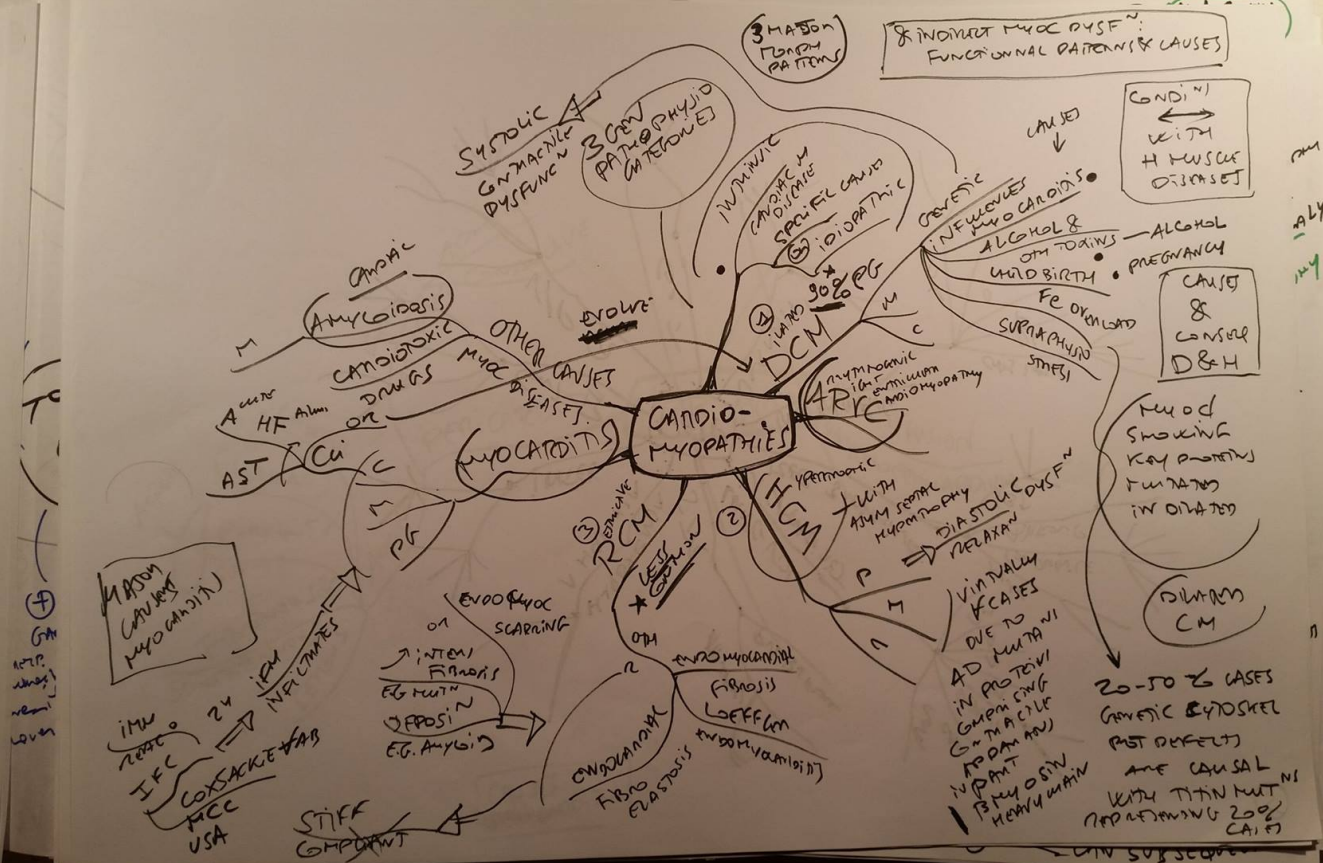
3

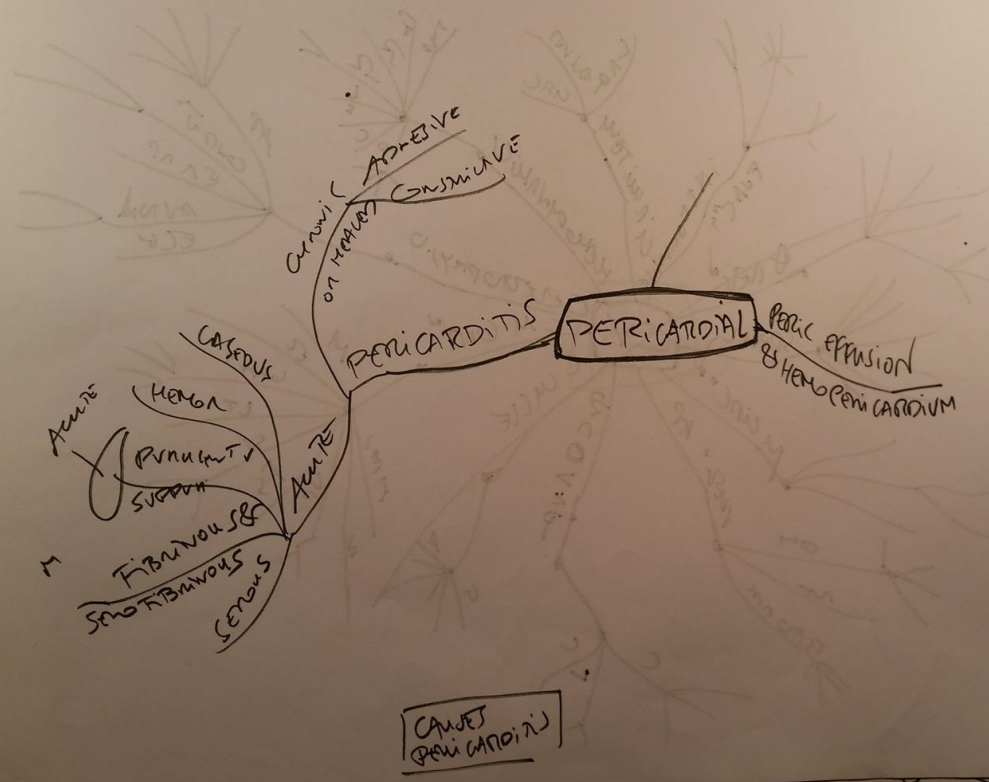


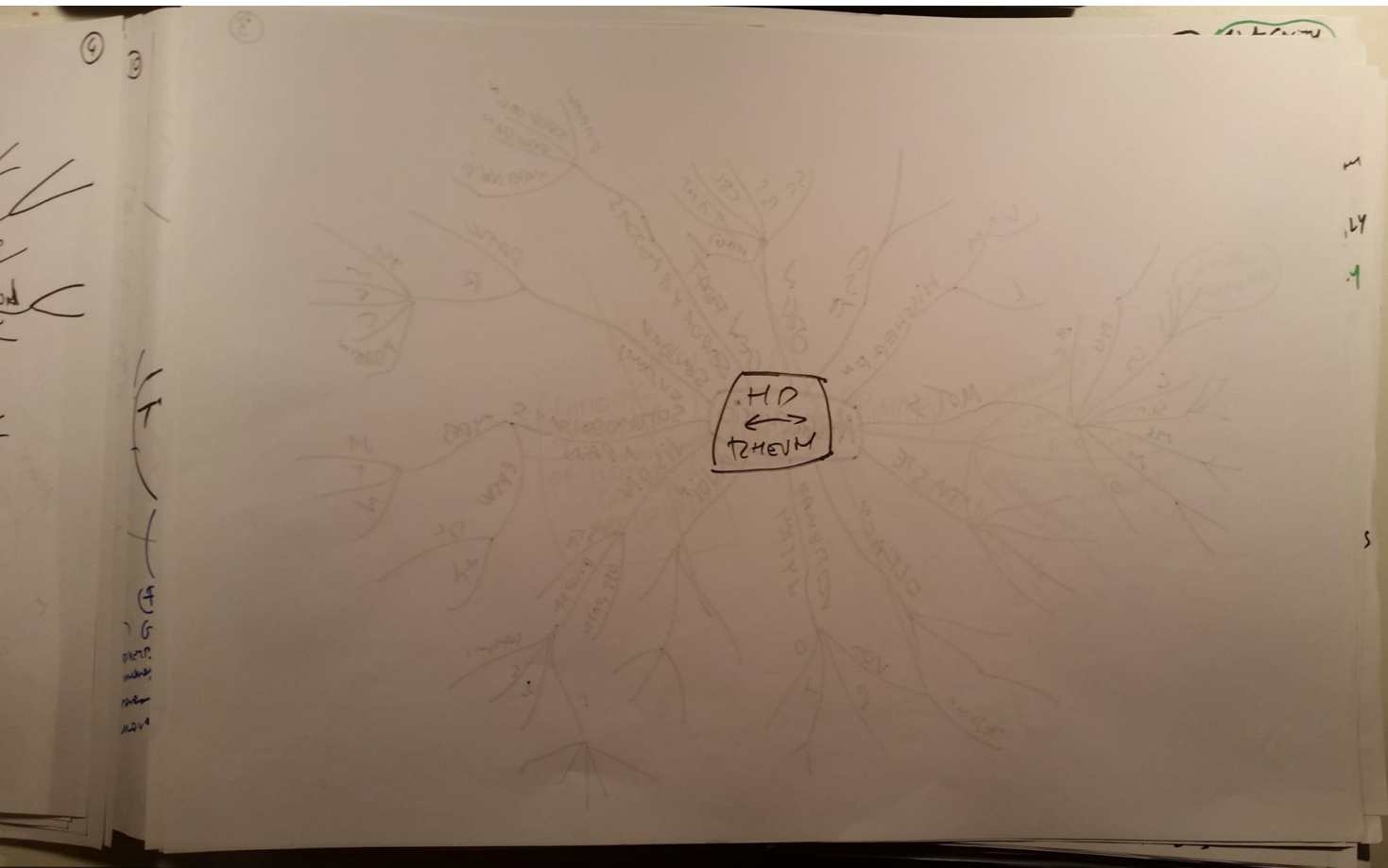
⊕
Gm
not p
unusual
normal
never



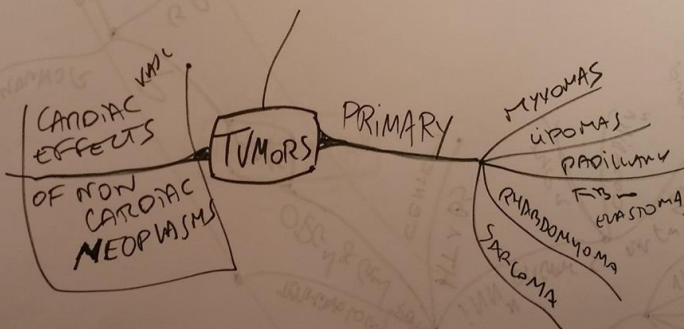








③))



ATMIL
HYXOMA

COMPLICATIONS

TRANSPLANT

TO

⊕
Graft
rejection
infection
malignancy
renal failure
hypertension
diabetes
heart disease
stroke
cancer

Myocardium

Mal
biology
Failure
Disease

1 DAY

300 yr

ALL DEATHS

THINNESS

PREJUVILE

100 A

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

100

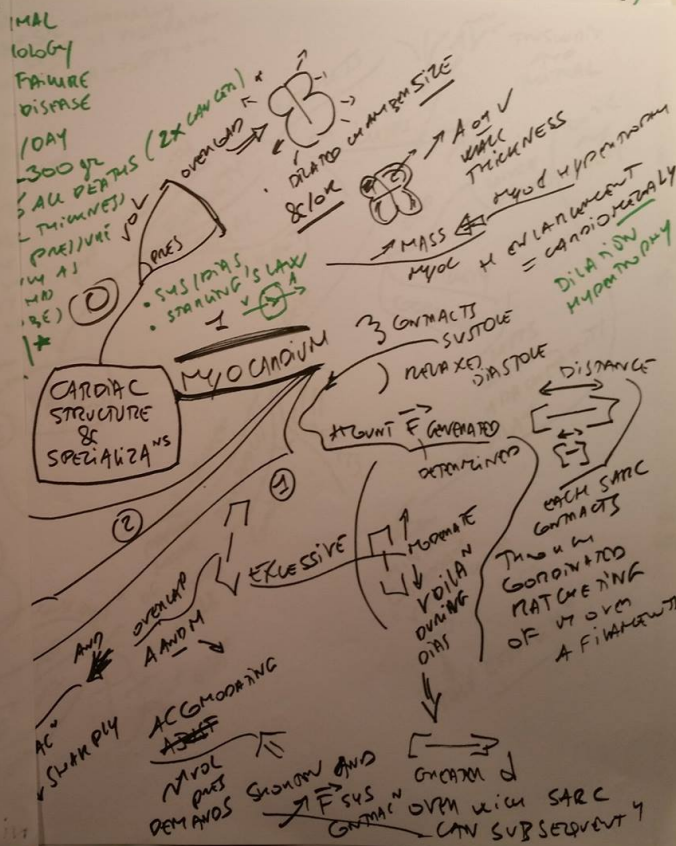
100

100

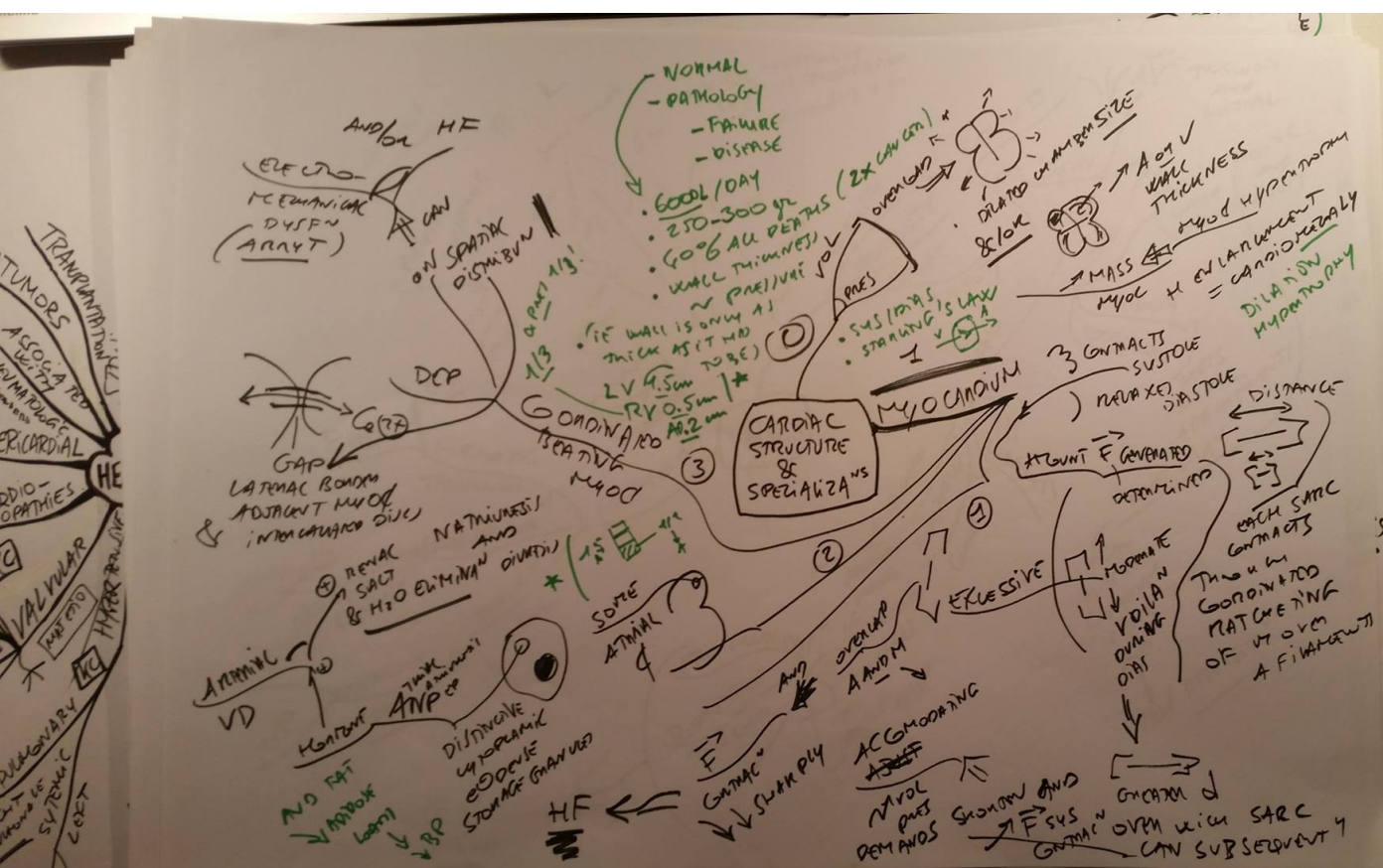
100

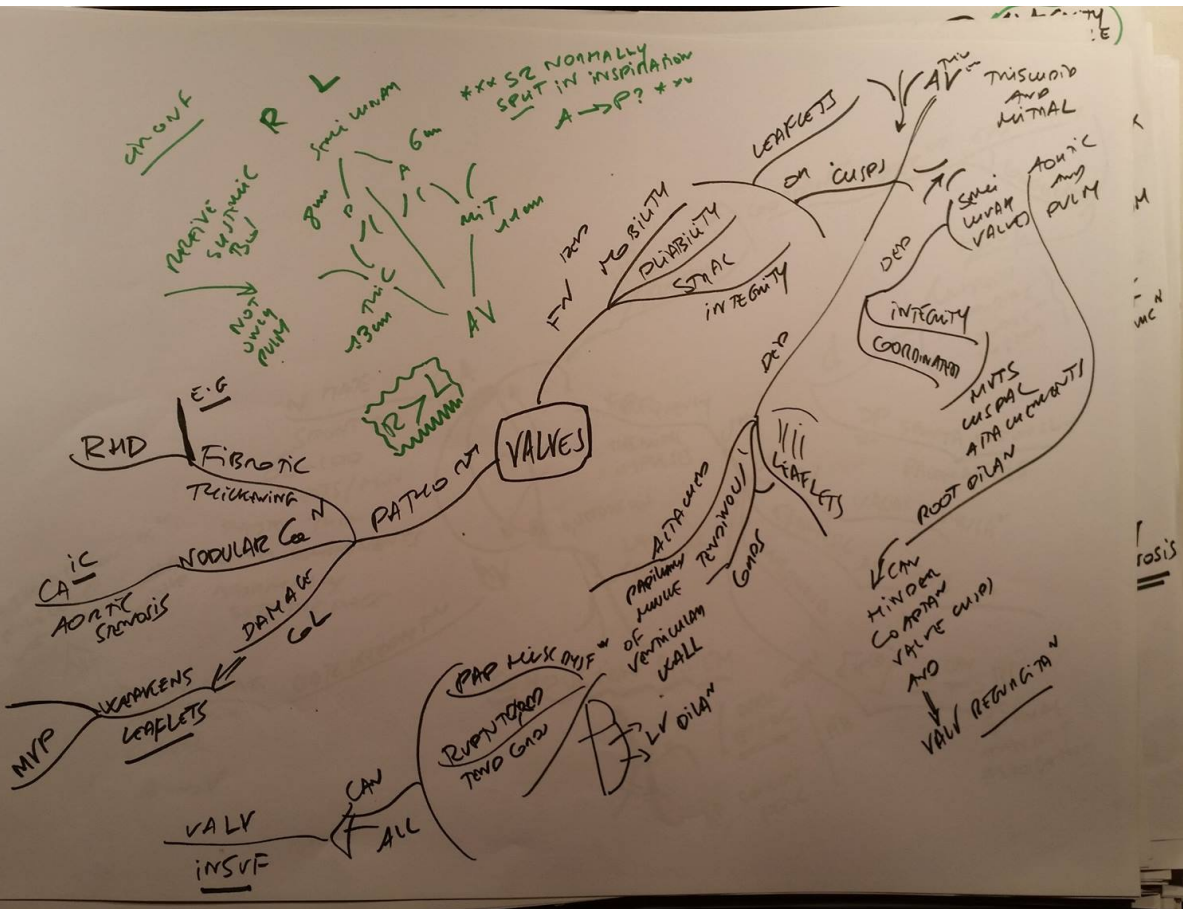
100

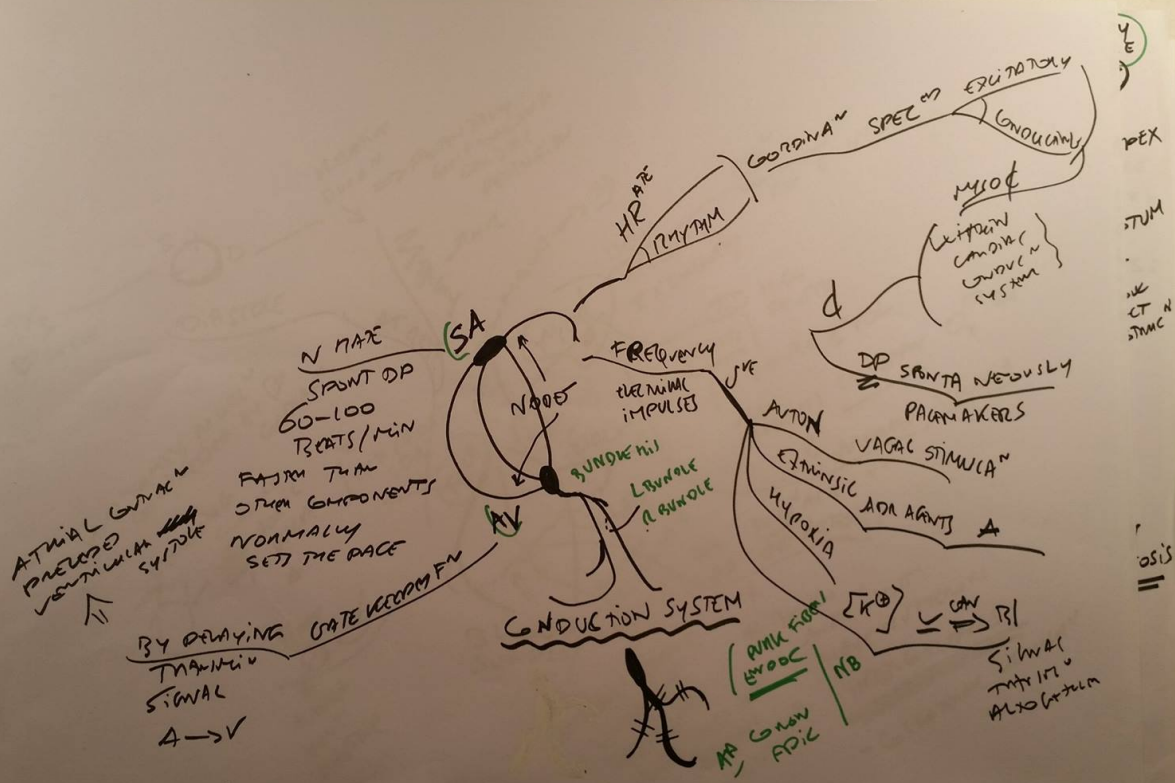
100

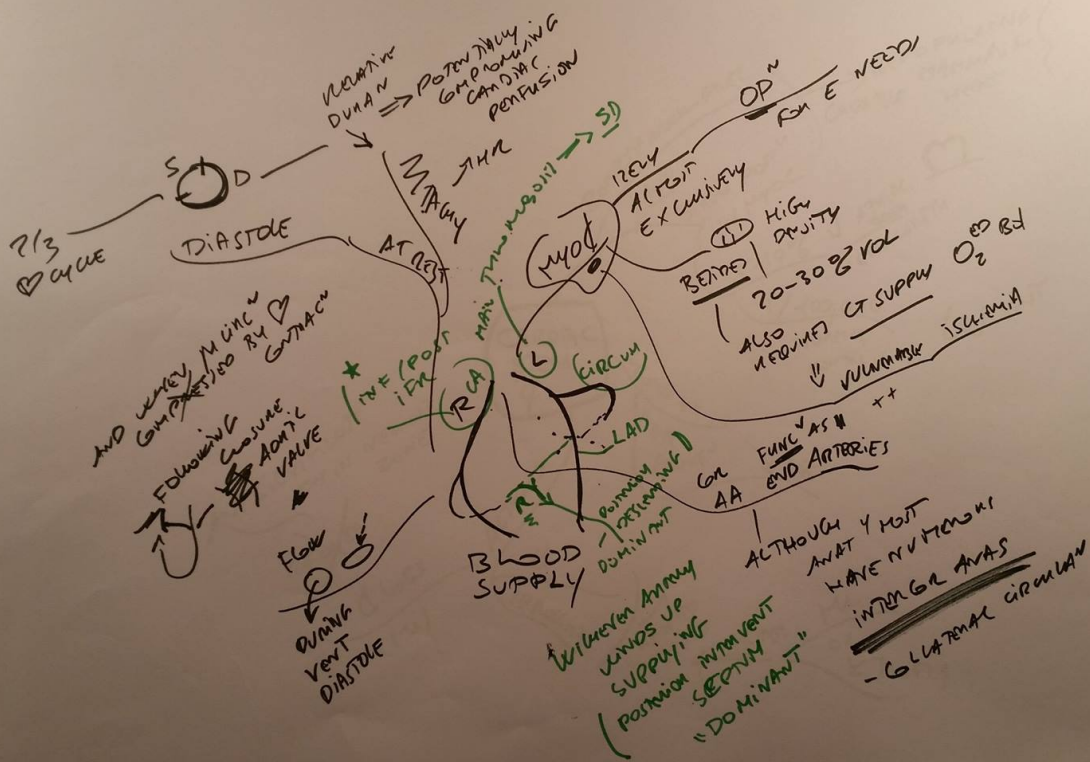


STRUCTURE & SPECIALIZATION

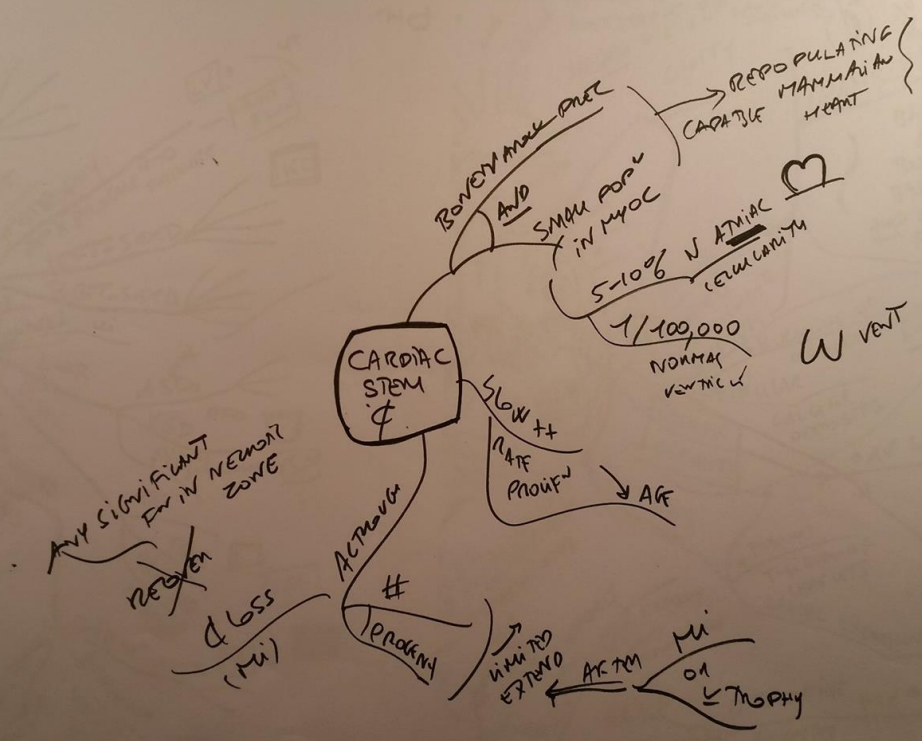








HYPER...
NOT...
NEW

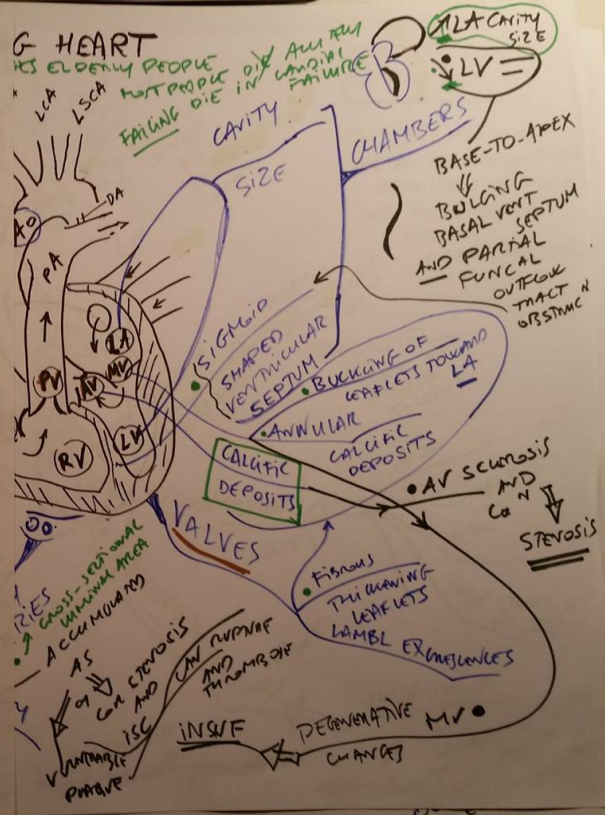


SIZE

APEX
T
PTUM
L
L
ZONE
ACT
STIMC

S
↓
VOSIS

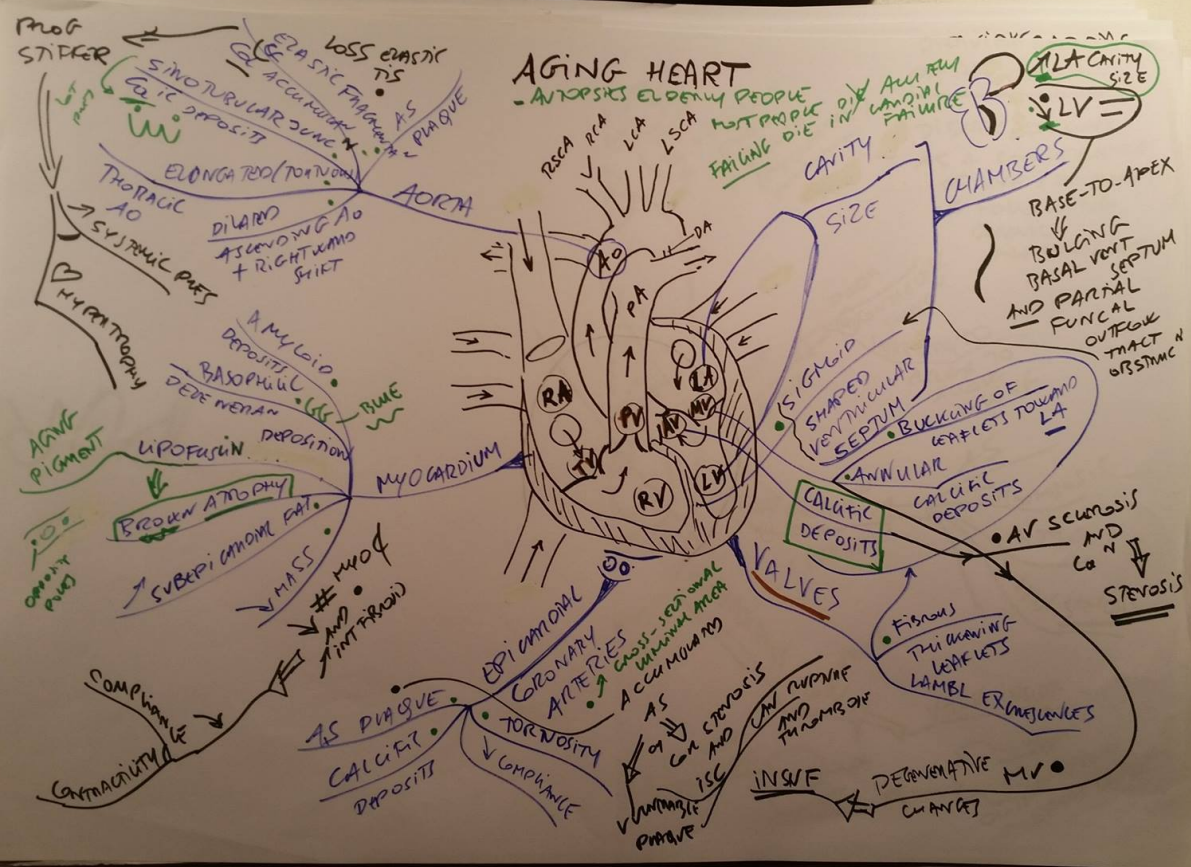
PHARMACOLOGY OF THE HEART
PERIPHERAL



AGING

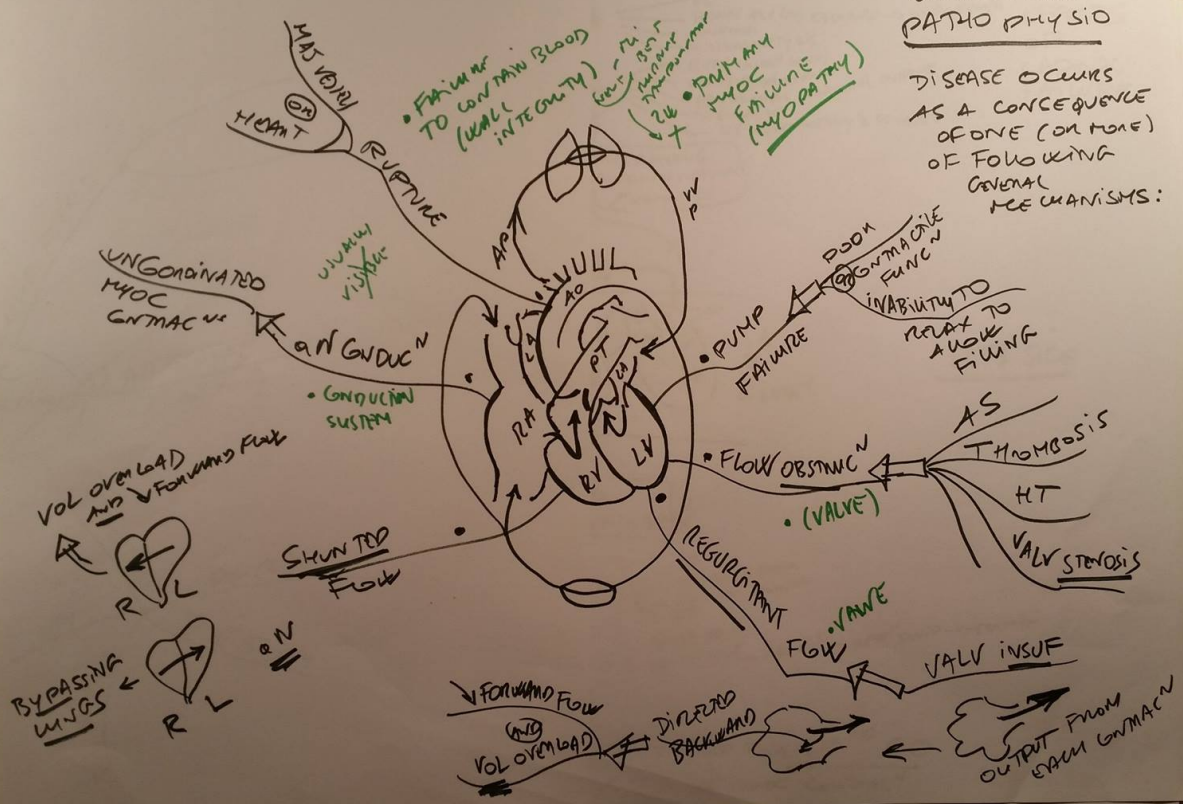
PURINES

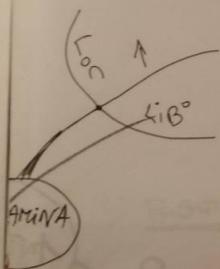
SEN.



OVERVIEW CARDIAC PATHO PHYSIO

DISEASE OCCURS AS A CONSEQUENCE OF ONE (OR MORE) OF FOLLOWING GENERAL MECHANISMS:





HF

- A — PE
- Failure ALV gas exchange → HYPOXEMIA
- S3
- HEMOPTYSIS
- C — Cxrd congestion
- S4
- S3
- PULMONARY HYPERTENSION
- PULMONARY EDEMA
- RV HYPERTROPHY & FAILURE

EFFECTS
OF
CARDIAC
FAILURE

COMPENSATORY PUMPING
OUT OF VENOUS RETURN



LEFT SIDE

↓ CO

- A — SD
- Cerebral shock
- Myocardial failure
- HT

C — ↓ NS PRF

RENAL VC → RETENTION → RETENTION FLUID → EDEMA

COMPENSATORY MECHANISM FOR ↓ CO

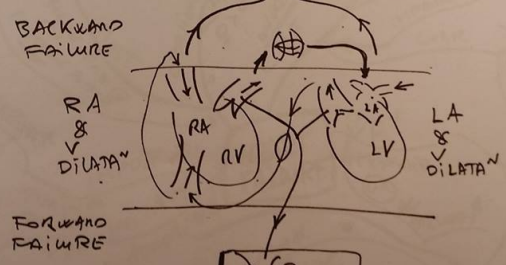
IN → CO → OF MYOCARDIUM

PARTIAL STENOSIS
 JVP
 BACKWARD FLOW
 STENOSIS (C) VENTRICLE
 EDEMA LEGS

PE
 FAILURE ALV GAS EXCHANGE $\rightarrow$ HYPOXEMIA
 CYANOSIS
 HEMOPTYSIS
 CRYSTAL CRYSTALLINITY
 EVIDENCE IN CLINICAL
 PULMONARY HYPERTENSION
 PULM HT
 RV HYPERTROPHY & FAILURE

EFFECTS OF CARDIAC FAILURE

INCOMPLETE PUMPING OUT OF VENTRICULAR VOLUME



RIGHT SIDE

LEFT SIDE

CO
ANTRIAL BARRAGE

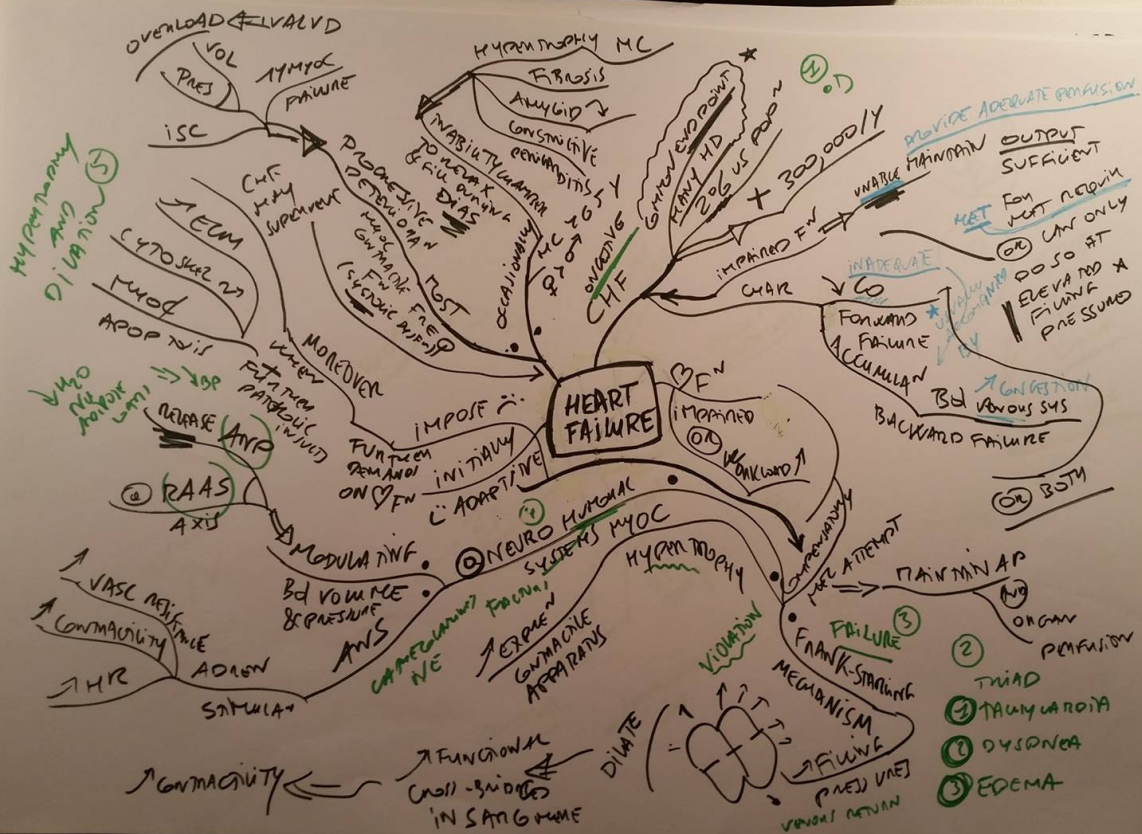
SD
 A
 SD
 ANGIOGENIC SHOCK
 MYOCARDIAL FAILURE
 AT

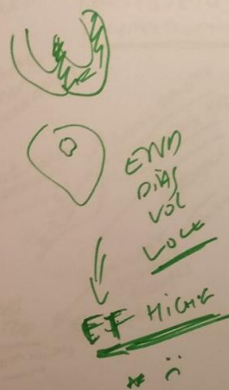
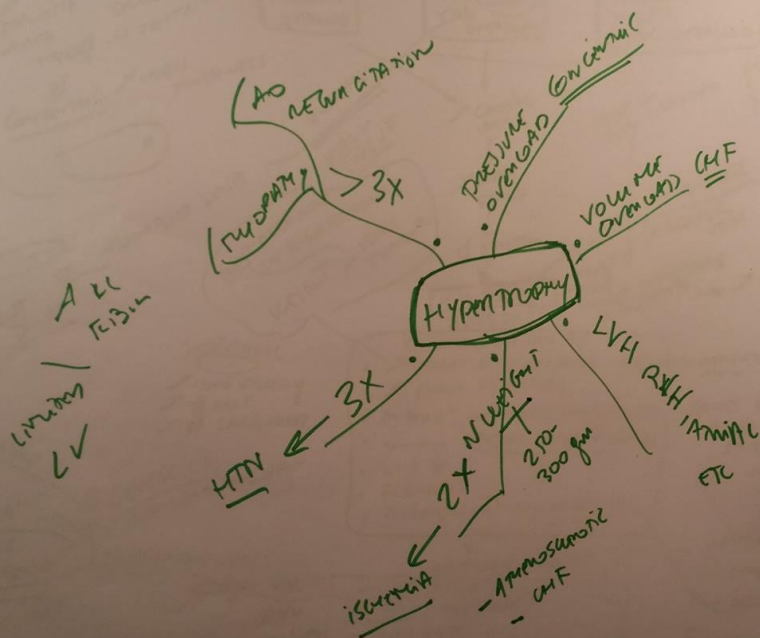
DOWNWARD
 PULM HT
 MINIMAL EFFECT

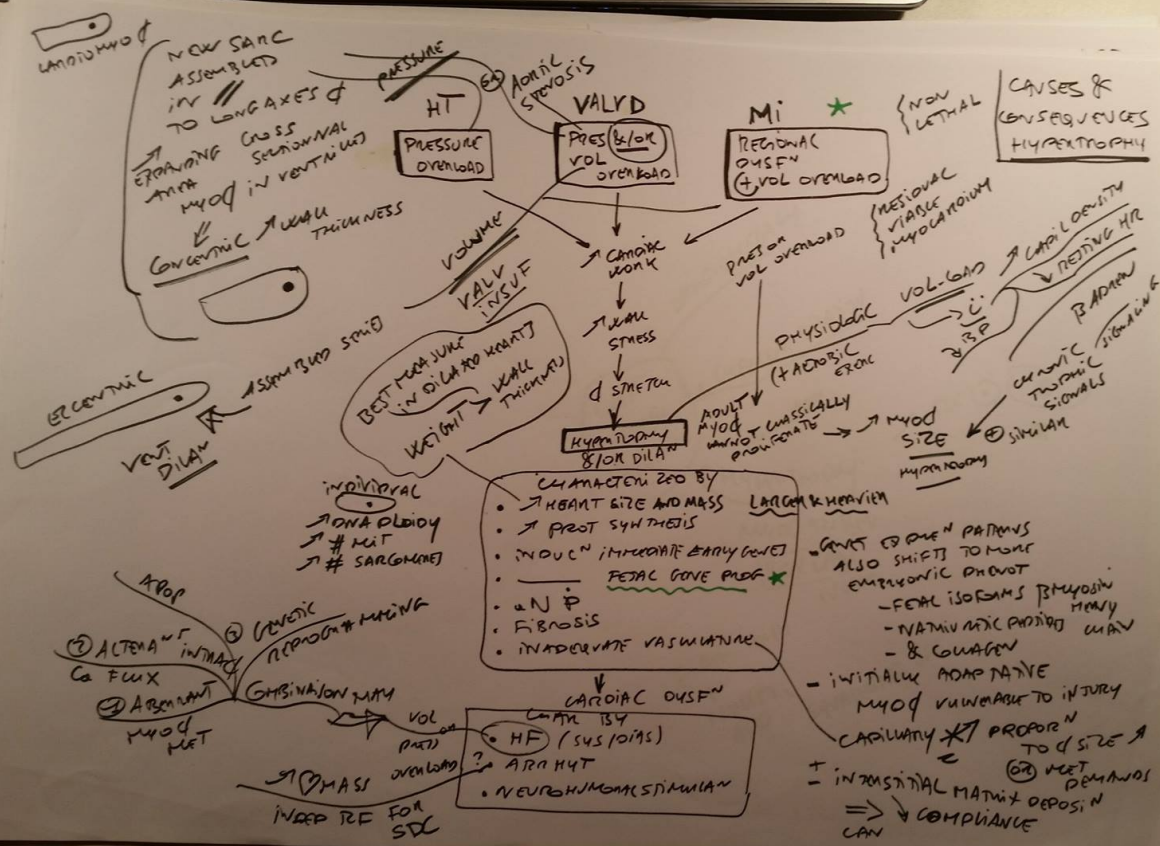
C
 RENAL VC $\rightarrow$ RENIN $\rightarrow$ ANGIOTENSIN $\rightarrow$ EDEMA

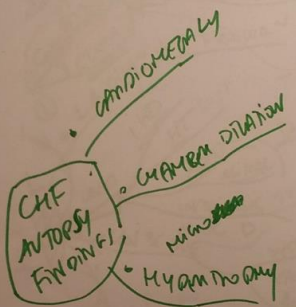
A = ACUTE
 C = CHRONIC

- DILATN PARTLY REPRESENTS A COMPENSATORY MECH FOR $\rightarrow$ ING CO
- STRETCHING MYOC FIBERS RESULTS IN $\rightarrow$ EFF OF MYOC CONTRACTN



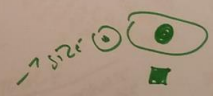






MYO FIBERS
BOX CAR
MULTI

FIBER WITH
+ NUCLEI
ALSO SQUARING OFF



MORPHOLOGY

HEMOGLOBIN STORE
IRON H₂O PHAGOS
MACROPHAGES
DIFFUSING BY MAC

3 TERTIAL SIGN
PREVIOUS EPISODE OF PE

COMMON SITE
STASIS BY ↑ THROMBUS
FORMATION
IN A. TRUNCUS
2Y DILAT
LA

IMPAIRED LV
FV
HILUS
NON-SPECIFIC
HYPO-
TENSIVE

SOME WROTE
EXTRACELLULAR
SPACE
WITHIN
ALV. SPACES
LUNGS
PULM
CONGESTION
EDOMA
LA DILAT + A FIBROSIS
MC
IHD
HT
SUST
ADIC
MIMAL
INTRINSIC TV
MYOC D

HEART
DEPENDENT
DISEASE
PROCESS
INTRINSIC
PATHO
VALVES
DETERM
STEN
MI
ETIOLOG
FOR
MYOCARDIAL
UNUSUAL RCM
USUALLY HYPERTROPHIC
OFTEN DILATED SOMEHOW
MASSIVELY

ALTHOUGH
SYSTEMIC HYPOTENSION
REGURGITANT FLOW
IMPAIRED PULM OUTFLOW

2Y CAN
REFLOW
POOR FLOW
SACCH H₂O RETEN
↑ Ki
PREFRONTAL ATROPHIA

RA @
NATRIURETIC
PEPS

LEFT
SIDED
HEART
FAILURE

HEAVY
DVT
PAIN RASC
& INTERC
EDOMA
ACUMUL
EDOMA
WIDENING
IN ALV
SERIES ALV SEPTA
SERVITY
PAIN
INTERC
SEPTA
KIDNEY
B&C
LINES
CHEST XRAY
CHT

SYSTOLIC
FIBRIL - INADEQUATE
CONTRACTILE
FV
DIASTOLIC
L
POOR FLOW

KEY CONCEPTS

STs 14 PULMONARY

- DYSPNOEA *
 - ON EXERCISE
 - AT REST → SLEEP
- ORTHOPNOEA *
 - REDISTRIBUTION OF PULMONARY FLUID
 - GRADED BY NUMBER OF PILLOWS NEEDED
- PND ^{ANNOYING} ^{ACTUAL} ^{SYMPTOM} *
 - OR SUDDEN

REVIEW

RD AND ~~SPUTUM~~ SPUTUM

CYANOSIS

ELEVATED PULMONARY "WEDGE" PRESSURE

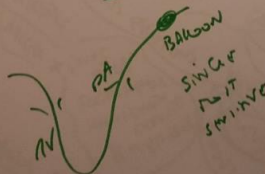
PCWP / N = 2-15 mm Hg

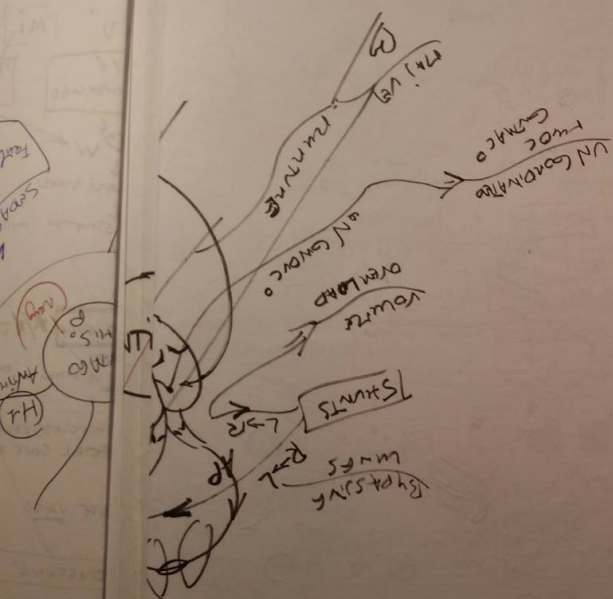
WIDENING

✓ ~~WIDENING~~ 14 PULMONARY

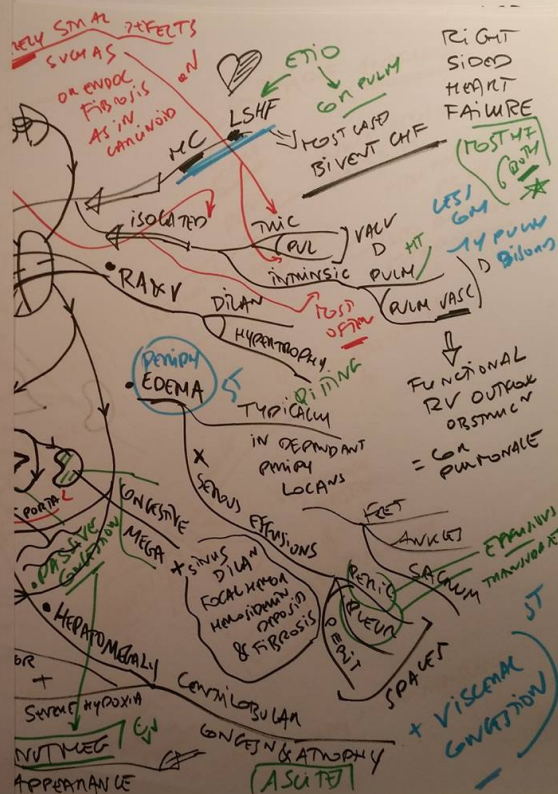
PCWP

SERIAL-GANT
OR THERM
FOR
MONITORING





RIGHT-SIDED HF



RIGHT
SIDED
HEART
FAILURE

POST-HEART
FAILURE

LESS
GAIN
14 PULM
BILION

FUNCTIONAL
RV OUTFLOW
OBSTRUCTION
= CON PULMONARY

FEET
ANKLES
EDHEMIA
TRANSUDATES

ST
+ VISCERAL
CONGESTION

SPACER

RENAL
PULM
HEART

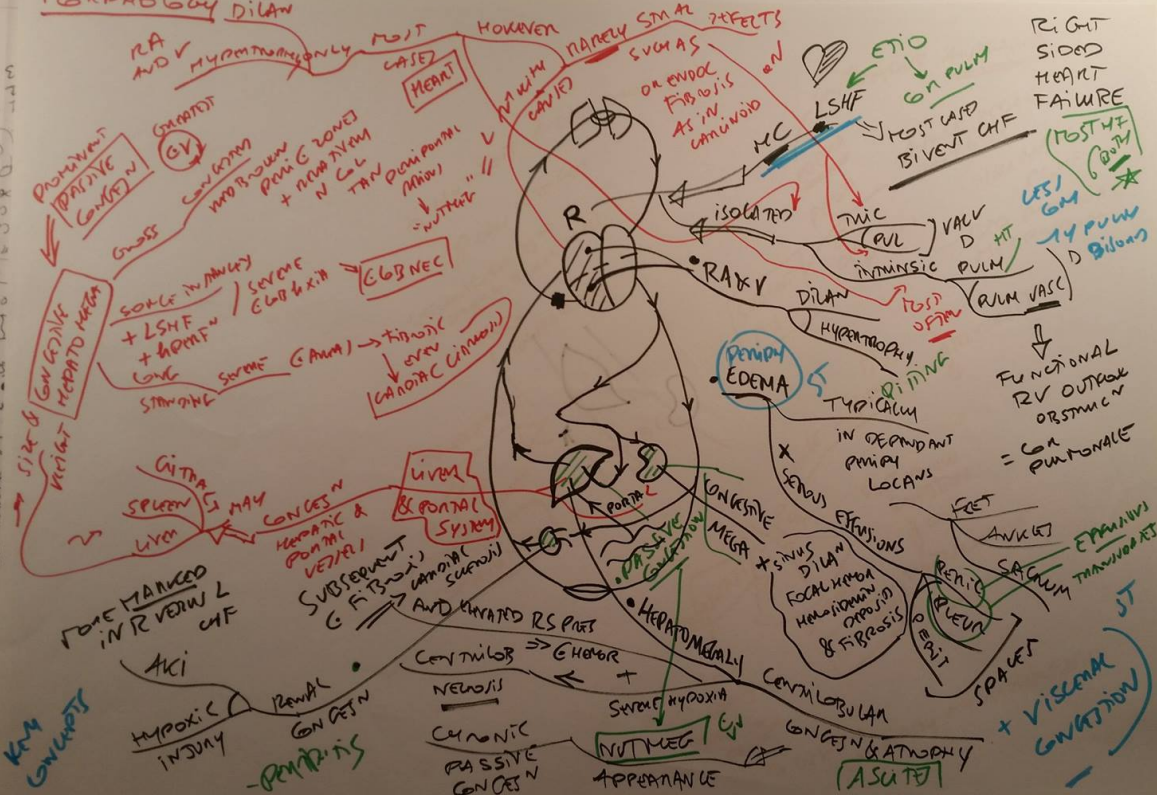
CONGESTION
& ATROPHY
(ASUET)

CONGESTION
& ATROPHY
(ASUET)

CONGESTION
& ATROPHY
(ASUET)

CONGESTION
& ATROPHY
(ASUET)

MORPHOLOGY Dilan



→ Δ system in money market basis

FATIGUE
"DE PAIN DE L'ART" EDEMA
JVD
HYPOTAT METAL (CONVULSION)
ACUTE PLEURAL EFFUSION
G
CYANOSIS
LXO
PVP
(CVP) N 2-5 mmHg

"DIPLOMAT" CODENA

HYDRATE METAL (CONCENTRATION)

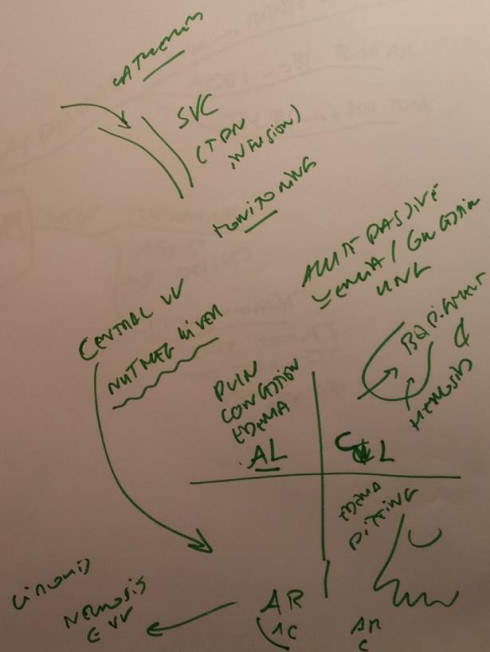
As the plume expansion

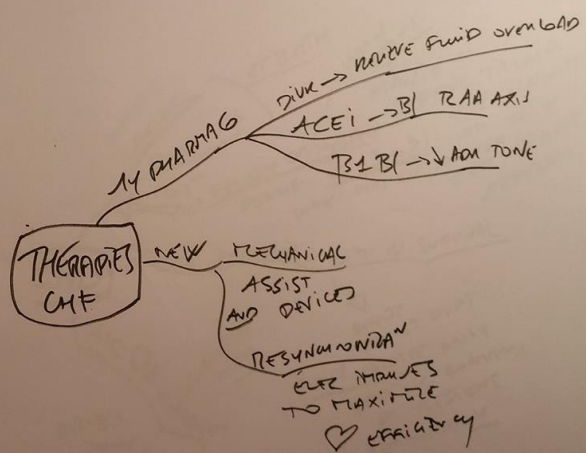
CYANOSIS

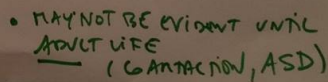
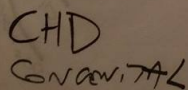
✓ PVP
(CVP) ~ 2-5 mm Hg

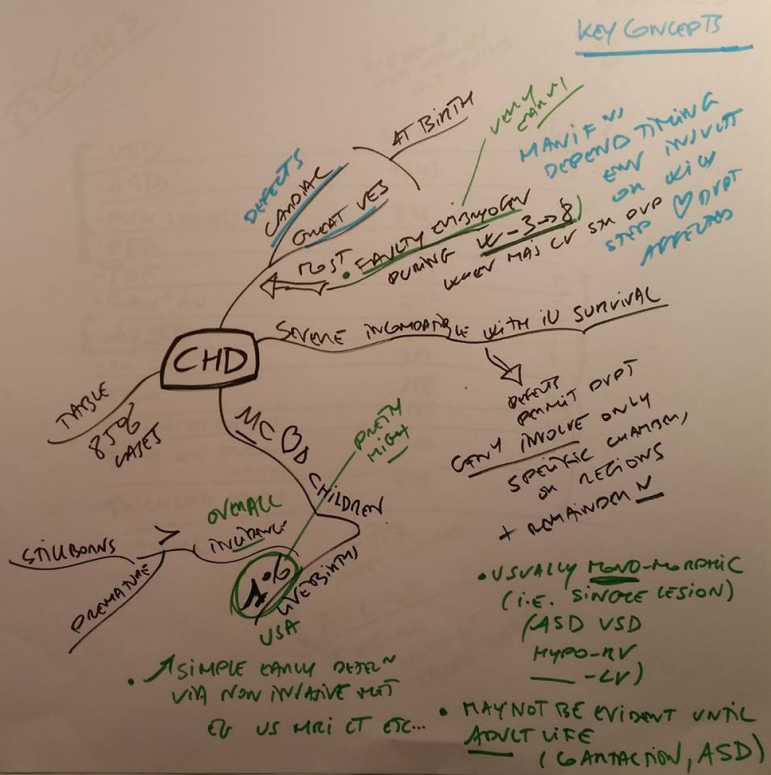
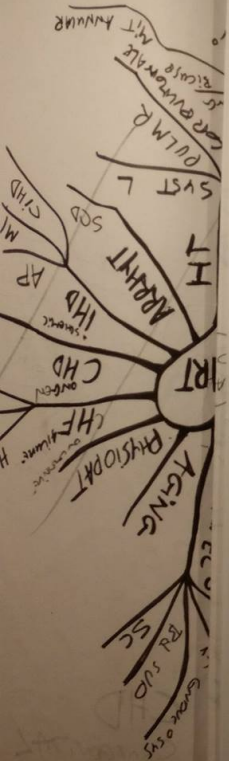
200

14 segments









80% CASES

FREQUENCIES

Incidence
per million
live births

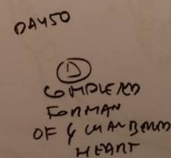
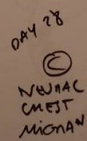
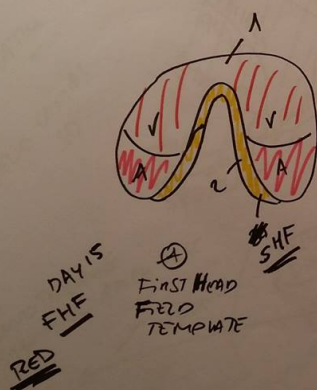
%

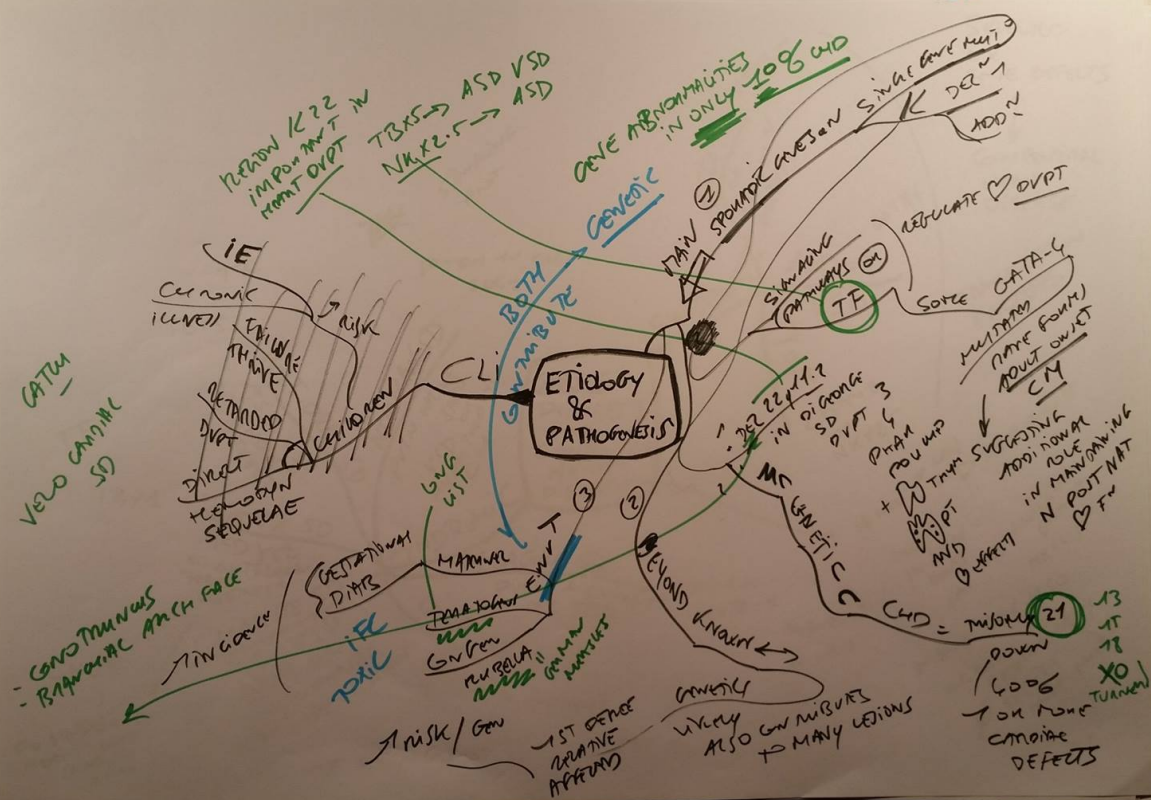
-VSD	4487	42 ✓
-ASD	7043	10 ✓
-PULM STENOSIS	836	8 ✓
-PDA	781	7 ✓
-TOF	577	5 ✓
-Coarctation AO	456	5 ✓
-AVSD	396	4 ✓
-AS sten	388	4 ✓
-TGA	388	4 ✓
-Transposition	736	1 ✓
-TOTAL LUN PULM CONNECTION	120	1 ✓
-TRICUSPID ATRESIA	118	1 ✓
TOTAL	9757	

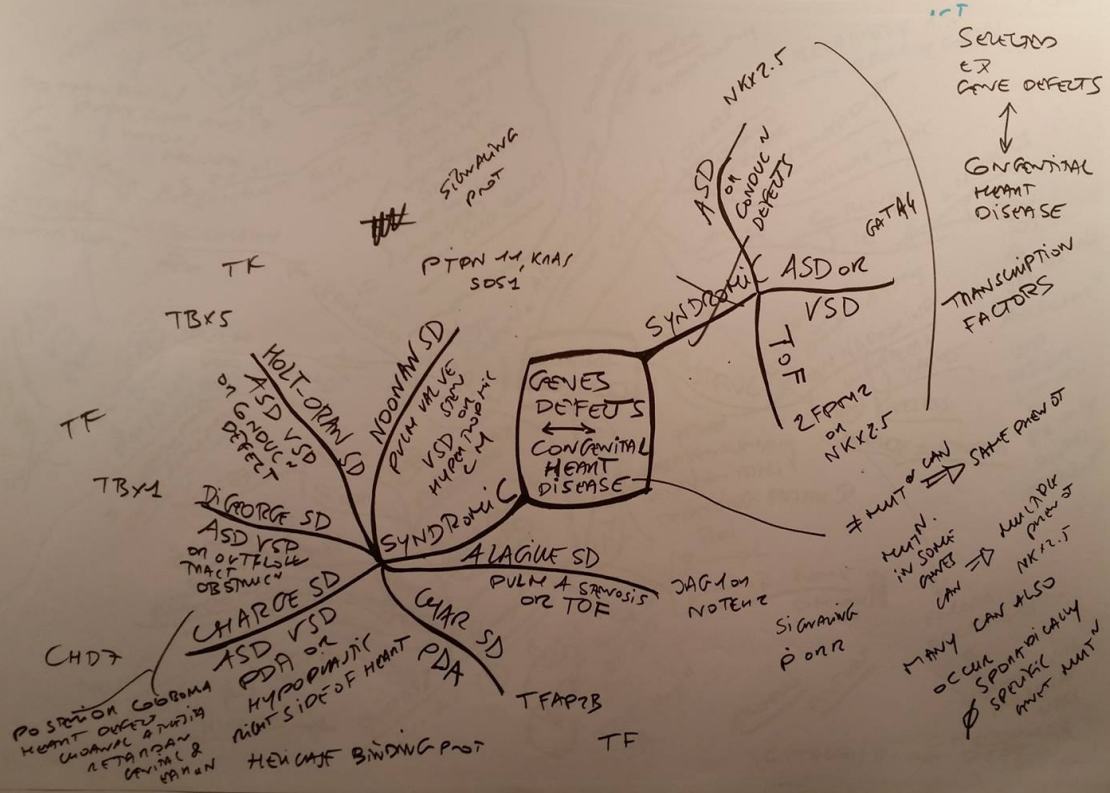
Highway → L → R
 T → R → L
 "BLUE BABIES"

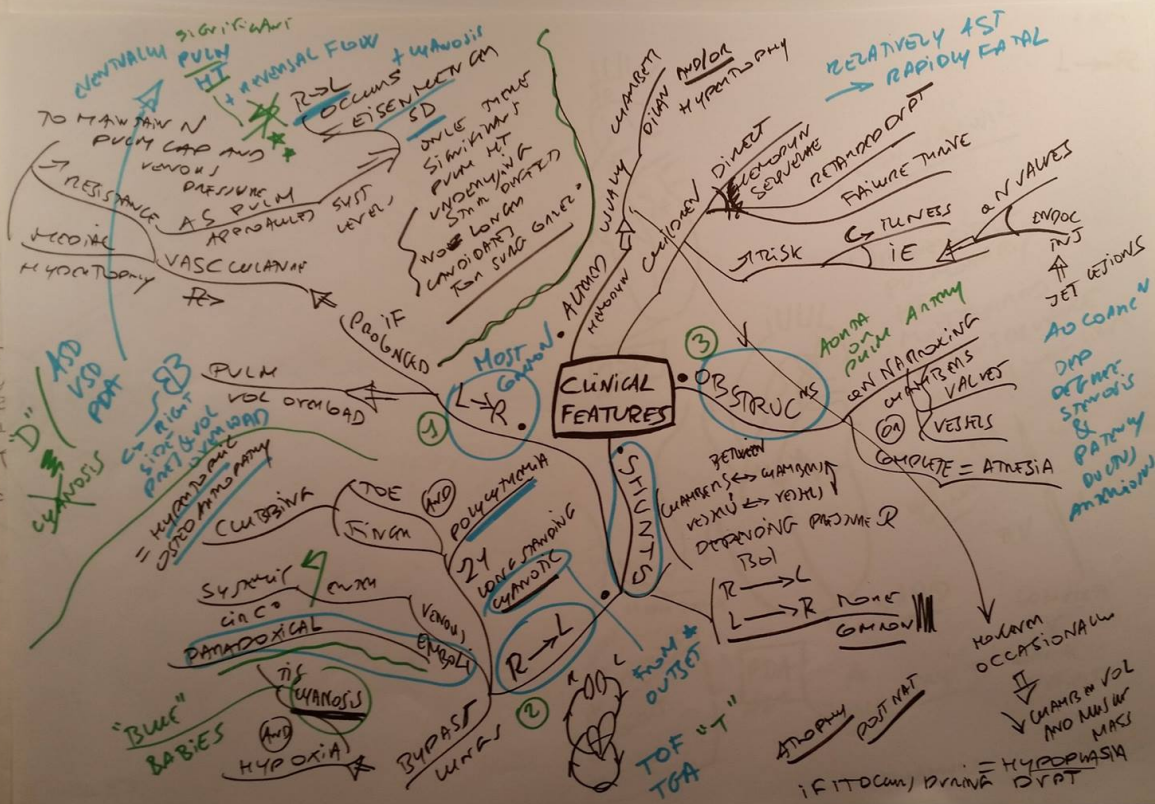
- 1ST HEART FIELD
- 2ND HEART FIELD
- CARDIAC NEURAL CREST

HUMAN
CARDIAC
DYOT
EMPHASIZING
3 MAIN SOURCES
OF CELLS









1 CD

L → R

• NON 4A W OIC

• IRREVERSIBLE PULMONARY HT
MOST FRAGILE
* CONSEQUENCE
(NOW VERY LOW LIKEL)

NA / LA

NOT GROW

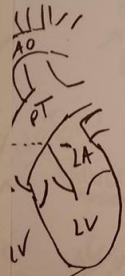
NV / LV

3 ASD

COMPLEX
AV
CANNAL
DELET

PDA
*
"MACHINERY"

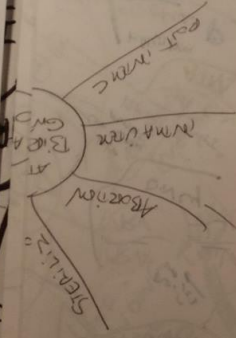
WIDE FUNCTION



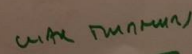
ASD

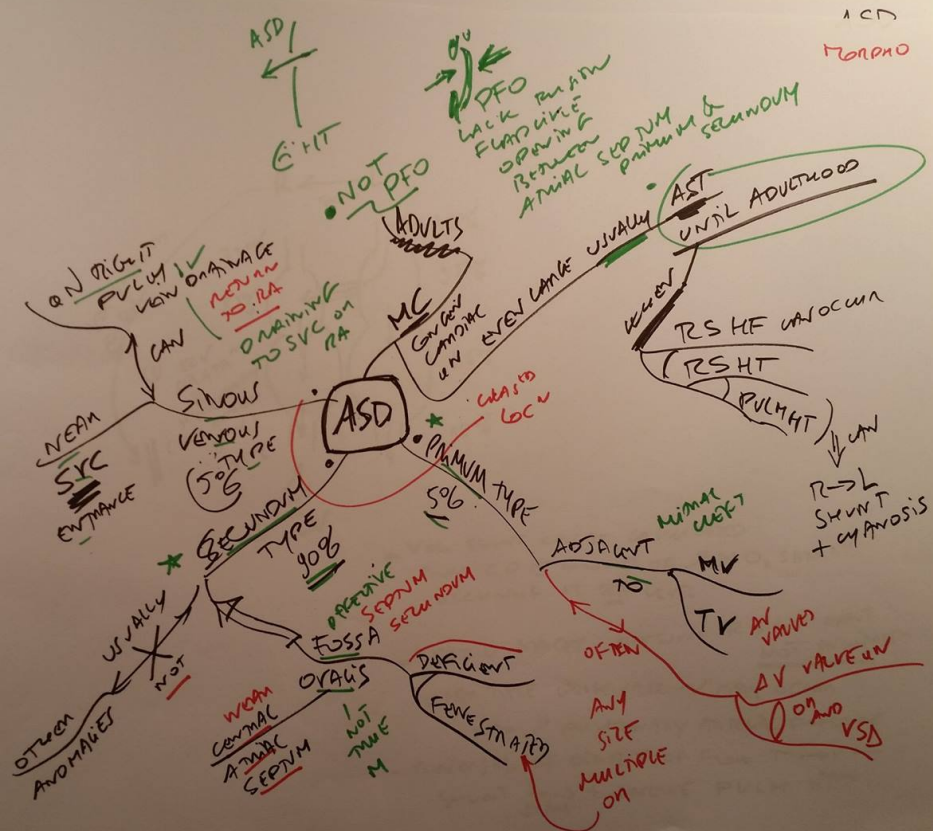
* VOLUME
HYPER TENSION

SHUNT
ARE CRITICAL
IN BOTH
VENTRICLES
PRESENT

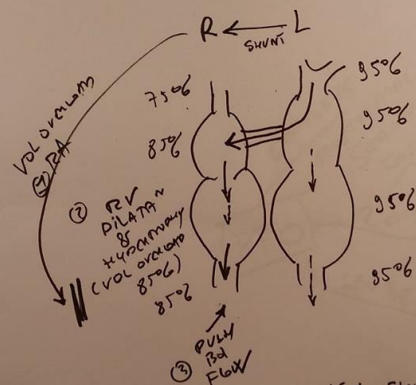


L → R





ASD



- VOL SHUNT CAN BE CALCULATED FROM CO & AMOUNT OF $\uparrow$ IN O_2 SATURATION OCCURRING AT RA LEVEL
- EARLY STAGES PULMONARY RESISTANCE NOT INCREASED WITH THE PULM VASC DILATION OCCUR
 $\Rightarrow \uparrow$ PULMONARY ARTERIAL PRESSURE
- REVERSAL OF DIRECTION OF FLOW THROUGH SHUNT $\rightarrow$ + SEVERE PULM HT JAM

DERIVATIVE
POSTNATAL
SEALING
FOSSIL ORAL FLAP
2006 INDIVIDUAL
PFO
CAN BE CONDUIT FOR PARADOXICAL
EMBOLI
IN SETTING
ELEVATED
RIGHT SIDED
PRESSURE

VSD

MORPHO

- MOST SINGLE
- THOPEIN MAY BE MULTIPLE

likely

within musc septum

= INFUNDIBULAR
IF MUSC BELOW PULM VALV

WHEN HAS
OTY 9006

TRANSDUCER
SEPTUM
NORM AORTIC
VALVE

LOCAN

VSD

ONLY
BUT 3006
20-
ISOLATED

FREQ
OPEN
OR 1/2

TOF

MC OVERALL
DEPENDENT SIZE

CLi

SURG

BEFORE R OVERLOAD
AND PULM HT DVP

EXT. ISIGUM

PULM
HT

LARGE
OWET

CLASSIFICATION

EVOLVING OUT

LATE HYPOXIS

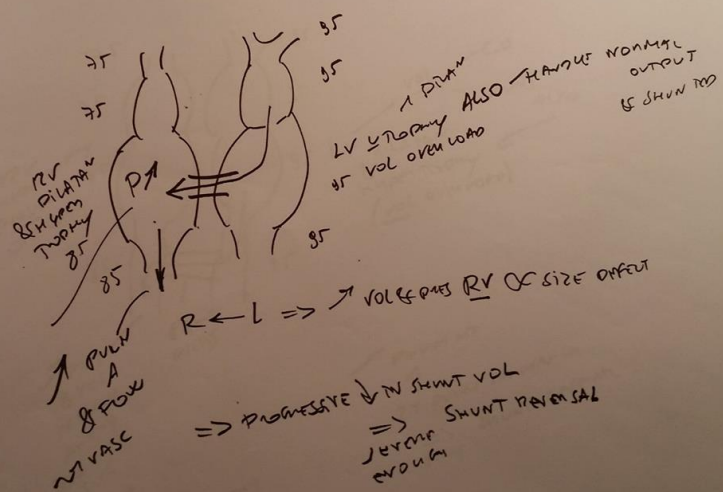
SPONTANEOUS

CLOSURE

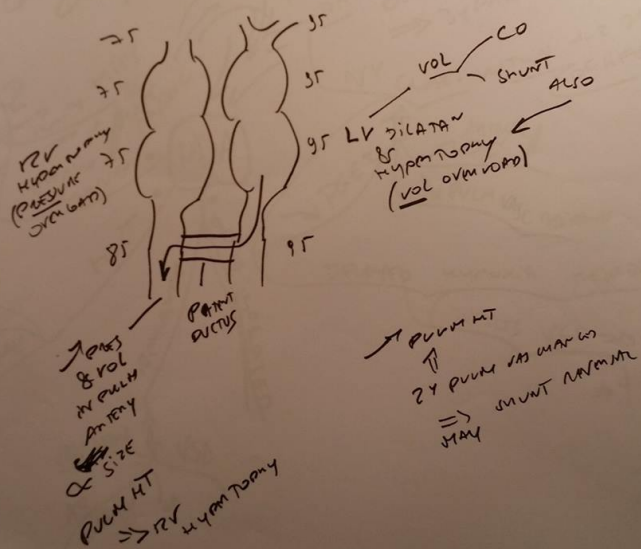
5006 MORE

< 0.5cm Ø

OPEN
SMALL
ONE



PDA



PGE1 TGA TA THORV
why TGA TA THORV
more
A OPEN
like
ipmuc

L-R
pulmonary RSC
AS PULM HT
APPROXIMATELY 90%

overlying
VOL
PNS

RSIDS
LAW
F LANGE

HANSH
CONTINUOUS
MACHINERY USE
MUNITION
ZONES
AST
RUT
INITIALLY

UNLESS OTHER
CONCURRENT
CHDS
ADVOCARD
SURG
MAY
BE
SAVING

ONCE PULM
BROWDER
SIST
PULM
LAMB WIT
ADAM
VANE
ANIM

PDA
90%
ISOLATED

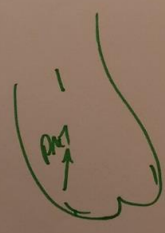
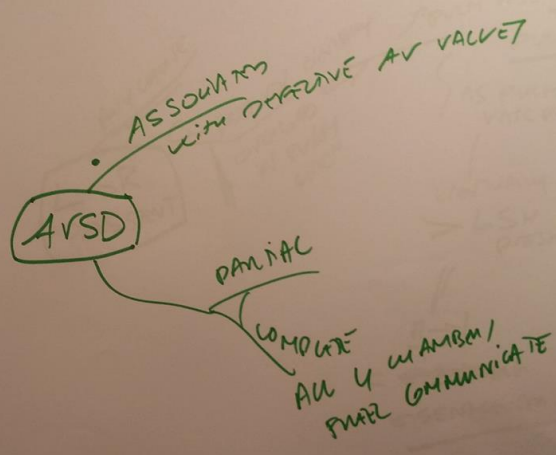
INUSSTEN
NO
CONTRACT
or
VSD

LEFT SUBCLAVIAN ARTERY
DA AORTA → PA AN
DURING FETAL DVT
⇒ BYPASSING LUNGS
NY CLOSES WITHIN 1-2 DAYS
SE LIFE

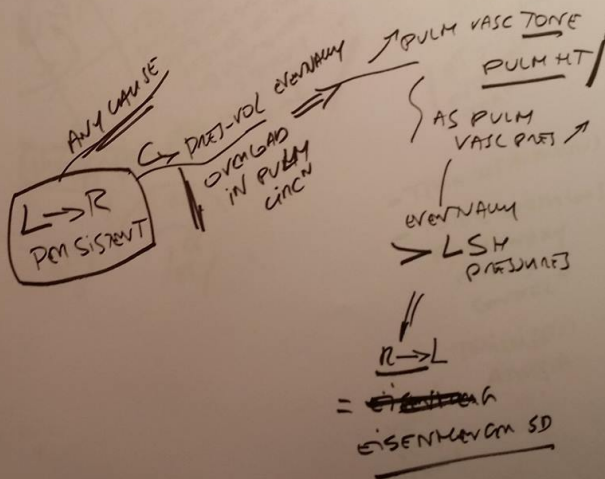
PGE2
ANIMAL
O2
PULM VASC RESISTANCE
DELAYED HYPOXIA RESP DISTURB

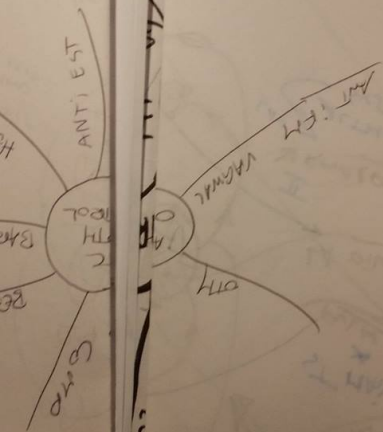
DEFECTS VSD ⇒ PULM VASC DISTURB

R→L



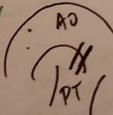
R → L
FURTHER





VSD
DIRECTION
DEPENDS ON
DEGREE OF THE
SUBPULMONARY
STENOSIS
WHEN SEVERE
R→L

Big one



R→L
CYANOTIC

UNO₂EN
Bd
B4 P4 in 6 weeks

PT =
PULMONARY
TRUNK

- Transcatheter Aortic
- Total anomalous pulmonary venous connection
- Transcatheter Aortic

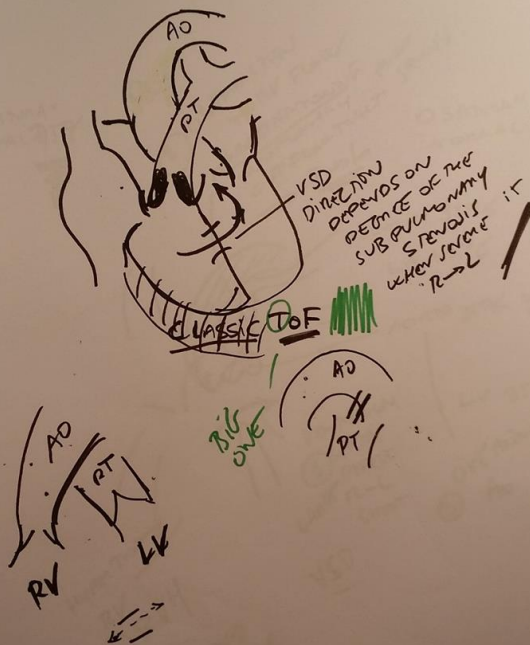
Very
very
serious

φ VSD

PURE TRANSPOSITION

GA

R→L



+ VSD ϕ VSD

COMPLETE TRANSPOSITION

TGA

R to L
CYANOTIC

UNO₂ed
Bd
B4 20/10/16
unlabeled

PT = PULMONARY TRUNK

- TRANS CATHETERS
- TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION
- TRICUSPID ATRESIA

Very very serious

下

ROVT

2. CONSTRUCTION FOR V. FLOW NUM. SIMULATIONS
MANIPULATING
INTERMEDIATE
OUT FROM TASK
 $\Rightarrow$ PROB

OSANNAN 950
NONAL

TOF
Post
Common

Sum 9506

LA 8506

CENTRAL

Aorta 2506 CYANOSIS

Ventricular
① SUBARACHNOID
SPACE
R-L
SHUNT
LARGE

LV 8506
Ove minning
③ A0

VSD

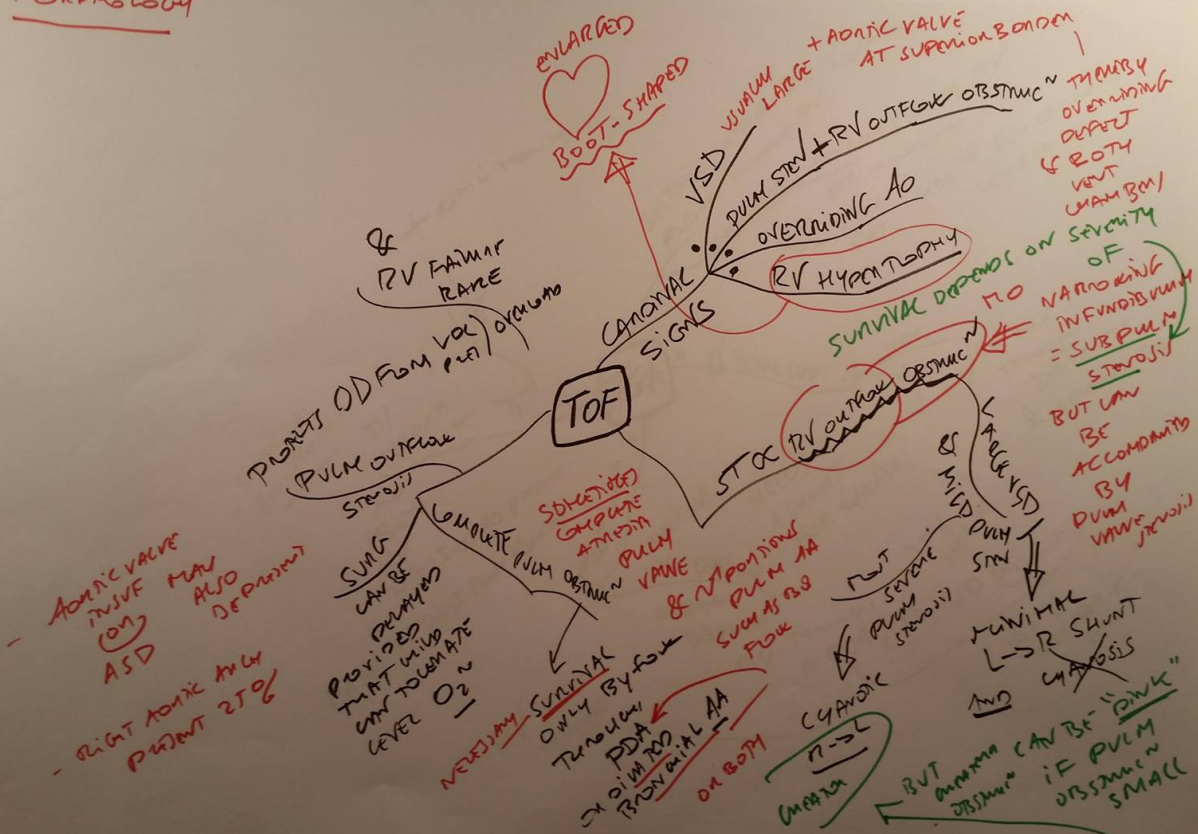
Hyper Thromb

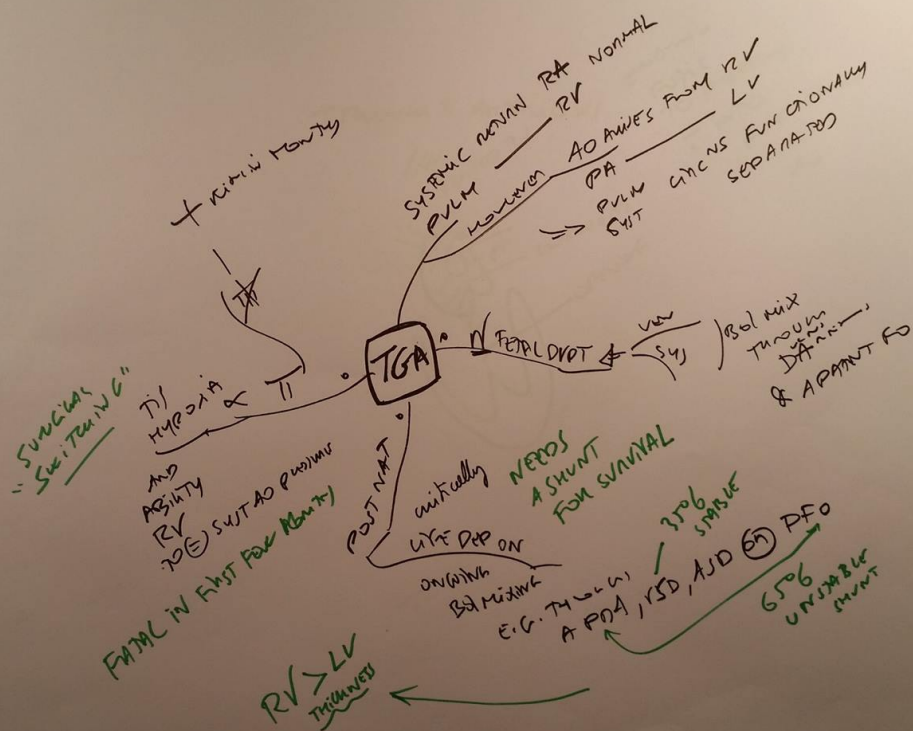
RV
④RVH

running
up stairs

✓ Showed to PUM Anthony

MORPHOLOGY





Transverse Anisotropy

1/4" Fixed Bd



opening

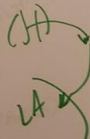
ensure
logic
to properly
into
program
as

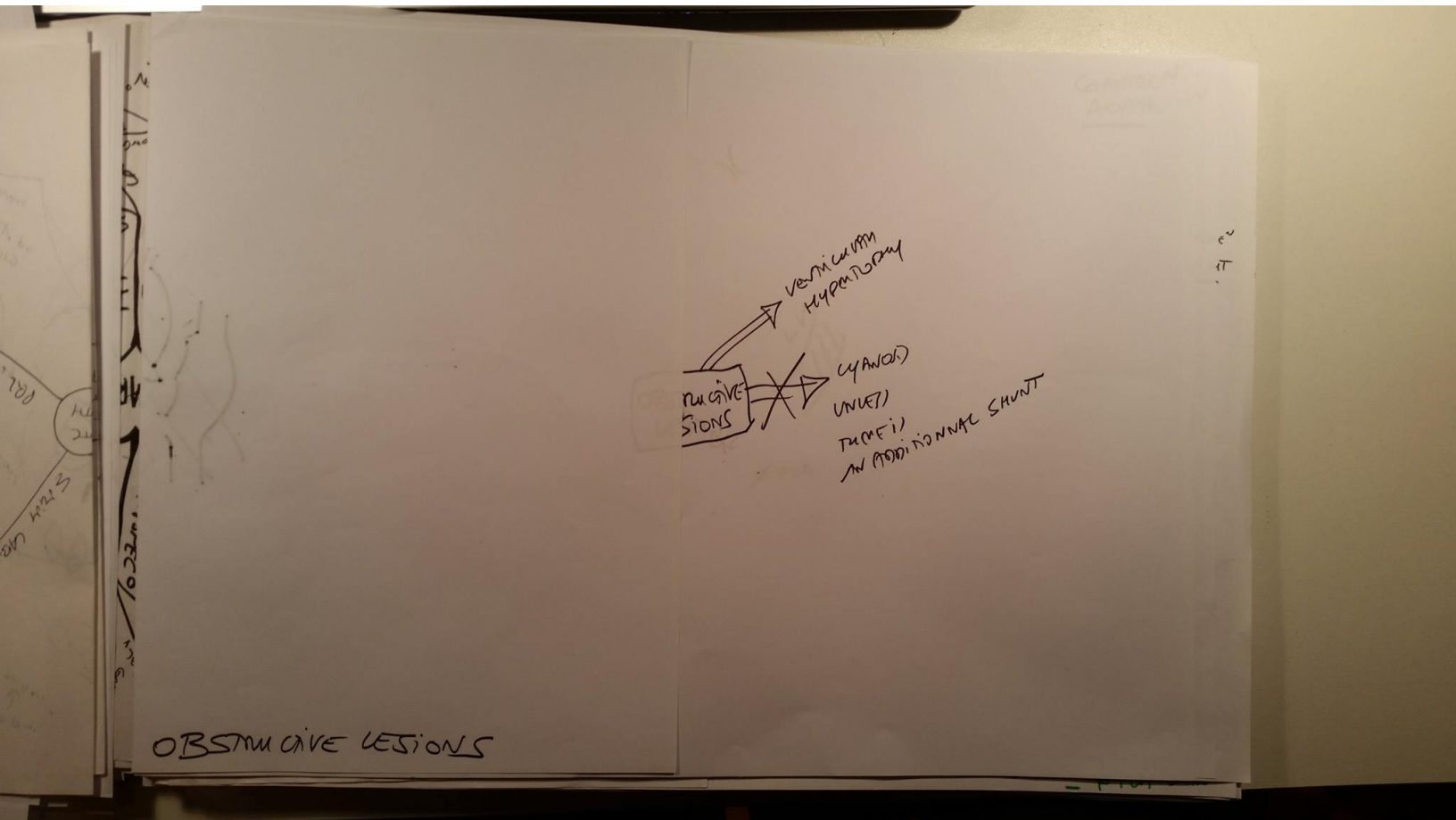
- (Tricuspid atresia)
- Hypoplastic RV
 - Needs a Hunt ASD VSD on PDA
 - High Mortality



^{one normally with even}
TAPVC

- PULM VV ~~GO~~ INTO LA
- BUT INTO L. INNOMINATE ON CONTRARY SIDE
- NEED A PFO or VSD
- HYPOPLASTIC LA





VENTRICULAR
HYPERTROPHY

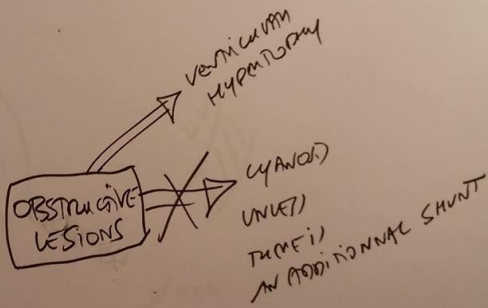
OBSTRUCTIVE
LESIONS

CYANOSIS
(UNMET
THERAPY)

IN ADDITIONAL SHUNT

OBSTRUCTIVE LESIONS

2/11



2
11

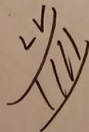
PTCA cath

COARCTAC^N
AORTA



+ PDA

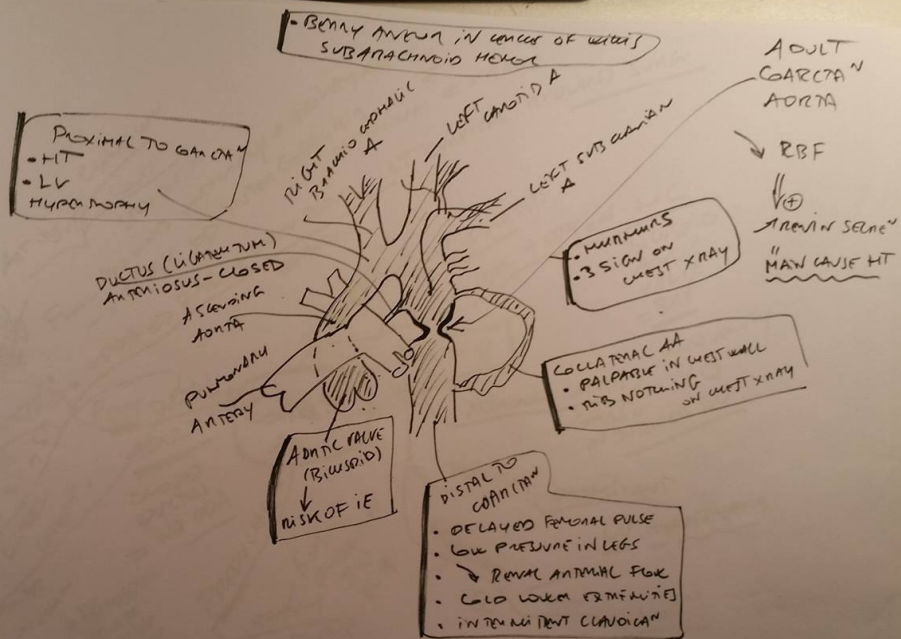
X



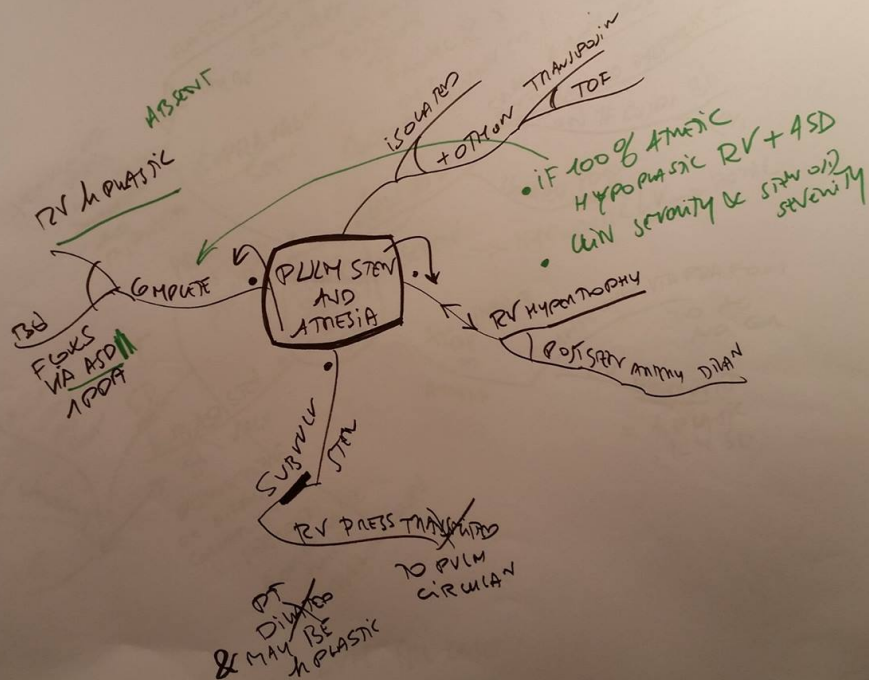
Ø PDA

2 F.

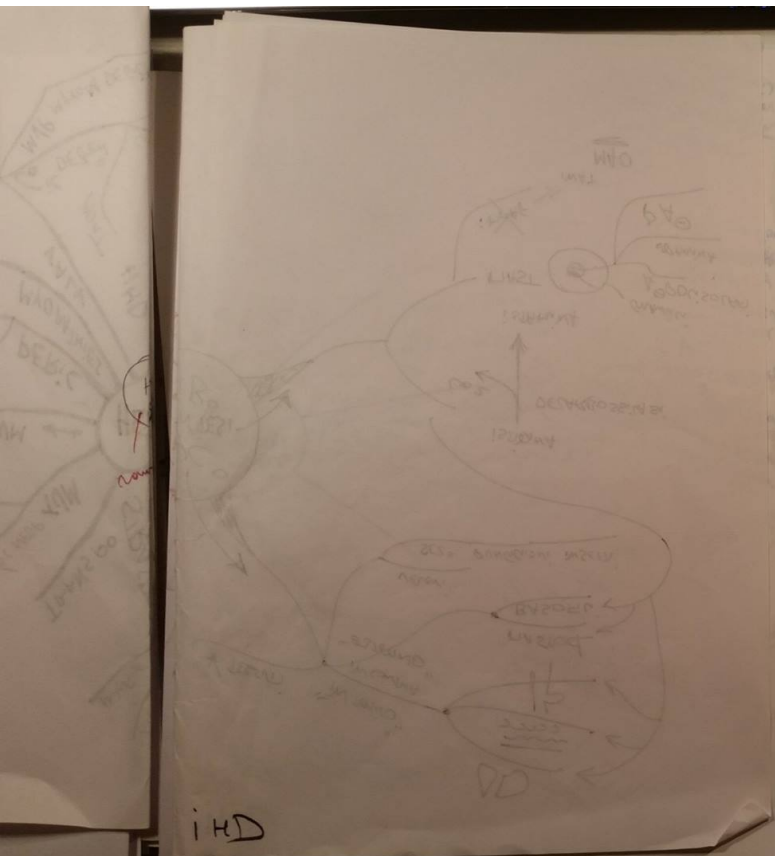
- EMBOL - PTCA CABG -



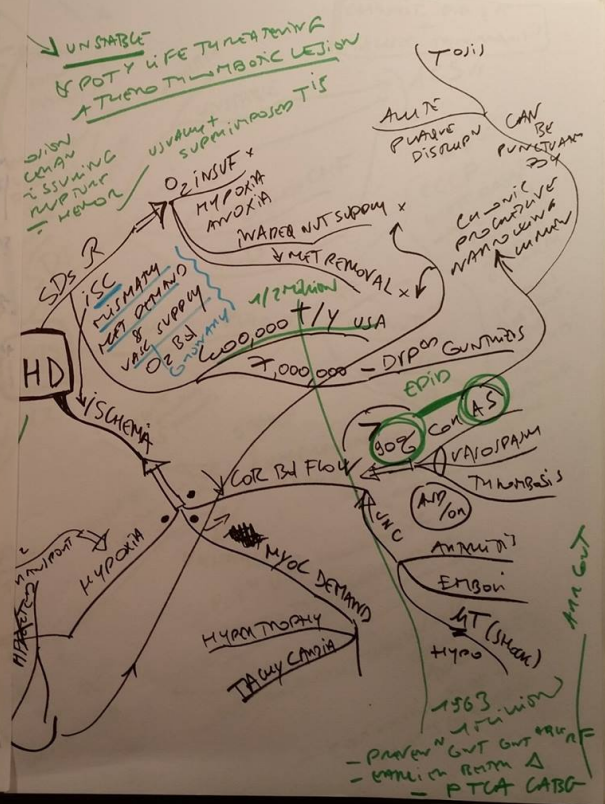
- PTCA CABG -



- example in PTCA CABG!

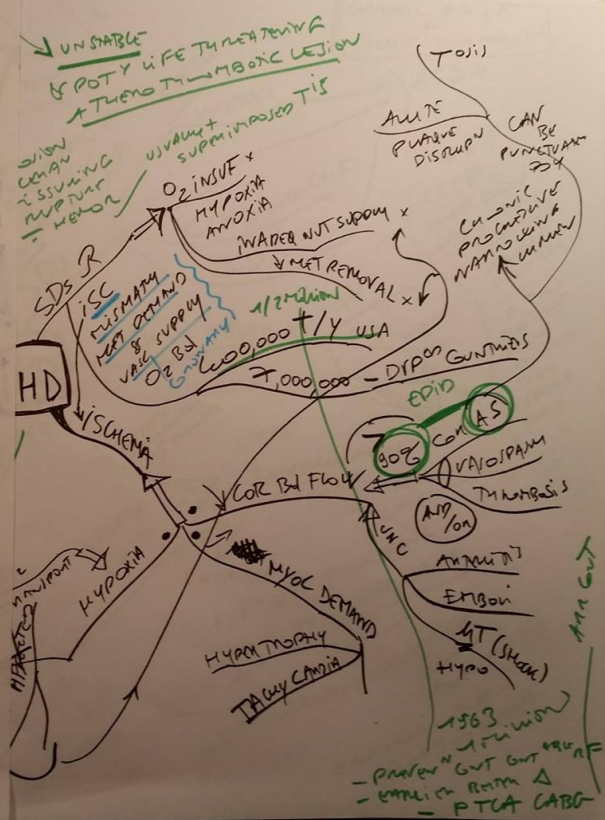


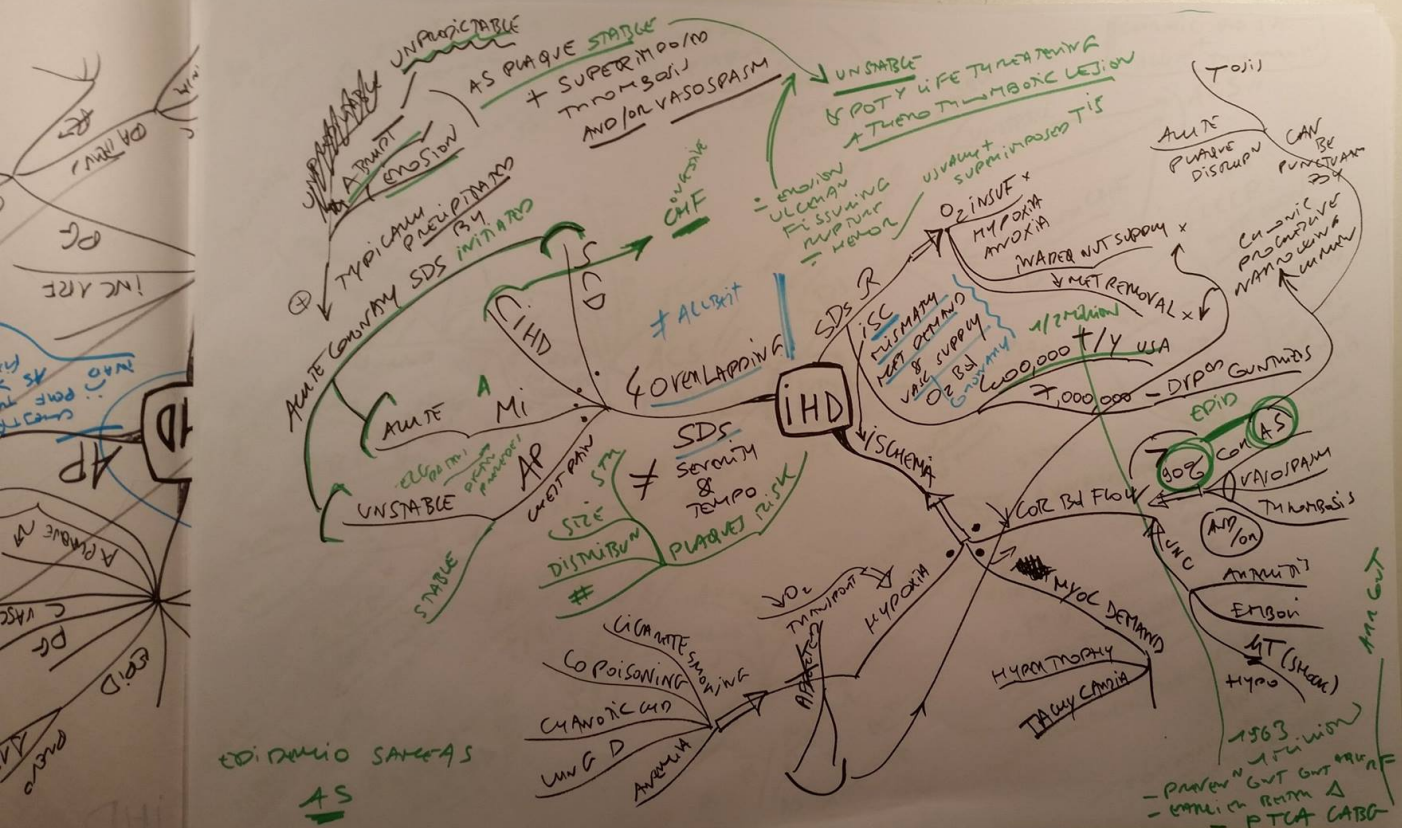
HD

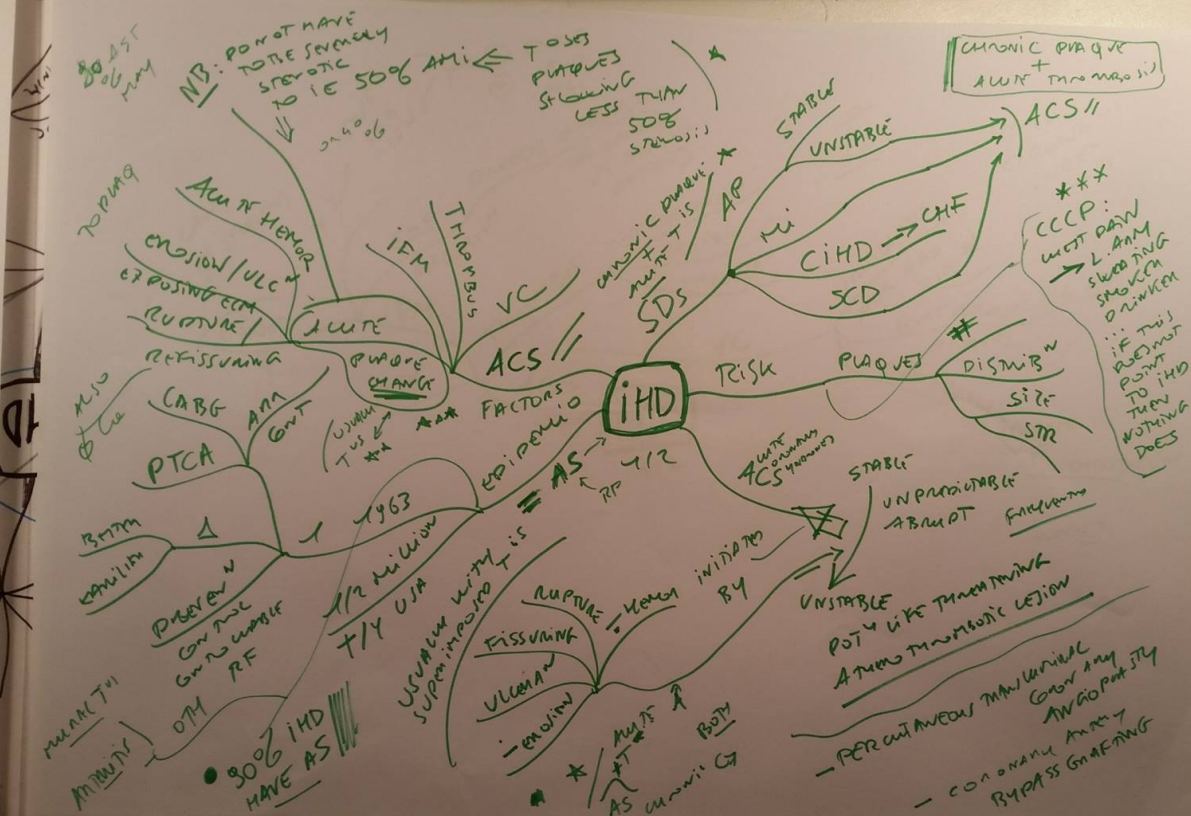


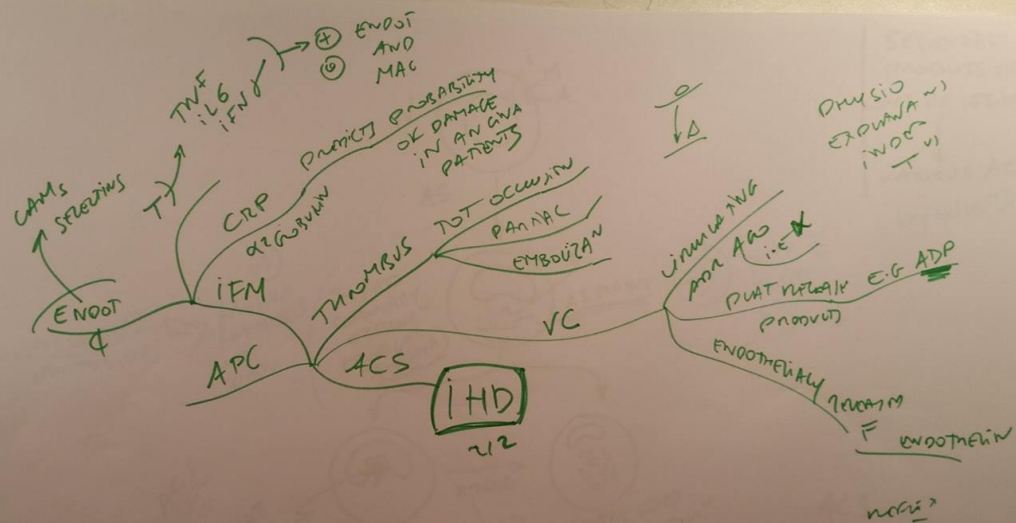


iHD

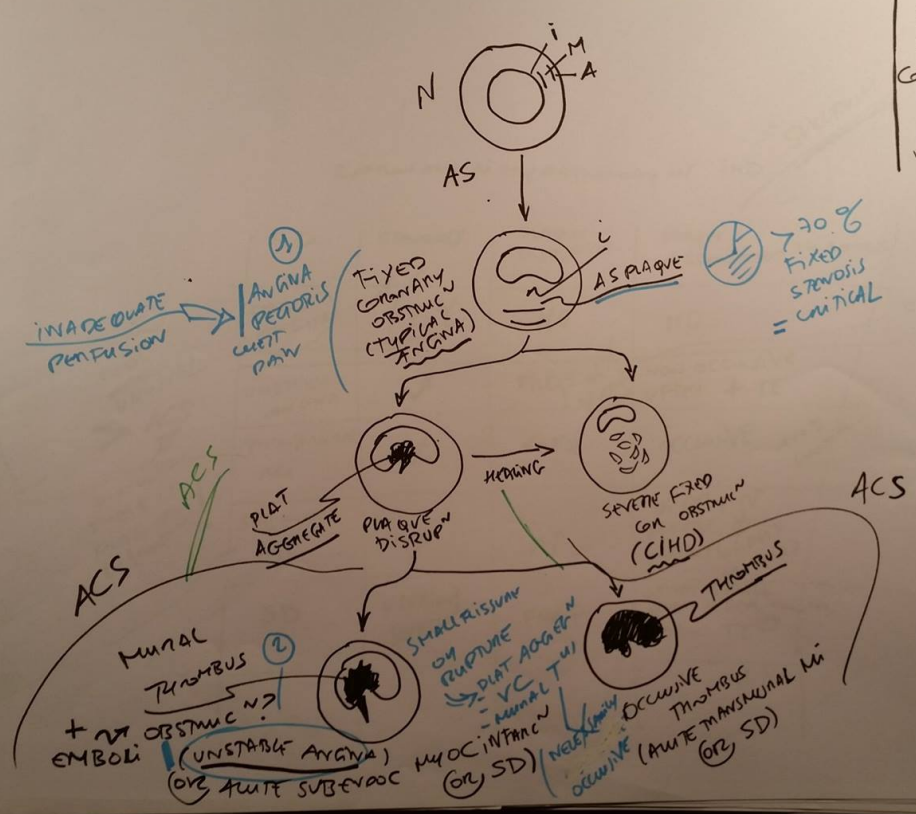








SEQUENTIAL
PROGRESSION
COR AA LESIONS
↓
VARIOUS ACS
KEY WORDS



70%
FIXED
STENOSIS
= CRITICAL

ACS

ACS

CONSIDER ANY ARTERY PATHOLOGY IN IHD

"SPECTRUM"

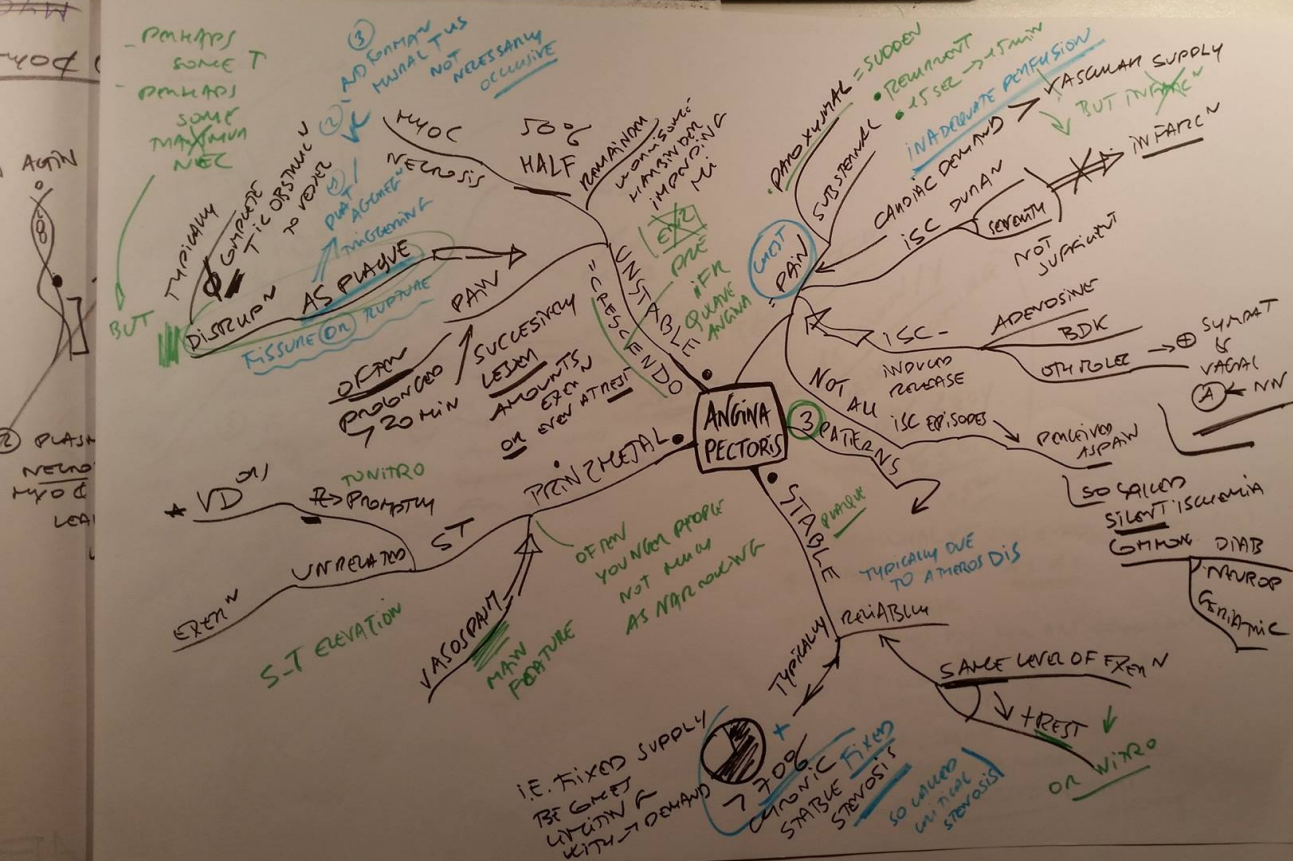
SD	STENOSES	PLAQUE DISRUPTION	PLAQUE + ASSOCIATED THROMBUS
(STABLE ANGINA)	0% 75%	NO	NO
UNSTABLE ANGINA	↗	FREQ ^{in Q wave}	NON OCCLUSIVE OFIM + TE
TRANS-MURAL MI	↗	FREQ	OCCLUSIVE 100% W/IN
SUB-ENDOR MI	↗	↗	WIDELY MAYBE PARIAL / 60-90% ON LVED
SD	USUALLY SEVERE	FREQ	OFIM SMALL PLAT 75-90% THROMBI AND/OR TE

NOT
UNUSUAL

→ ACS

ON WHICH
THERE IS
PLAQUE
DISRUPTION!





MI

4 + ~~4~~ VASE OCCURSION

US 15 Million / Y

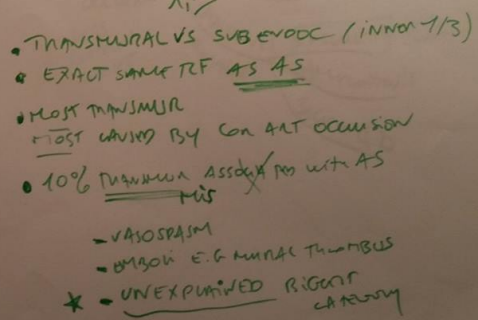
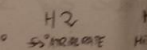
MIK with AGE

10% $\leq$ 40Y

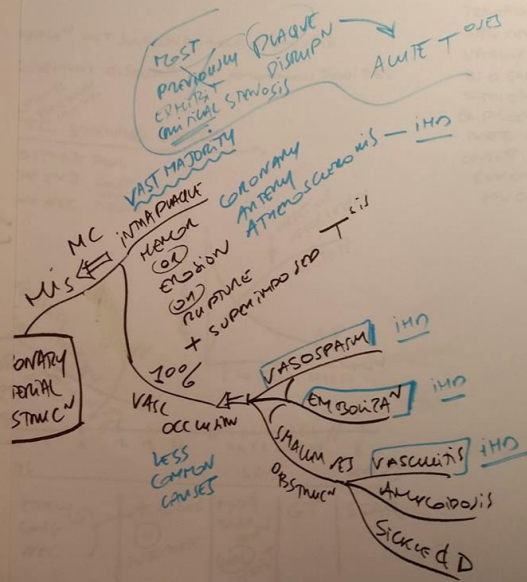
50% $\leq$ 65Y

- TRANSMURAL VS SUBENDOC (inner 1/3)
- EXACT SAME RF AS AS
- MOST TRANSMUR
MOST CAUSED BY CON AAT OCCASION
- 10% TRANSMUR ASSOCIATED WITH AS
MIS
 - VASOSPASM
 - MYOIN E.G. MYOIN THROMBUS
- * - UNEXPLAINED RIGIDITY
CHANGING

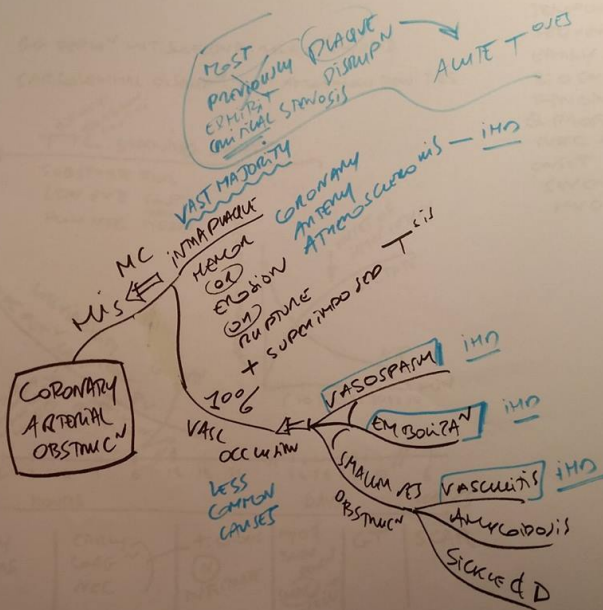
Mi^o



PATHOGENESIS

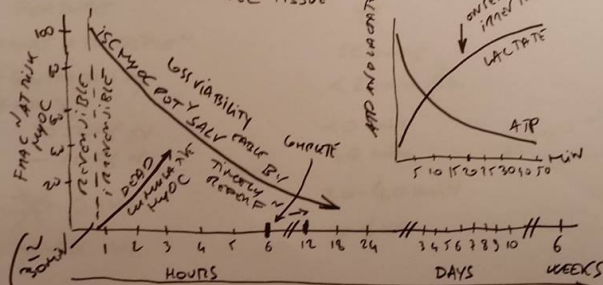


HARDEN
STABLE
ESCEVDO
PUNZIGHE



REV GG DEPLEN. BUT SWELLING. NECA Xⁿ MYOFIB
 EM < IIR SARCOLEMMA DISAPⁿ BUT ANATOMICAL IDENTITIES

HC ^{ROUNDED ENDOPLASMIC RETICULUM}
 TTC STAINING DEFECT
 SUBSTRATE FOR
 LDH ENZ LOST
 FROM NEC TISSUE



TEMPORAL
 SEQUENCE
 EARLY
 BIOCHEM
 FINDINGS
 & PROGRESSION
 NEC AFTER
 ONSET
 SURVIVAL
 MYOCARDIUM

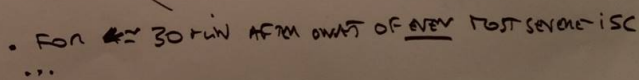
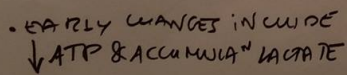
LM	WARY FIBERS	EARLY COLLAPSE NEC	+ STUNG (N) INFILTRATE	MYO CARDIOMYOCYTE INFILTRATE	GT	SCAR
CROSS			PALLOR	SEMIC BORDER + VASCULAR SOFTENING	SOFT + VASCULAR EDGE DEPRESSION	SCAR

MYOCARDIAL RESPONSE

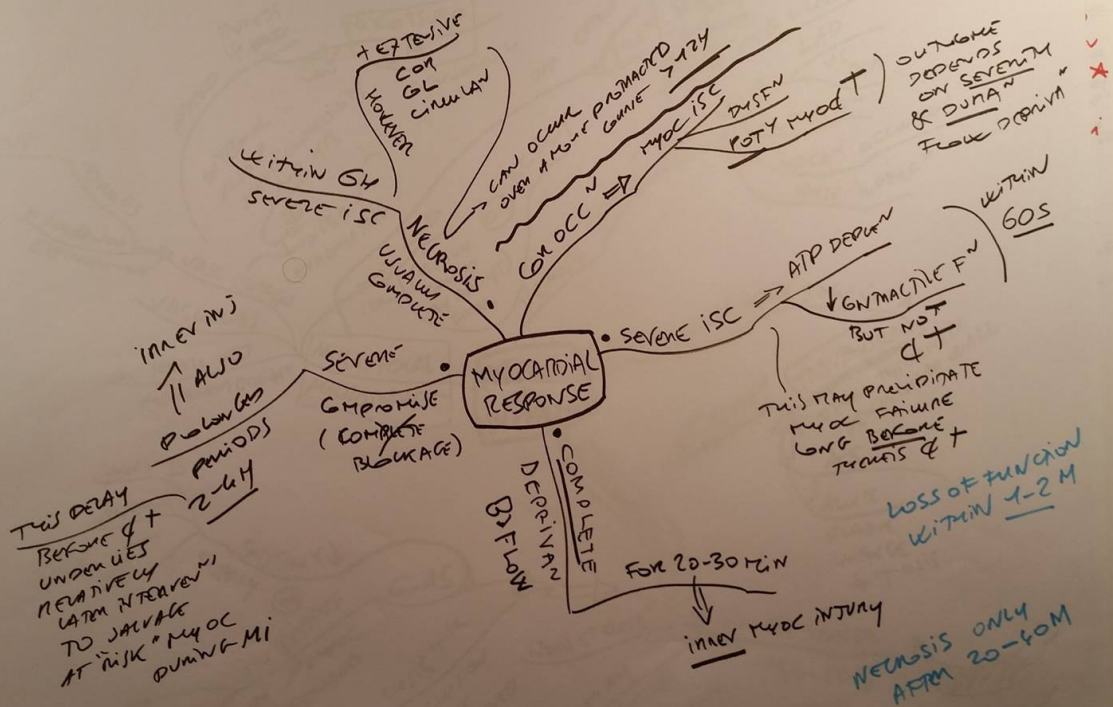
APPROXIMATE τ ONSET KEY EVENTS IN ISC $\heartsuit$ MYOC

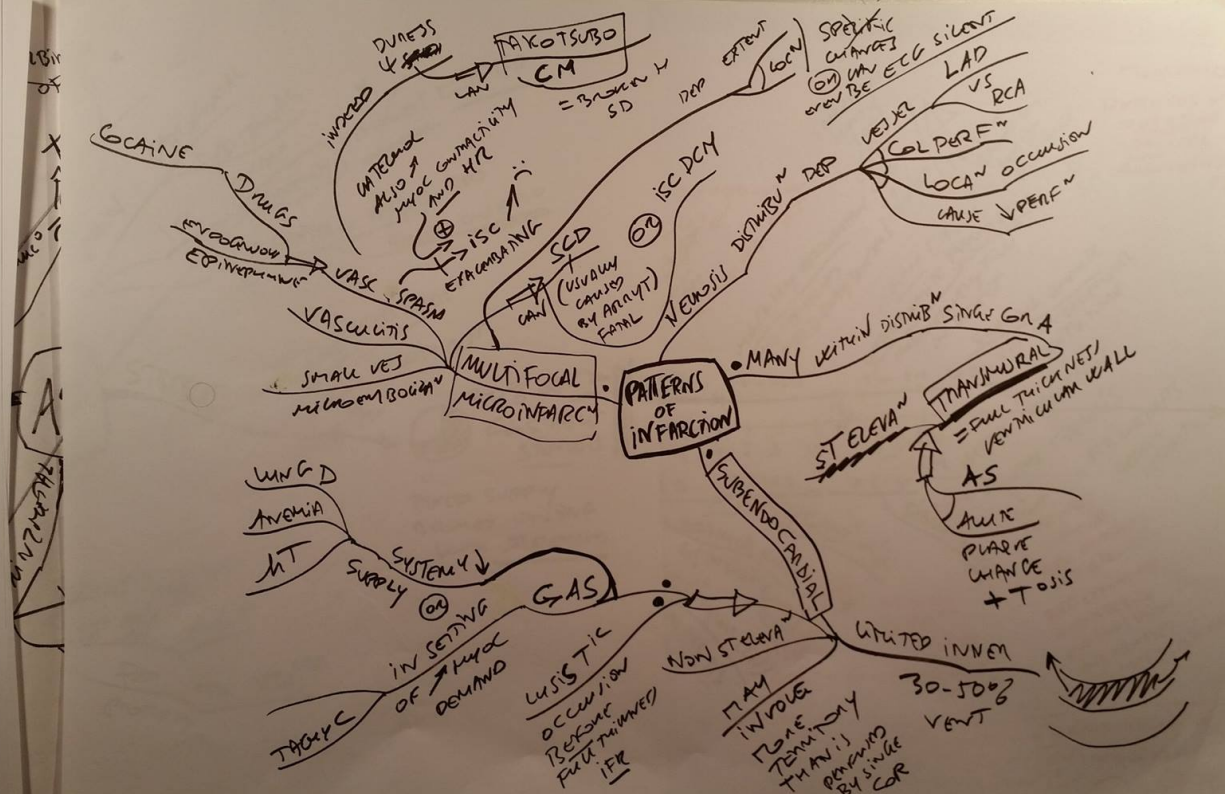
FEATURE	τ
• ONSET ATP DEPLEN	SECONDS
• $\downarrow$ CONTRACTILITY	< 2 min $\star\star$
• ATP $\downarrow$ 70-50% IV — 10% N	10 min 40 min
• $\star$ $\downarrow$ INJ	20-40 min
• $\downarrow$ VASC INJ	> 1 HR

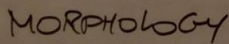
ATP $\approx 0 \Rightarrow \star\star$ INJ

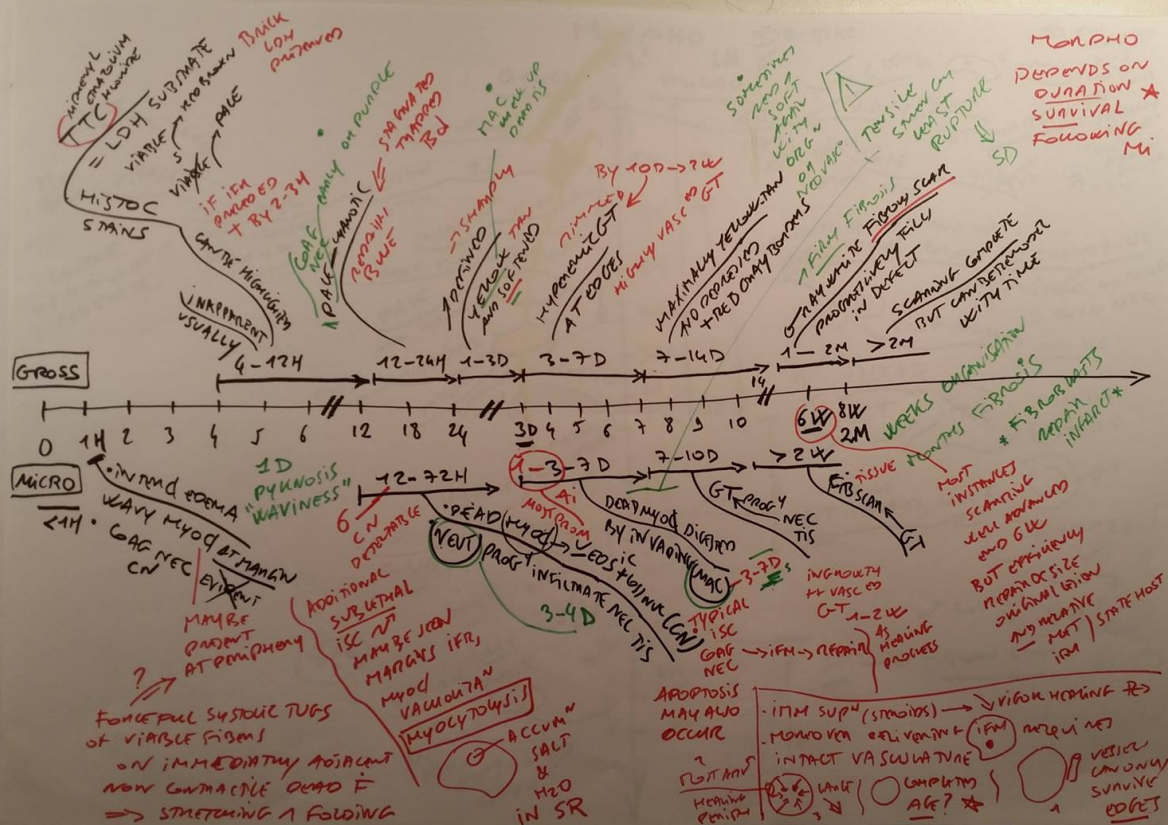


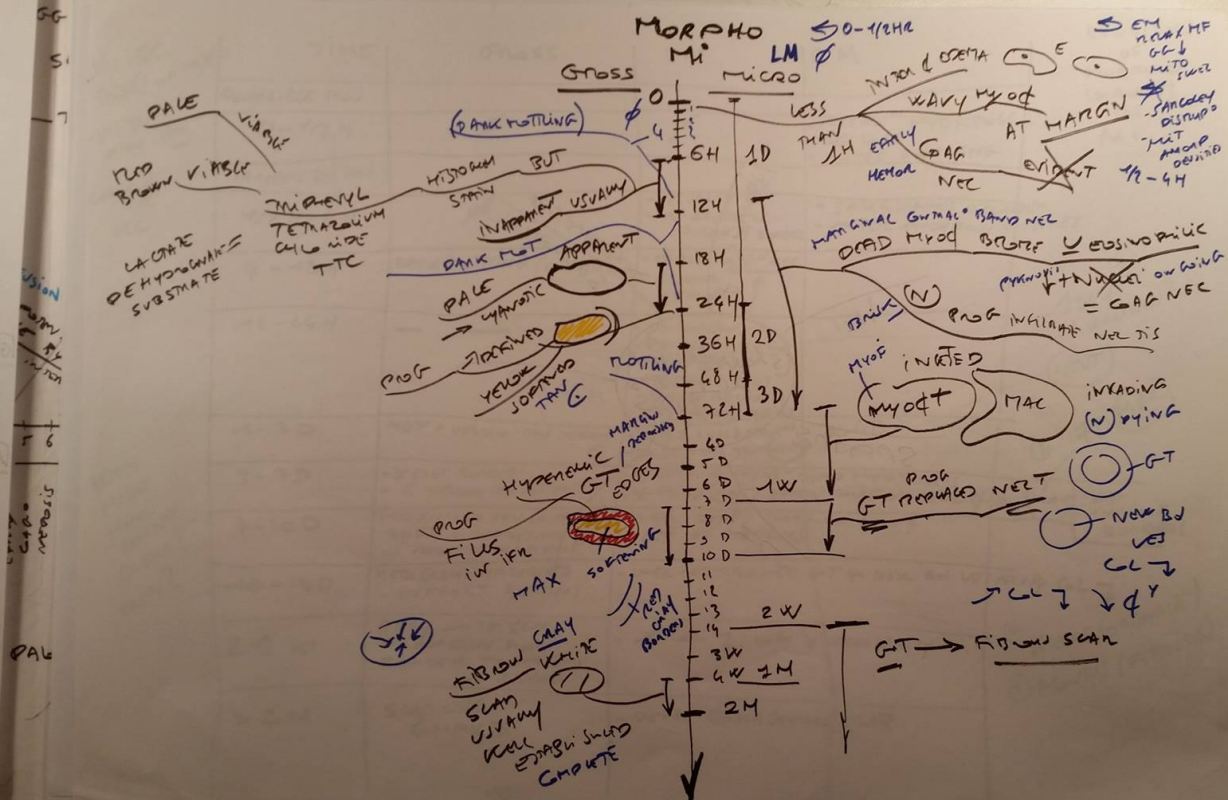
TEMPORAL
SEQUENCE
OF EARLY
BIOCHEM
FINDINGS
↳ PROOFⁿ
OF NECROSIS
APPR ONSET
OF SEVERE
MYOCISC











IN ISC
Gross (on hist)
- visible only in
cannot see
ISC

When
might
myof
appear?
why?

TIME	GROSS	LM	EM
PERMISSIBLE INJ			
0-1/2 H	Ø	Ø	- RELAX MYOFIBERS - GC LOSS - MITOC SWELLING
IRREVERSIBLE INJ			
1/2 H-4 H	Ø	- USUALLY Ø - MYXINOSITY FIBROBLAST BORDER	- SARCOLEMMA DISORDER - MITOC AMORPHOUS DENSITIES
4-12 H	DARK BOTTLING (OCCAS)	- EARLY COAG NEC - EDEMA - HEMOR	
12-24 H	- - -	- ONGOING - PYKNOTIC NUC - MYOFIBERS - MARGINAL COAG - BAND NECROSIS - EARLY (MYO) INFILT	
1-3 D	MOT + YELLOW-TAN INFILTR	- CN + INFL & STIMULI - BASIC INFILTR (INFILTR)	
3-7 D	- YENIC BORDER - C YELLOW TAN SOFTENING	- BEGINNING DEGENERATION OF MYOFIBERS - DYING (NEUT) EARLY PHASE OF DEAD Q	
7-10 D	MAXIMALLY YELLOW TAN & SOFT + DEGENERATION AND TAN MARGINS	- WELL DEVELOPED PHASE OF DEAD Q - GT AT MARGINS	
10-14 D	RED GRAY DEGENERATION INFILTR BORDER	- WELL ESTABLISHED GT + NEW RD VESSEL & GL	
2-8 W	GRAY WHITE SCAR, POSSITIVE FROM BORDER TOWARD CORN OF INFILTR	- COL 7, WITH V. REGULARITY	
> 2 M	SCARRING COMPLETE	- DENSE GLUCANOUS SCAR	

EVOLUTION
OF
MORPHO
CHANGES
IN
MI

1 DAY
"WATERY"

(NEUT)

3-4 D (2)

7 D (MAC)

RY MAC AT INFILTR BORDERS

Weeks (on granular)
Months (FIBROSIS)

② MIOC
USUALLY
SOMMAMAT
MEMOR

ISC VAS INJURY
APPEF
NORMAL
BAND NEC
AND TRIMIC
CONTRACT

Ca²⁺
OVERLOAD

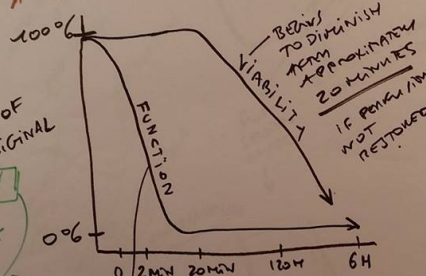
REPERFUSION

- THROMBOLYSIS
- PTCA
- CABG

CANNOT RESTORE
NECROSIS ON DEAD F
ONLY SY INJURED
ONES,
AND PREVENT
FURTHER
NECROSIS

-FR
-IL

PERMANENT OCCUSION



THEN MANY
VASC IN AFFECTED
REGION
SUFFER
DEATH

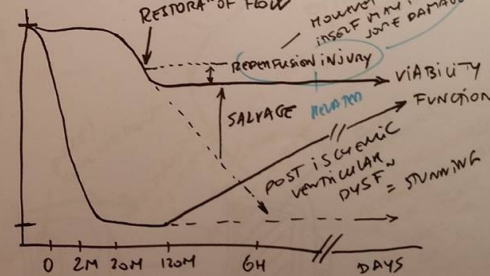
oxygenation
OXYGEN
DEPRIVATION
IF FLOW
PASSES
TOO
SLOWLY

DEAD
FUNCTION

LOOED
POORLY
CONTRACTILE
(STUNNED)
1 → 2D
MAY
RECOVER
VIABILITY

REFLOW
TO ①
PREVIOUSLY
INJURED
& THROMBOSIS
SALVAGE
BETTER
AT RISK
POTENTIAL
INDUCES

TEMPORARY OCC + REPERFUSION



SOME NEC
IS PREVENTED
MIOC SALVAGED
AT LEAST
SOME FN
CAN RETURN

③ CAN
1 REC
AND PREVENTION -
INDUCED
MYOCARDIAL
+ LATE OCCUSION

EFFECTS OF
REPERFUSION
ON

MIOC
VIABILITY & FN

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

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REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

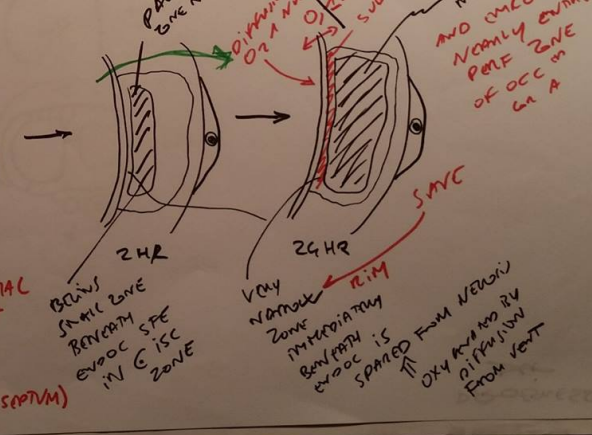
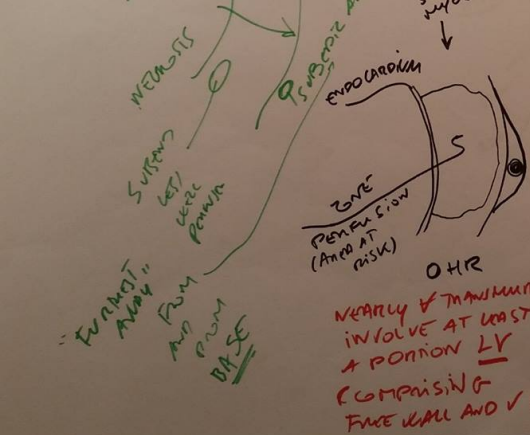
REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY

REPERFUSION INJURY



MORPHOLOGY

PROGNE ~
MYOC NEC
AFTER
COR A OCCLN

BASE
APEX
WALL THINNING
ENDOCARDIUM
PERICARDIUM
NECROSIS
SUPPORTS LEFT VENTRICLE PUMP
"TUNNEL" AWAY FROM AND FROM BASE

NEARLY 1/3 TRANSVERSAL INVOLVE AT LEAST 1 PORTION LV (COMPRISING FREE WALL AND V SEPTUM)

24 HR
BRAIN SMALL ZONE BENEATH EVIDENT IN EISC ZONE

VERY NARROW ZONE IMMEDIATELY BENEATH IS SPARED FROM NERVOUS OXY AND NO RV DIFFUSION FROM VENT

CONTRACTED INFARCT INVOLVING ENTIRE AREA AT RISK

OR A NIT FROM VENT IN HEAVY SUBENDOCARDIAL

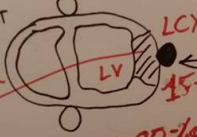
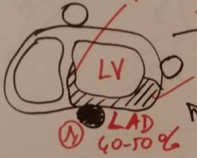
ZONE NECROSIS AND COMPREHENSIVE PERI ZONE OF OCC IN CA A

SMVC

MORPHOLOGY

OTHER LOCATIONS
SOMETIMES
LEFT MAIN COR ARTERY
- 24 DIAG BRANCHES
LEFT & DEFC
ON
MARG BR
L UNC

TYPICAL RIGHT DOMINANT HEART TRANSMURAL INFARCTS



PERM OCC
PERMANENT OCCLUSION
LAT WALL
EXCEPT AT THE APEX
15-30% RIGHT COR OBSTRUCTION
PERM OCC (OR ITS POSITIONING BRANCH)
EXTENT FROM TI FREE WALL OF SEPTAL POSITION
LL INTO ADJACENT RV WALL
ISOLATED IFR RV UNUSUAL 1-3% AS IFR AMIA

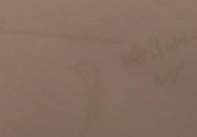
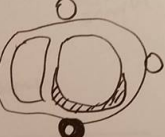
PERM OCC
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15-30% RIGHT COR OBSTRUCTION
PERM OCC (OR ITS POSITIONING BRANCH)
EXTENT FROM TI FREE WALL OF SEPTAL POSITION
LL INTO ADJACENT RV WALL
ISOLATED IFR RV UNUSUAL 1-3% AS IFR AMIA

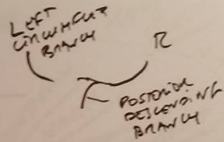
NON-TRANSMURAL INFARCTS



DISTRIBUTION
MYOCISC NEC
CORRELATES
W/ TI
(LOCAL & NATURAL)
PERFUSION
TRANSIENT / PARTIAL OCC
OBSTN
REGIONAL SUBENDOC INFARCT

GLOBAL
HYPO
CIRCUMFERENTIAL
SUBENDOC INFARCT

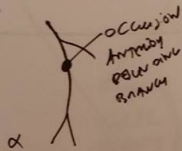
SHALL
INTIMATURAL
VESSEL
OCCLUSIONS
MICROINFARCTS



Bd
SUPPLY
Mi
Dep on Ant &
Col

Position

50%



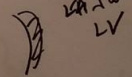
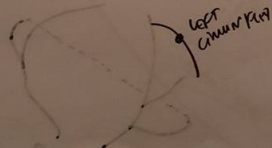
α wall
LV
+ on ϕ SEPTUM

30%



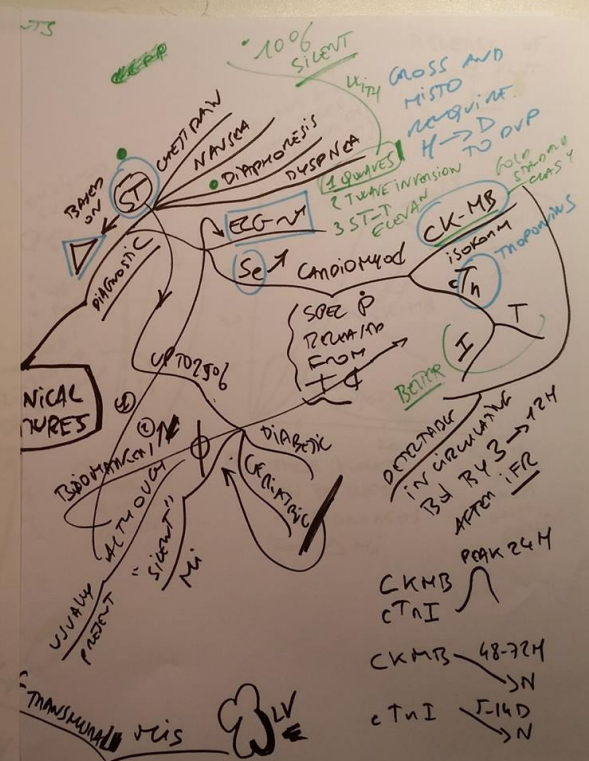
π wall
LV
+ on ϕ SEPTUM

20%

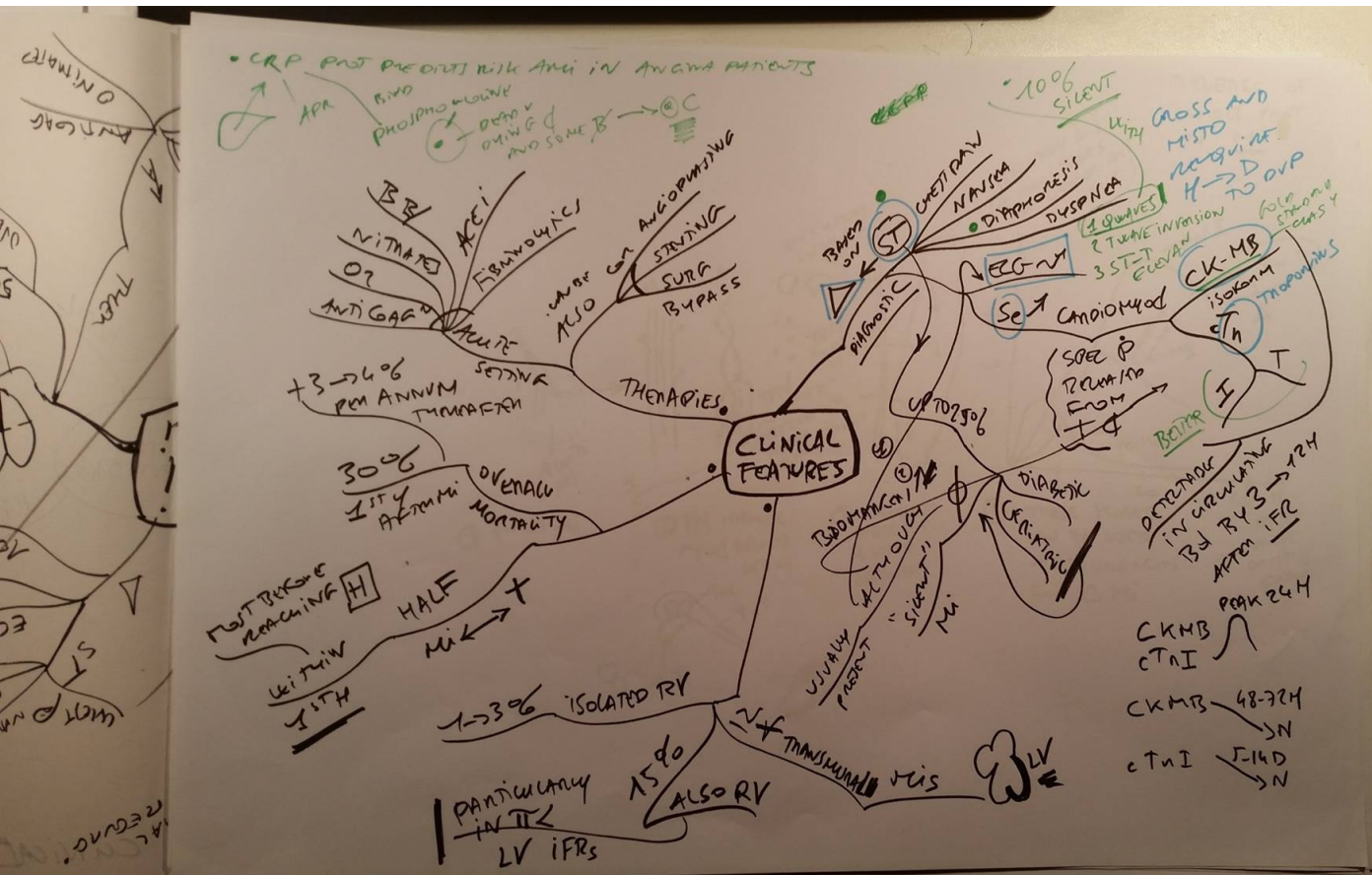


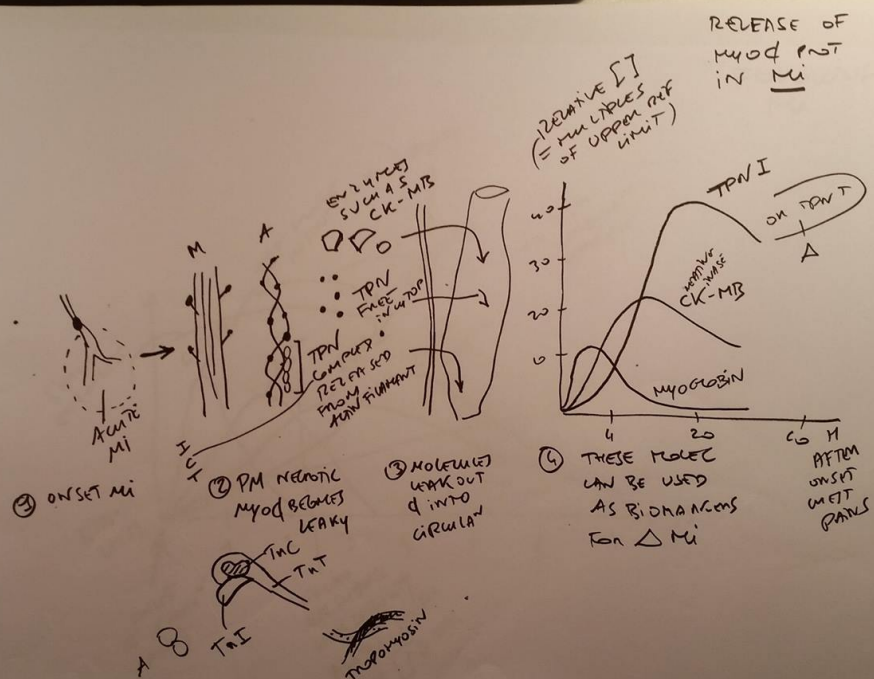
L wall
LV

NAIL
 HISSING PENDAGES
 HAIR
 H. HISI

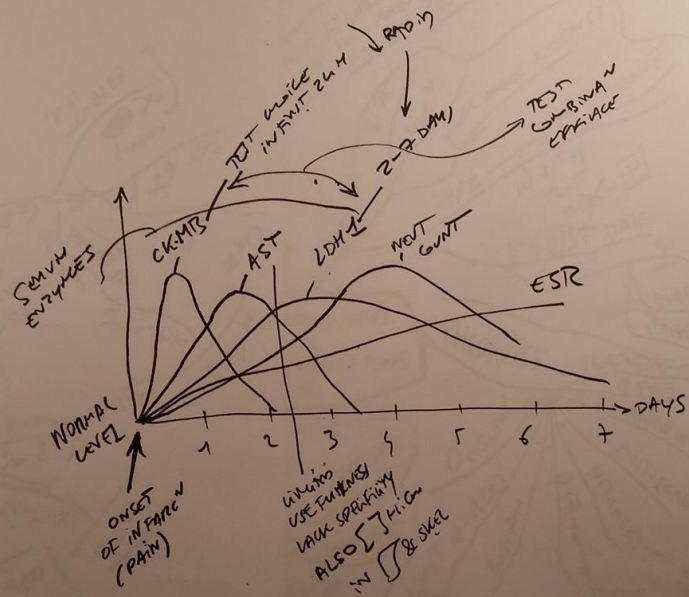


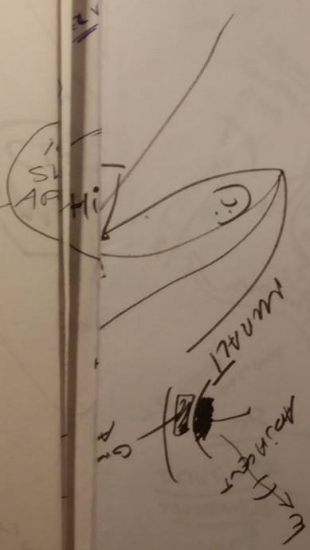
CLINICAL FEATURES



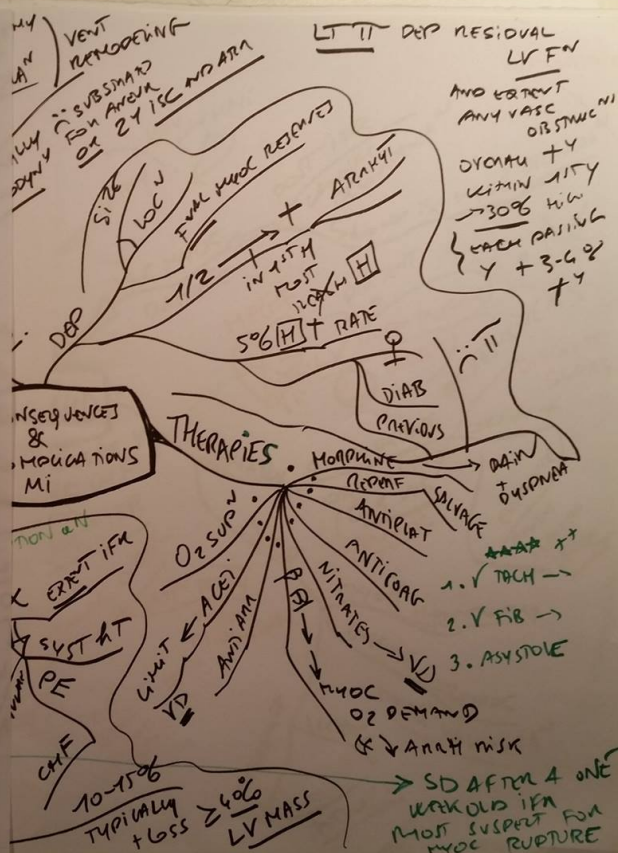


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MI

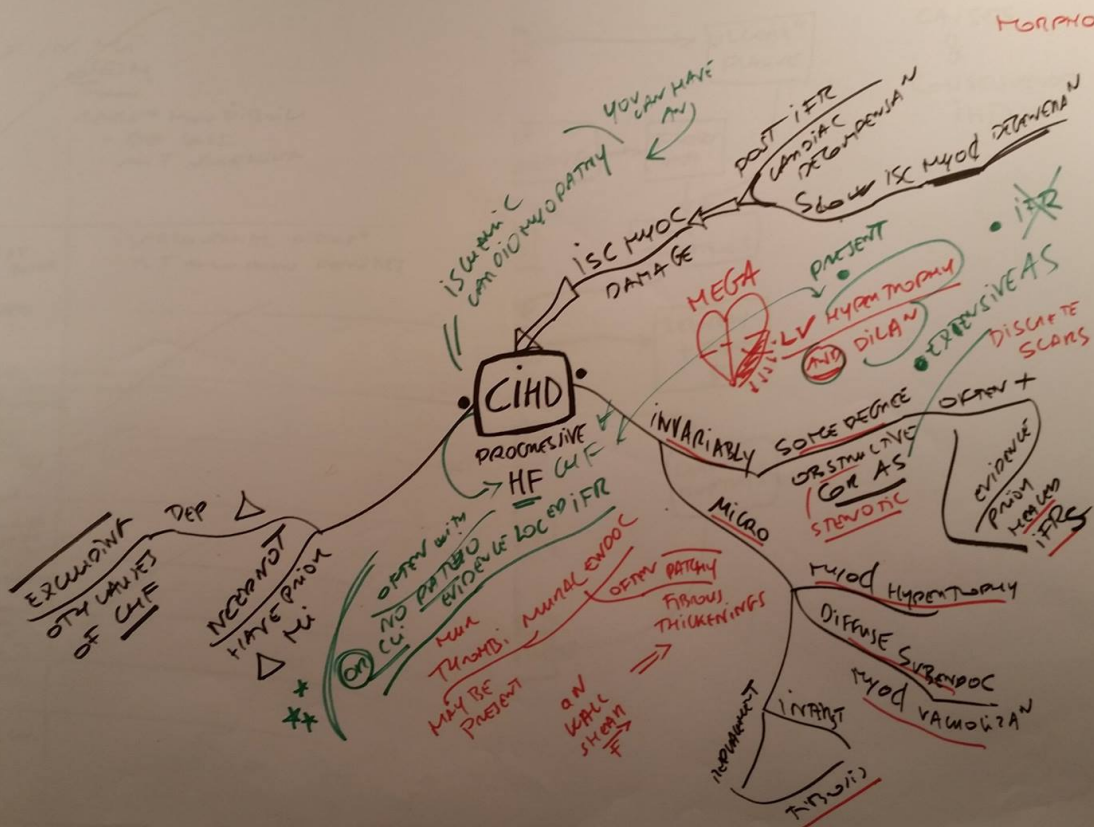




CONSEQUENCES & COMPLICATIONS

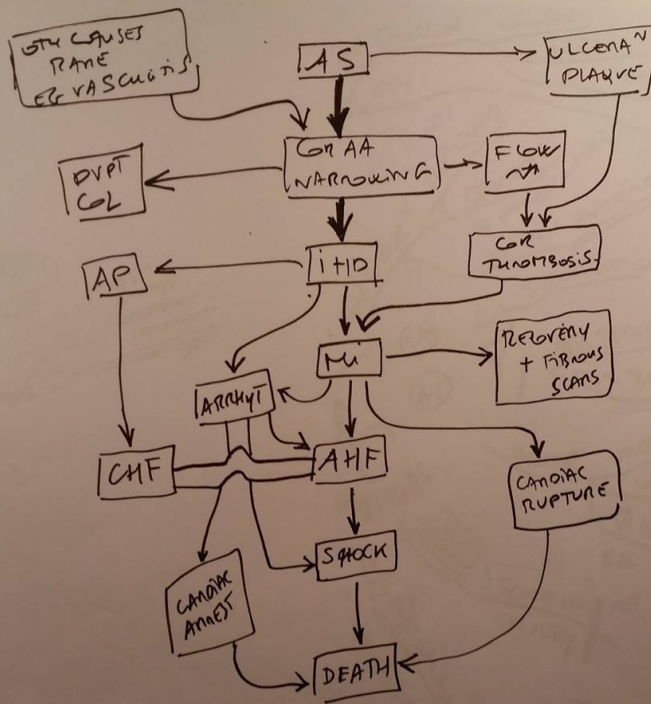


Evolution		
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9	9	9
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87	87	87
88	88	88
89	89	89
90	90	90
91	91	91
92	92	92
93	93	93
94	94	94
95	95	95
96	96	96
97	97	97
98	98	98
99	99	99
100	100	100



Handwritten notes on the left margin of the page, including:

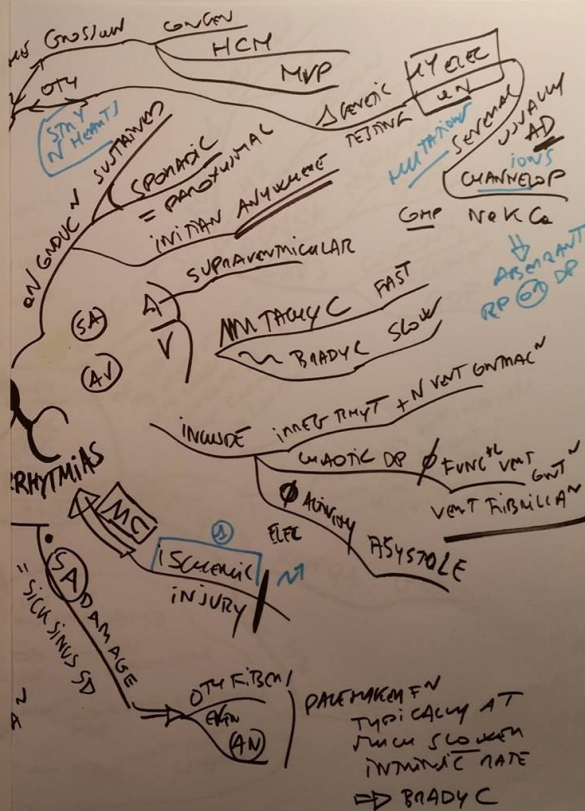
- UB19
- H1 H2
- ANY
- Foram + Diaphragm
- PLAN REL
- SEC
- FW
- ASTA
- AP OD
- GRAPE
- DIAPHR
- WATO
- P → PS
- TO
- DEM
- IP

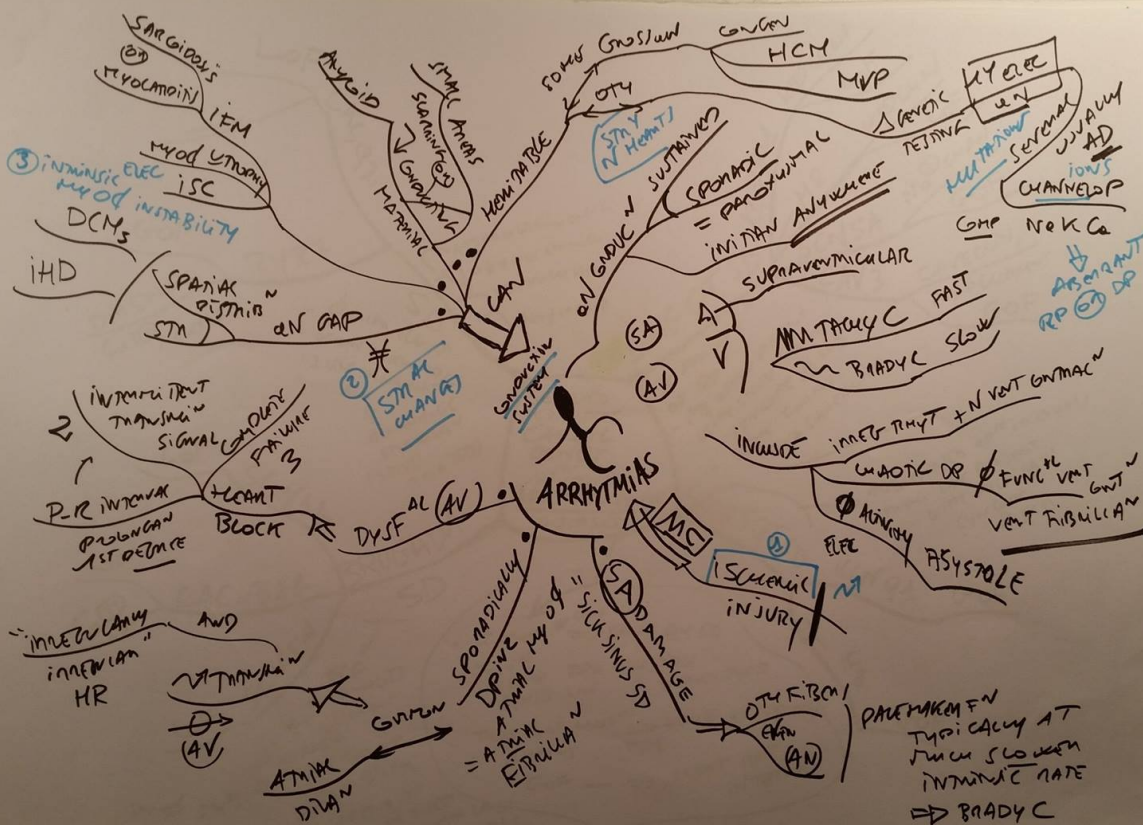


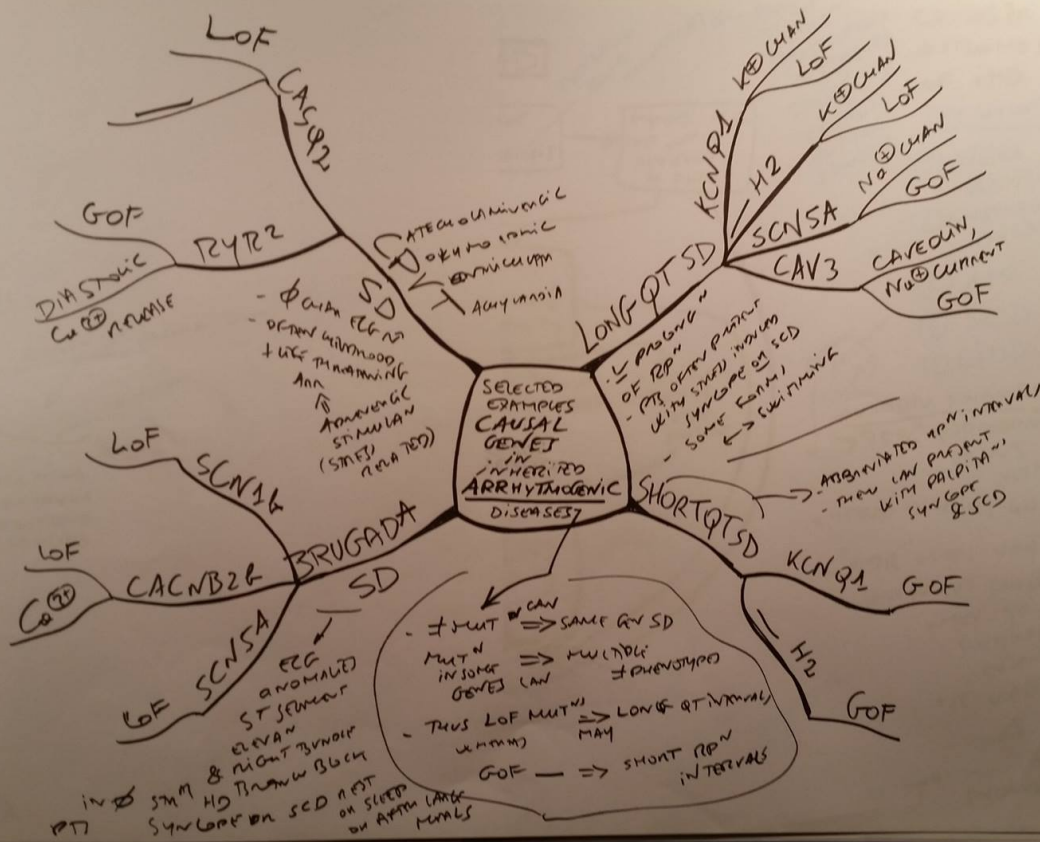
CAUSES
&
CONSEQUENCES
iHD

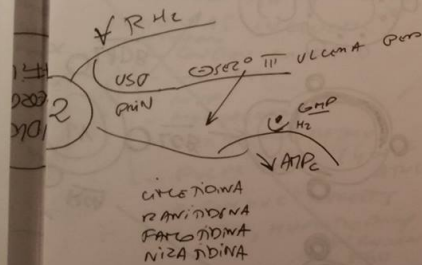
Hand-drawn diagram illustrating the development of the gut tube from the notochord. The diagram shows a central structure labeled 'NEC' (notochord) within a larger mass labeled 'gut tube'. The gut tube is shown as a tube-like structure with a central lumen. The diagram is labeled with 'gut tube' and 'gut tube'.

ARRHYTHMIAS

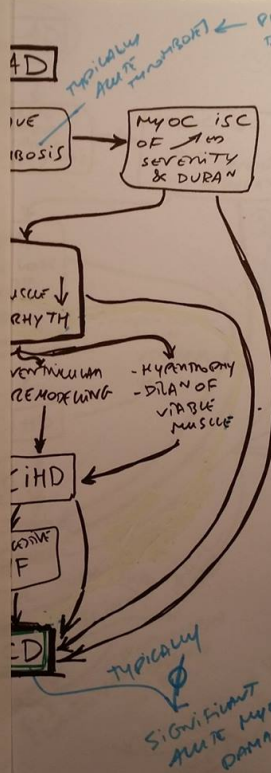








SCD



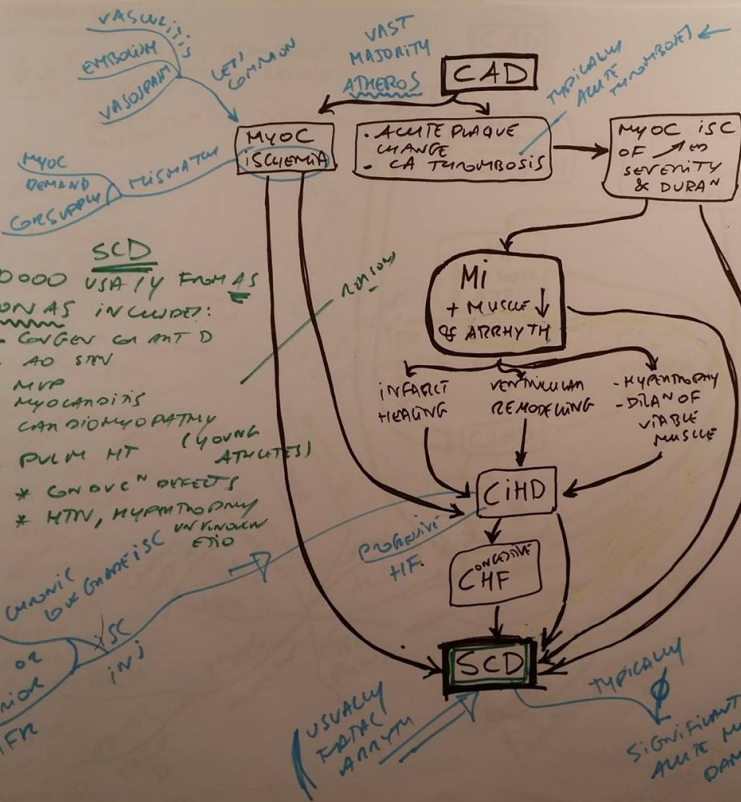
CAUSES & OUTCOMES OF IHD

- ① ANGINA PECTORIS
- ② UNSTABLE ANGINA
- ③ AMI
- ④ SCD
- ⑤ CHD

many previous PS

- AUTOPSY FINDINGS SCD
- 75% NARROWING OF L3 VESSEL
 - HEART INFARCT 40%
 - "ARR" often very GROSSLY CONCURRENT WITH NO AVLT FINDINGS PRESENT
 - IF WEABRGT

? T^u MOCV



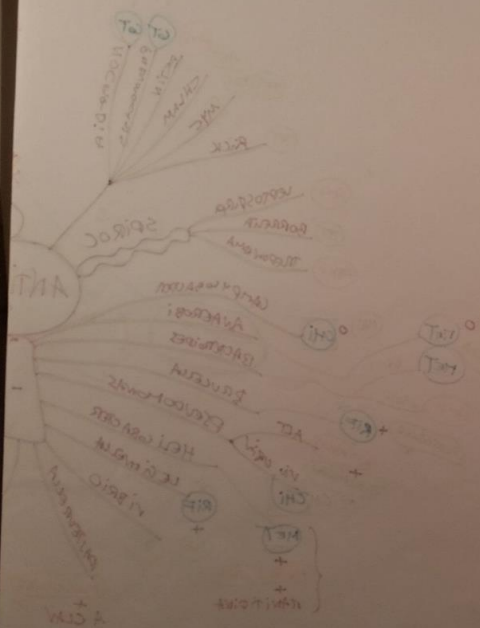
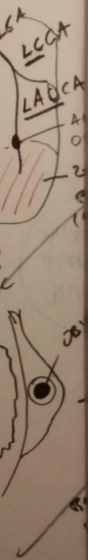
- SCD**
- 350,000 USA 14 FEMAS
 - NON AS INCLUDES:
 - CONGEN CA ANT D
 - AO STN
 - MVP
 - MYOCARDIOTIS
 - CAN DISORD PATRY
 - PULM HT (YOUNG ATHLETES)
 - * CONGEN DEFECTS
 - * HTN, HYPERTROPHY UNKNOWN ETIO

CAUSES & OUTCOMES OF IHD

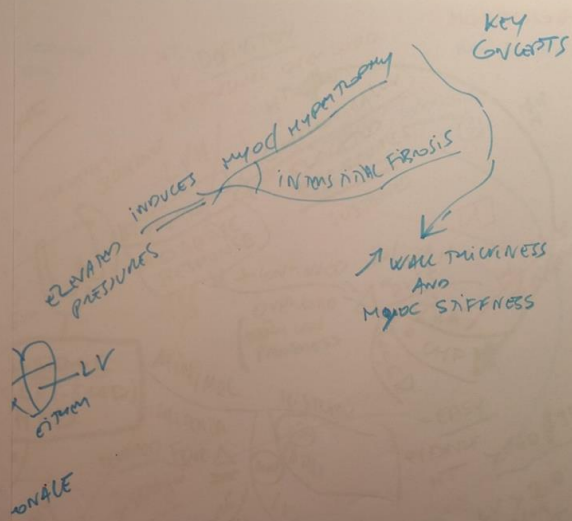
- KEY CONCEPTS
- 1) ANGINA PECTORIS
 - 2) UNSTABLE ANGINA
 - 3) AMI
 - 4) SCD
 - 5) CIHD

- MAJOR FINDINGS
- 75% NARROWING OF L3 VETRI
 - HEART INFARCT 40%
 - "ARR" OETRY VMY CONVIERT CON LUMEN WHEN NO ANET FINDINGS PRESENT * IF WEABASHT

? T_{MI} MOLAT

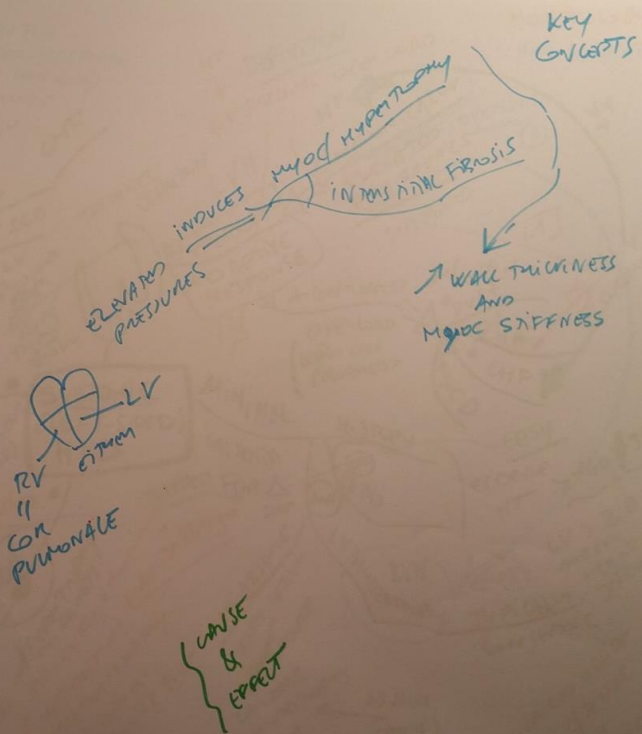


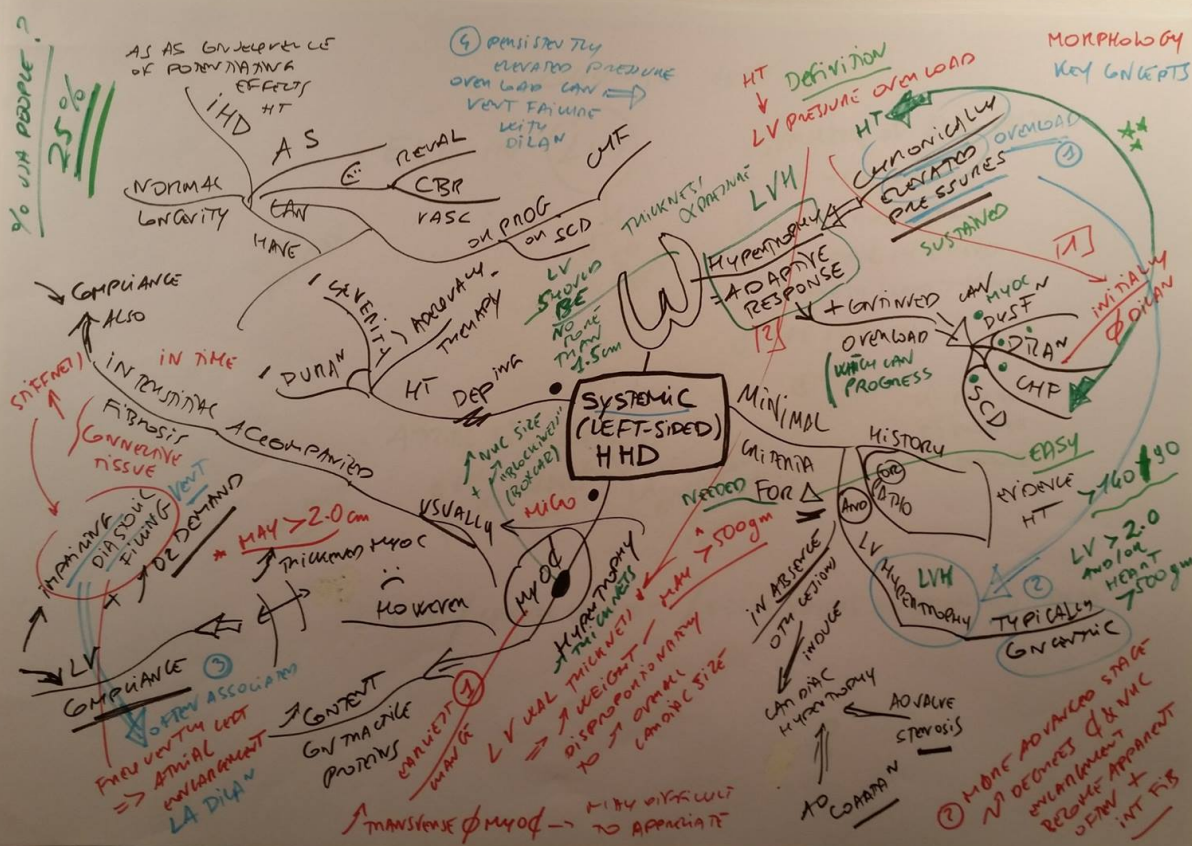
HHD



CAUSE & EFFECT

NO CARDIA
OC
ANTI
PULMONARY
+ CLAV





CLINICAL LEFT

• EKG in **LVH**

WMM
ANALYSIS

NOT
SO
EASY

SUMMARY OF LVH CRITERIA

- (1) $R-I + S-II > 25mm$
- (2) $S-V4 + R-V4 > 35mm$
- (3) ST-T DEPR. in L LEAD
- (4) R WAVE in L LEAD $> 11mm$
- (5) LAE + OTHER CRITERIA

⊕ VE CRITERIA

- 1 = POSSIBLE
- 2 = PROBABLE
- 3 = DEFINITE

• ATRIAL FIBRILLATION

obvious

NO P-WAVE

LA

ENLARGED
DILATED
"STRETCHING"

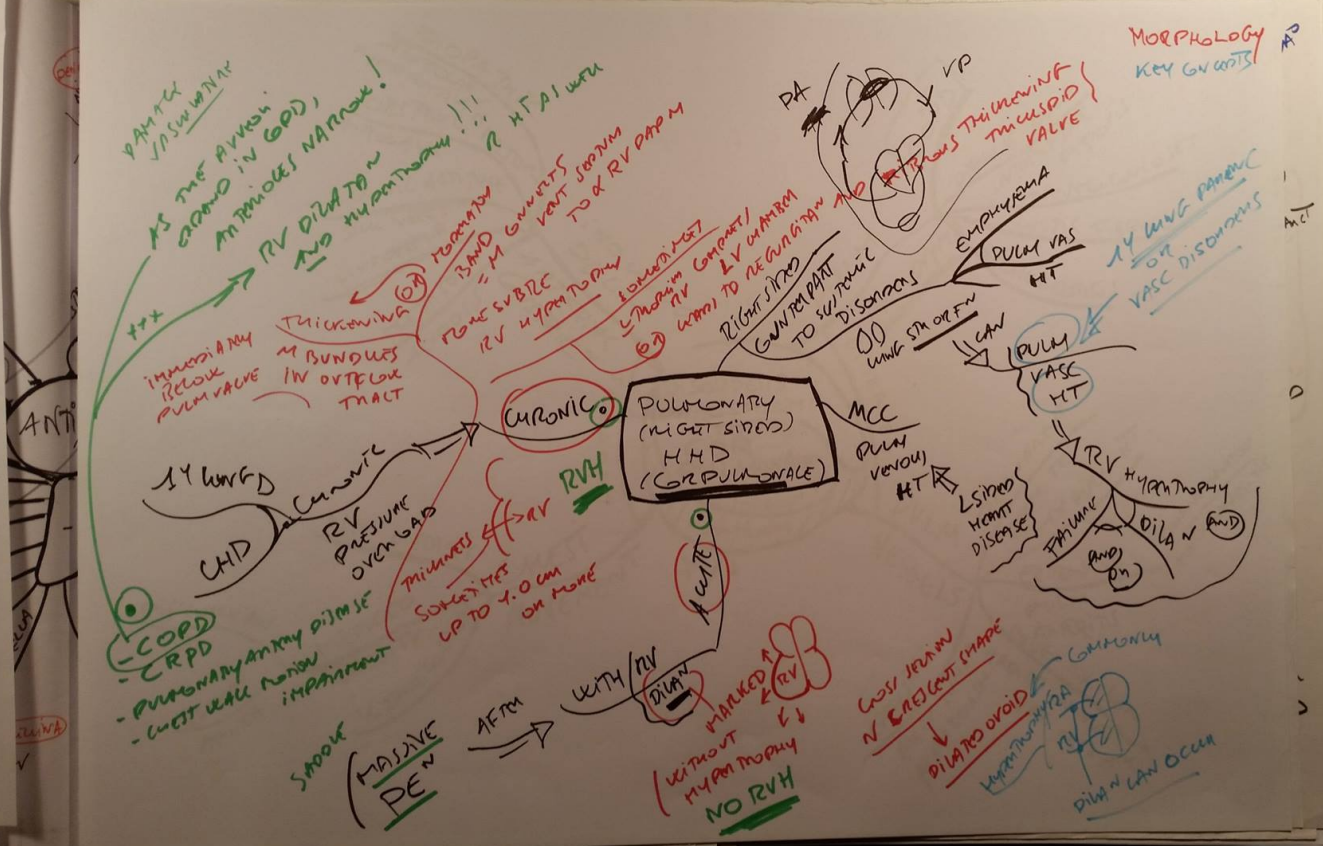
• CHF / CARDIAC DILATATION / PULM VENOUS CONGESTION AND DILATATION

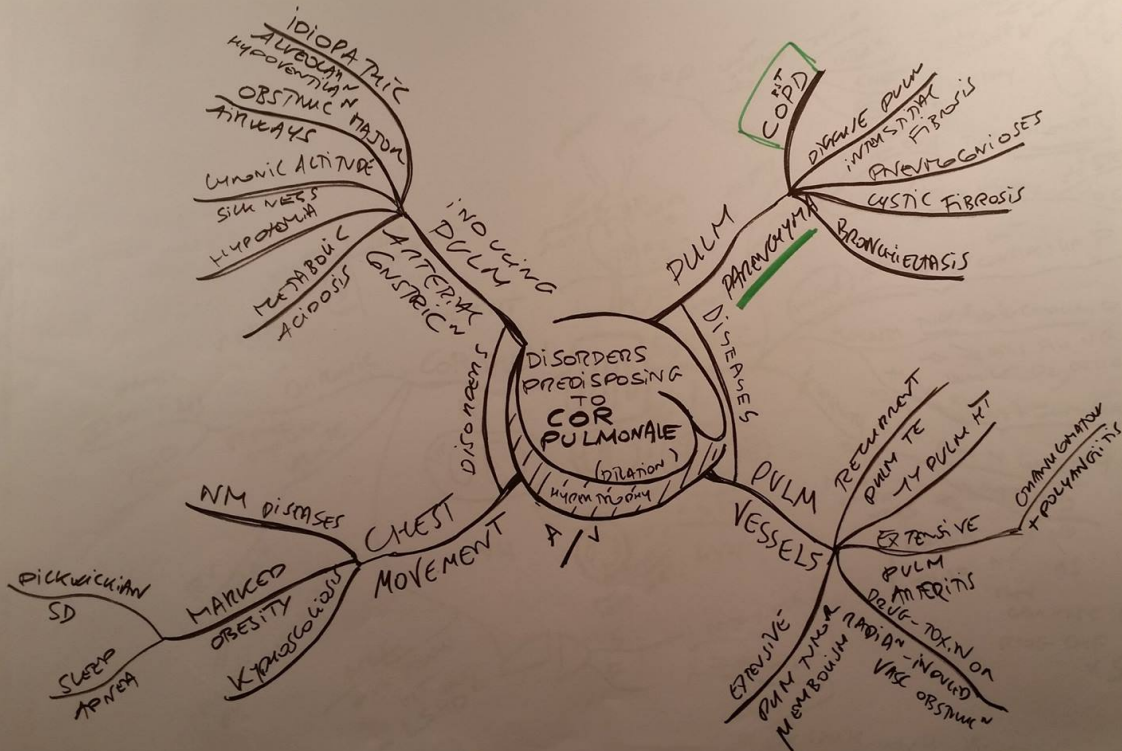
EDV?

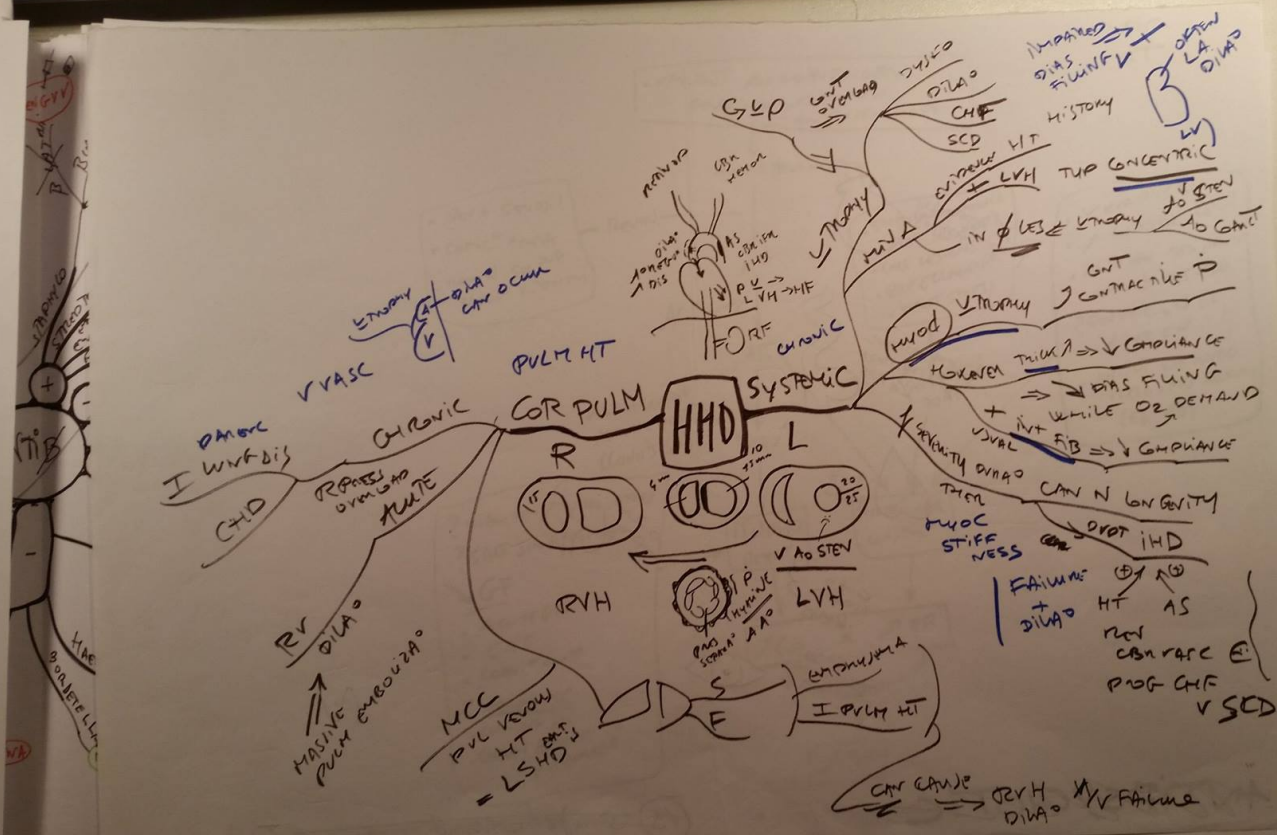
COURSE LEFT

- NORMAL LONGEVITY $\neq$ OTH CAUSE
 - PROGRESSIVE IHD
 - PROMINENT NEW DAMAGE
HEMORRHAGIC CVA (Ventricular?)
 - CHF
- $\approx$ COLD
THERM
CAUSE
- VENTRICULO
IN BASE
GROW OUT

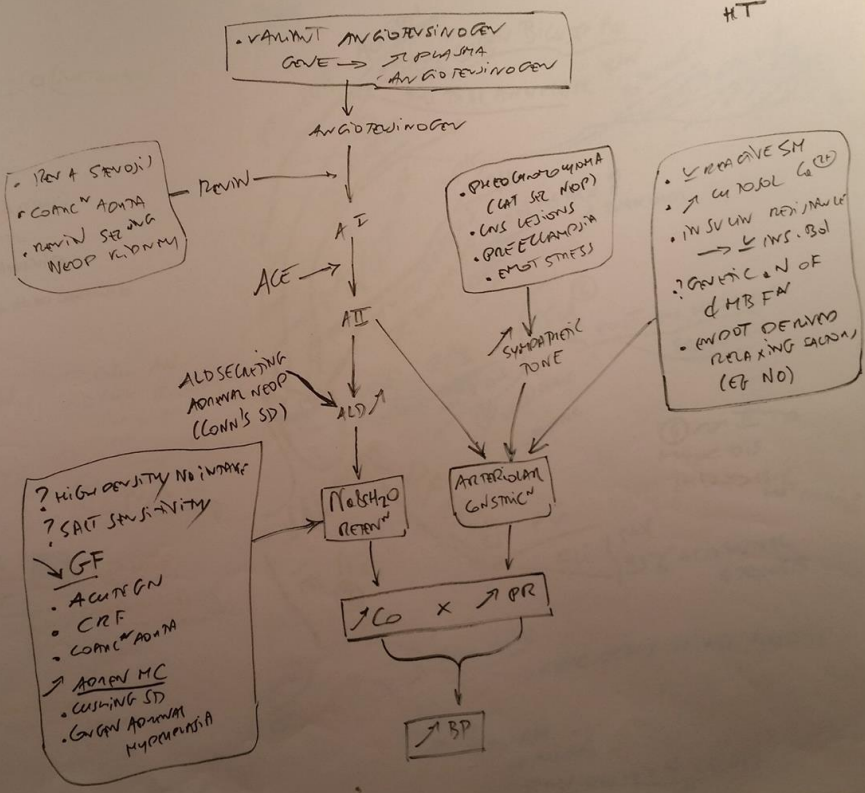




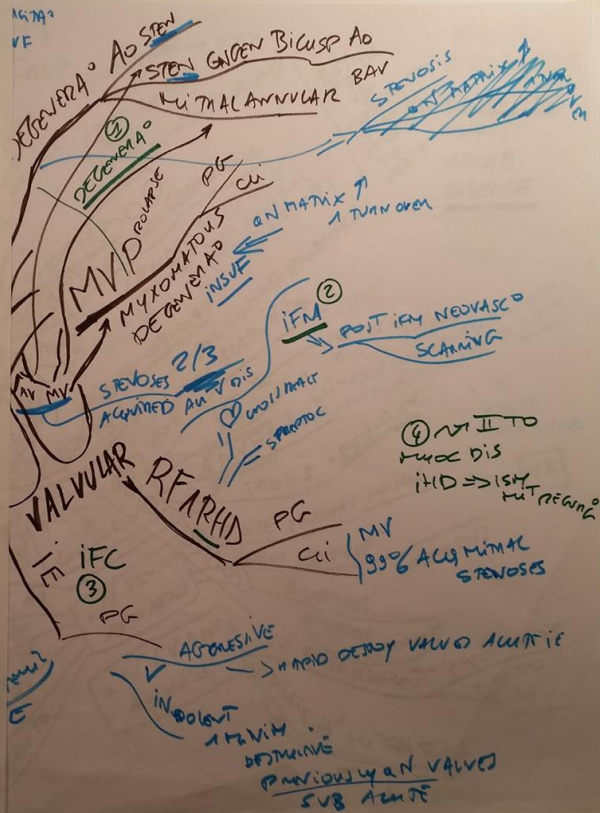


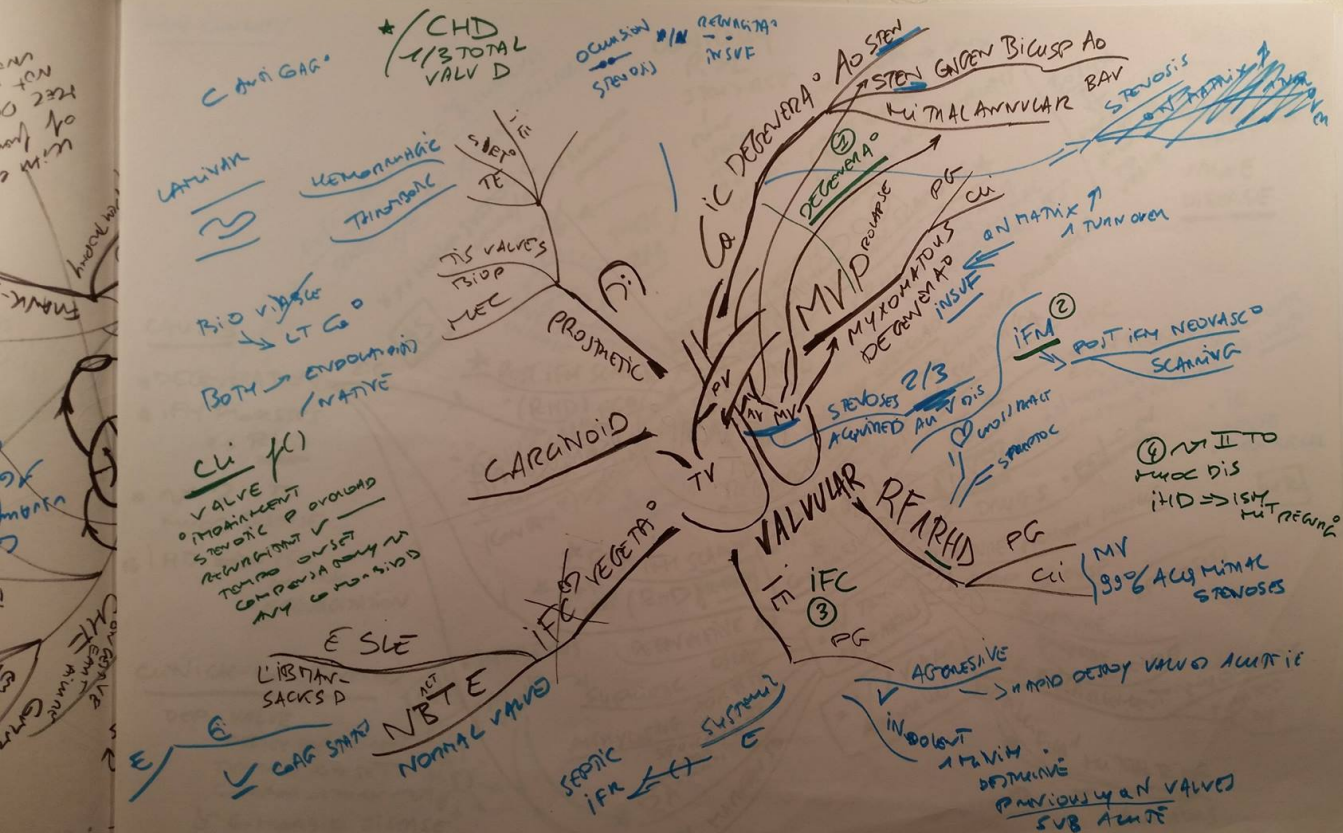


PG
HT

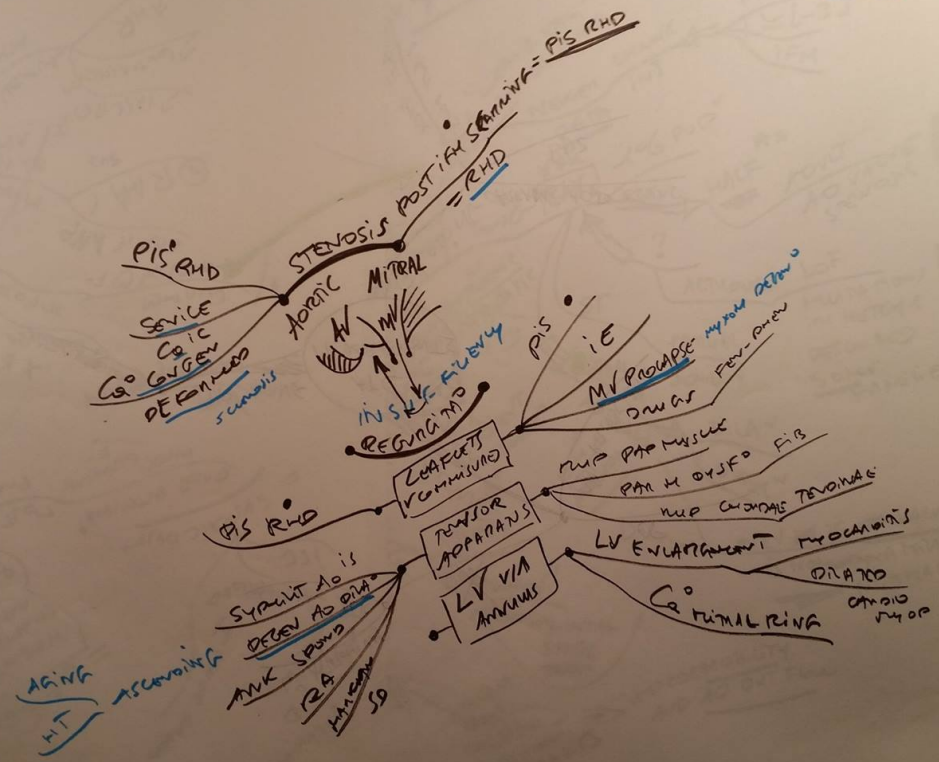


VALVULAR ✓ HD

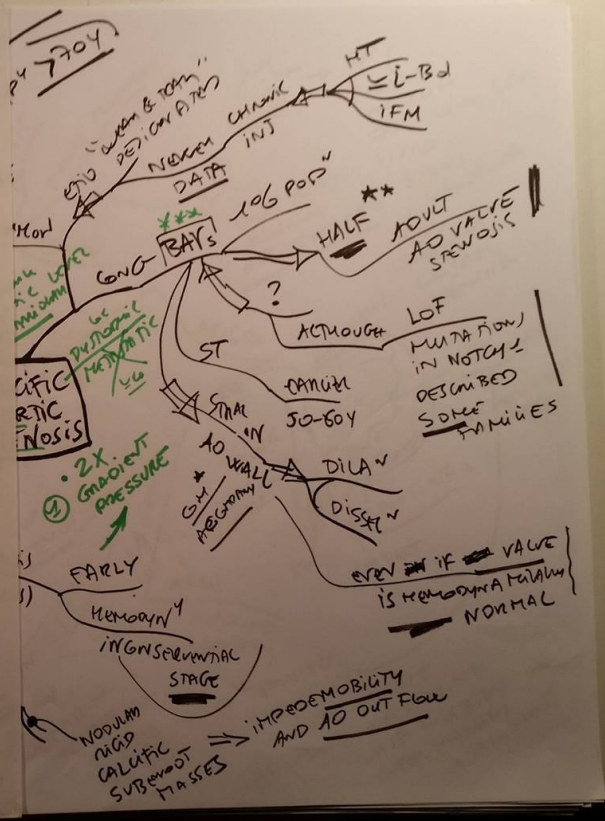
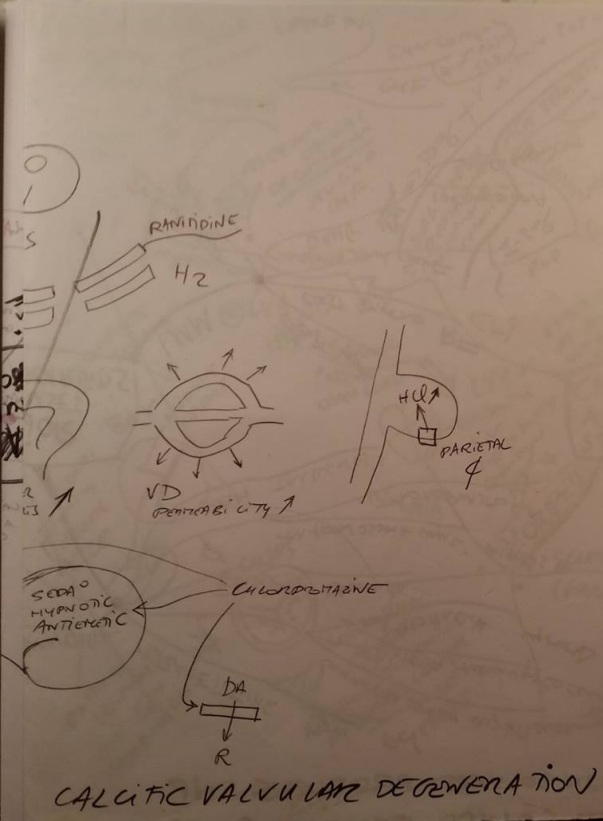


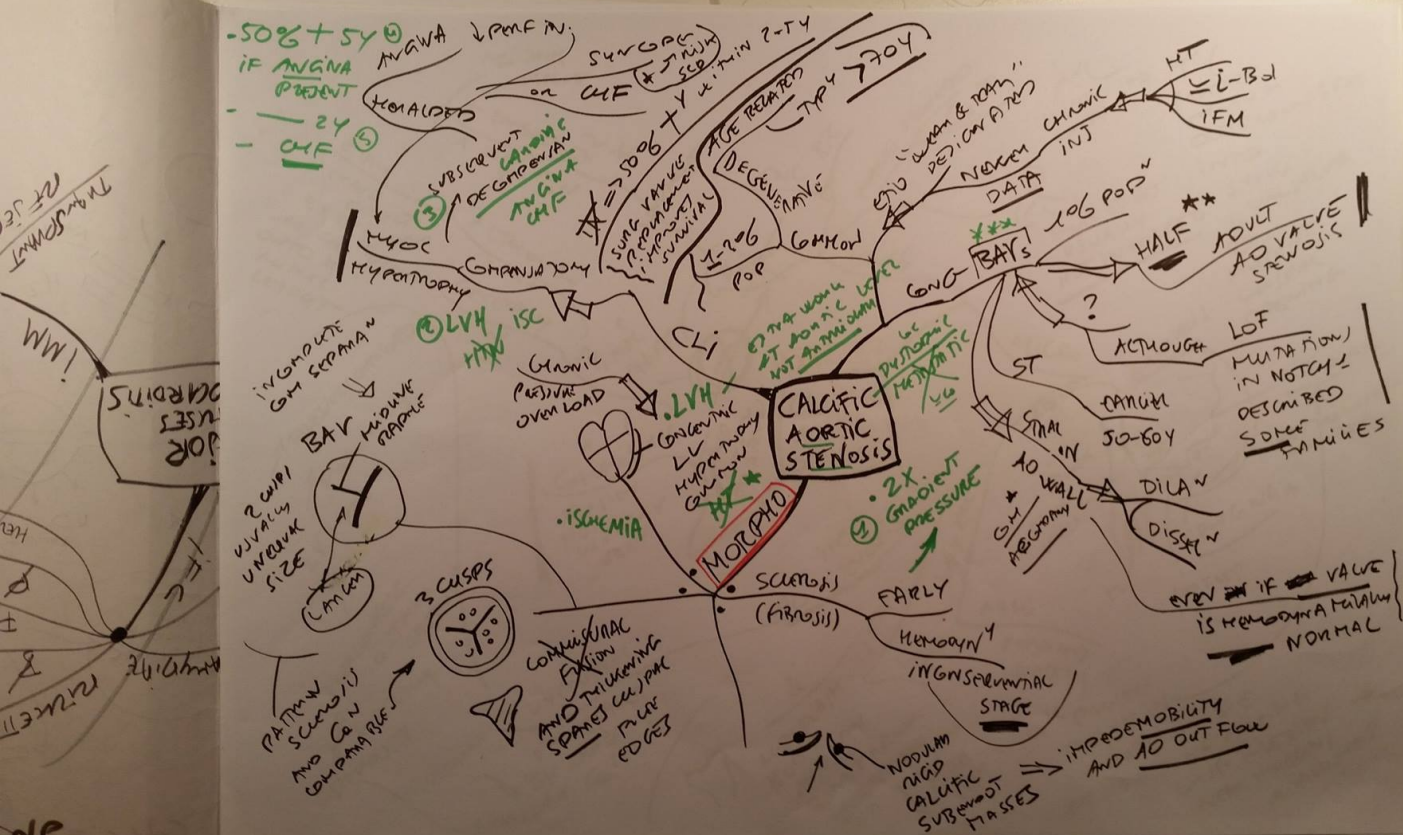


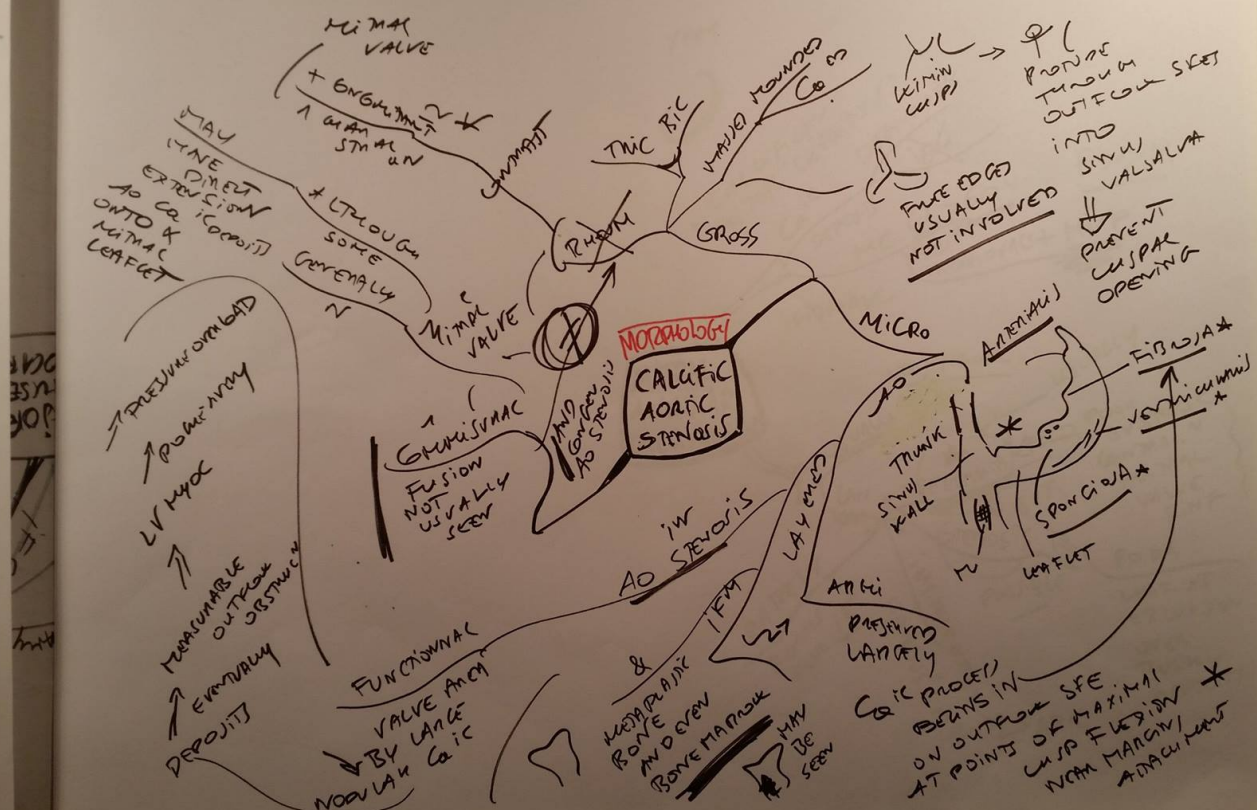
MOST
COMMON



HYPERTENSIVE
 HT
 ESTROGENS (E)
 ANDROGENS
 PREGNANT / POSTNATAL (P)
 AGO







XRAY



• calcification
mitral skeleton

Ca⁺⁺ deposits
MC Q760
INDIVIDUALS + MVP

F >> M

idiopathic

MITRAL ANNULAR CALCIFICATION

NO DYSFUNCⁿ

USUALLY

USUALLY ϕ

REGURGITATION

BVT

POOR SYSTOLIC CONTRACTION

MITRAL VALVE RING

RARELY

Focus for IE

IMPAIRMENT CONDUCT PATH

POSSIBLE

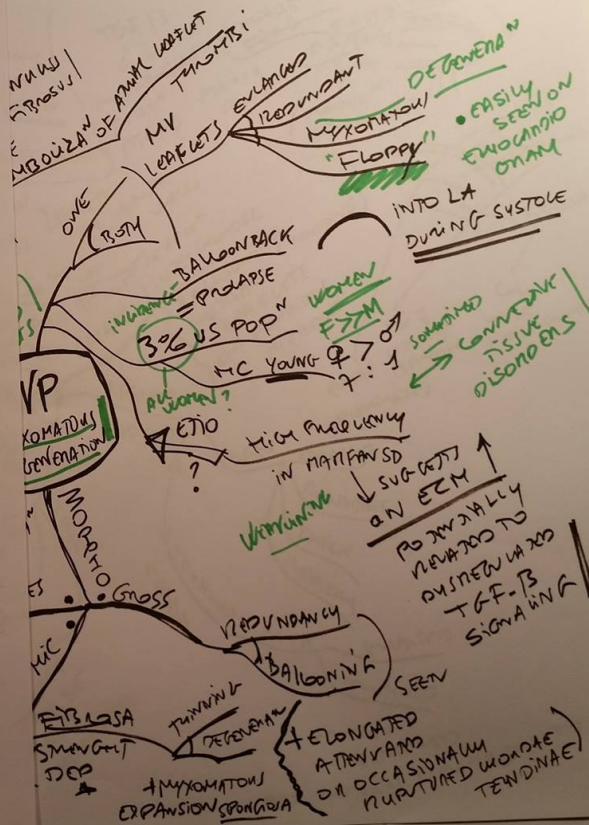
POOR

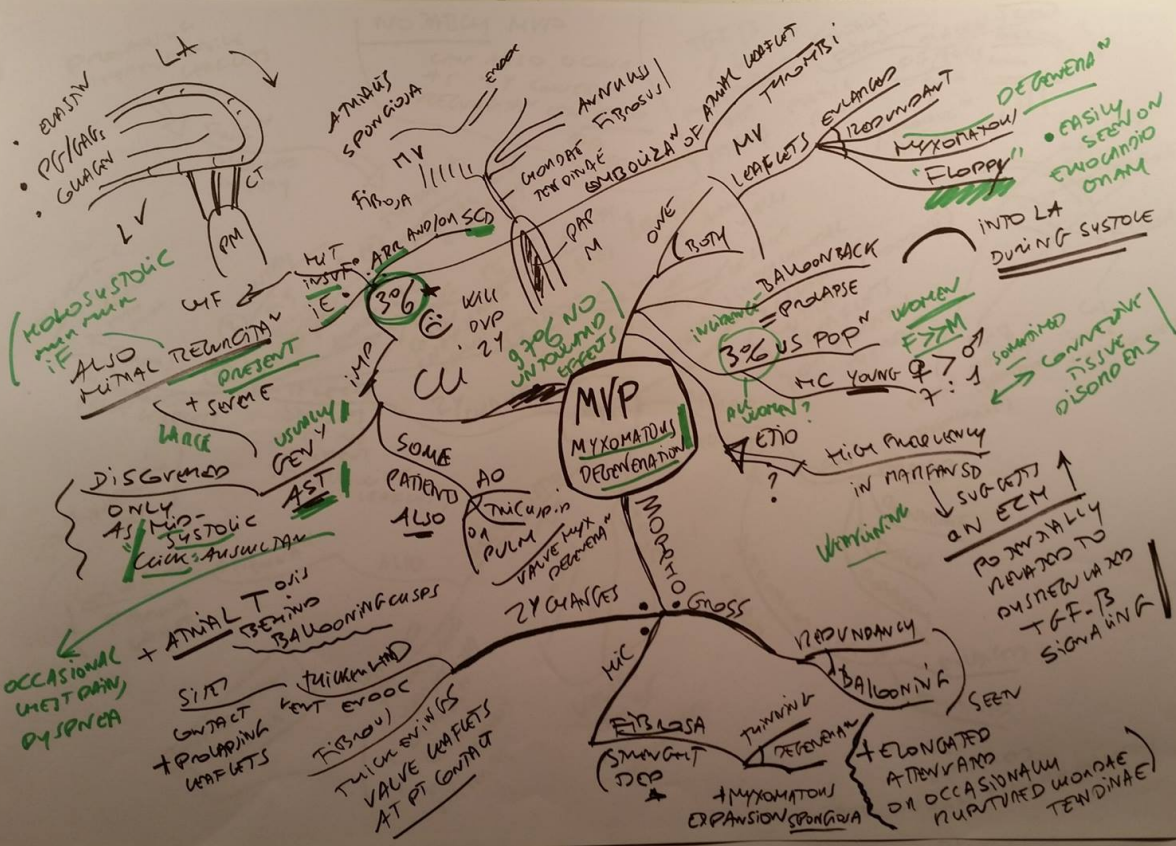
LEFT VENT EXCURSION

OVER BULKY DEPOSIT

APR

MVP (MYXOMATOUS DEGENERATION)





Handwritten notes on the left margin, including chemical structures and text fragments.

Handwritten notes on the left margin, including chemical structures and text fragments.

PROLAPSED HYPERMORPHIC LEAFLET

Friction
INDUCES INJURY

NOTABLY MVP

CAN ALSO OCCUR
AS 2Y GUSEP
RECURRING DTH END

E.G. ISC DYSF

TGF β

↓

MODULATING

PMMP

ATGEP

ATRIALIS

ENAB

PG/ECGS

COL

PRODUCN

1 PLACENTARY

ELASTIN + COL

↓

MATRIX

PRODUCN

↓

ATRIALIS

SPONGE

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ELASTIN

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PG +

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++ KNOCK
OUT
AGAINST
KNOCK
DOWN

Fig 10.10

THICKENING

VALVE LEAFLET

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STAIN INTRINSIC

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COLLAGEN

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FIBROBLAST

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HYPERTROPHIC

100% RHL (E)
SARCOIDOTIC P

DEFECT E TRANSFER
FROM MUTOC TO SANC
A/V DIRECT SANC OXIFO

HYPERTROPHIC PHEN

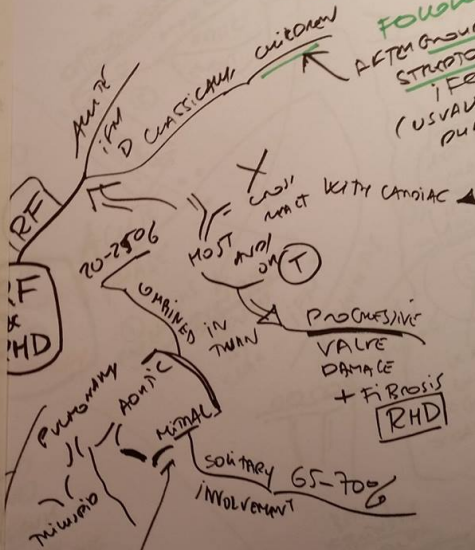
- ASYM FERTAL HYPOMY
- MYOF DISARRAY
- FIB, MYOS ARTERIOCLEROSIS
- LV OUTLINE MARK PLATE
- THICKENED SEPT VENTR

CLINICAL
HF
SD
MYO FIBROSIS
DYSPLASIA

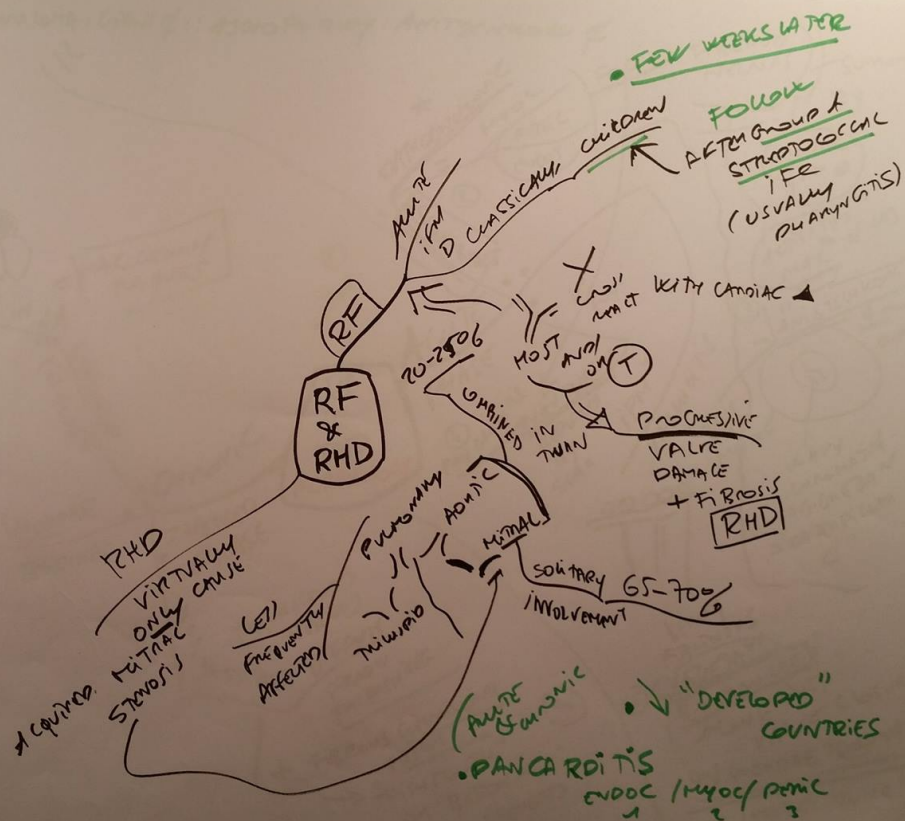
RF & RHD

• FEW WEEKS LATER

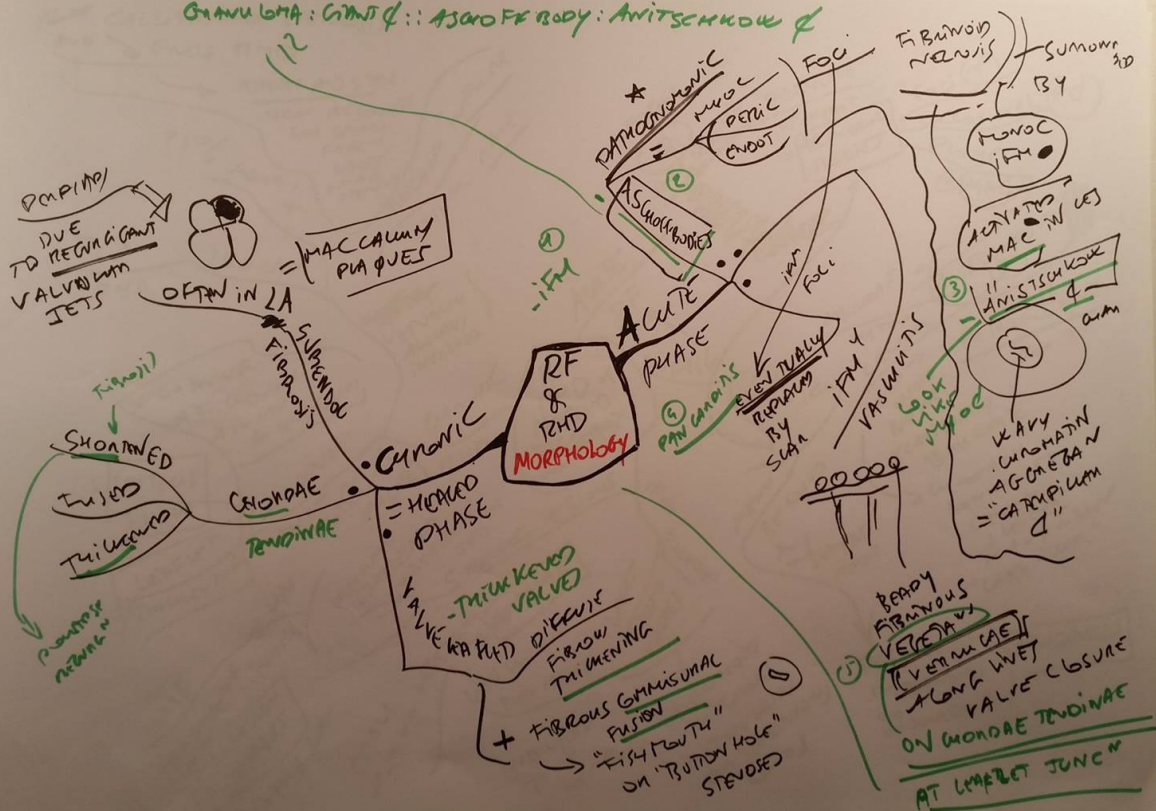
FOLLOW
AFTER GROUP A
STAPHYLOCOCCAL
IFE
(USUALLY PHARYNGITIS)

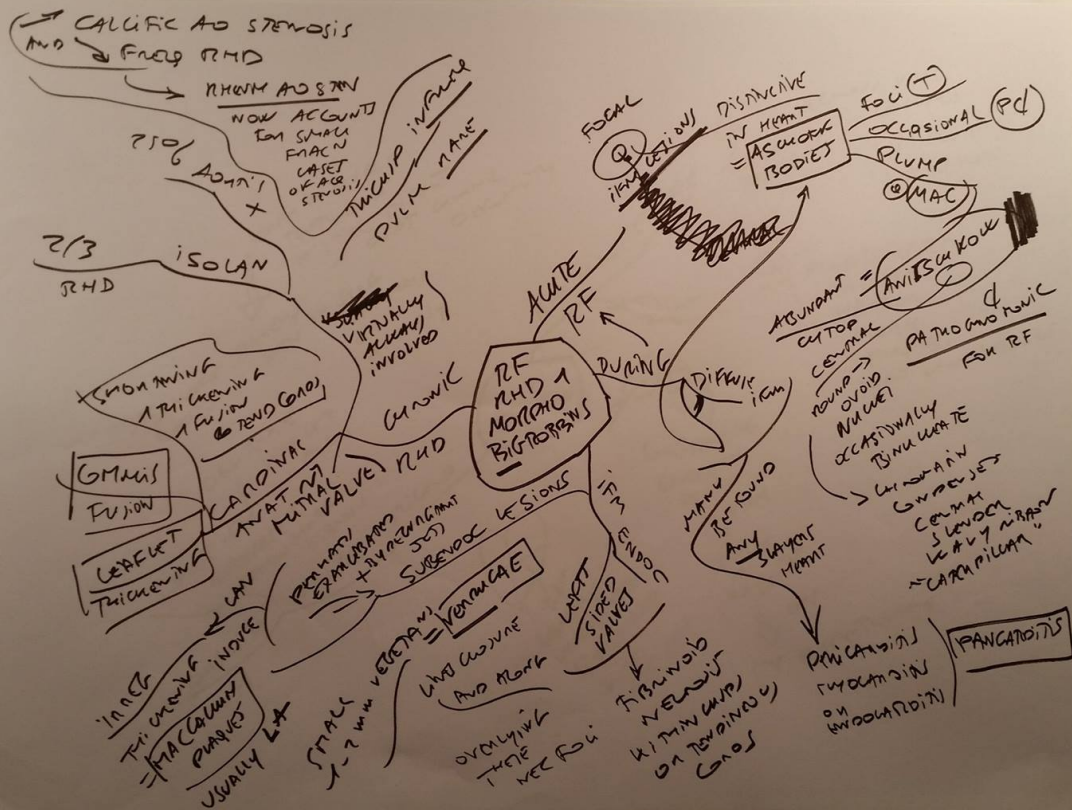


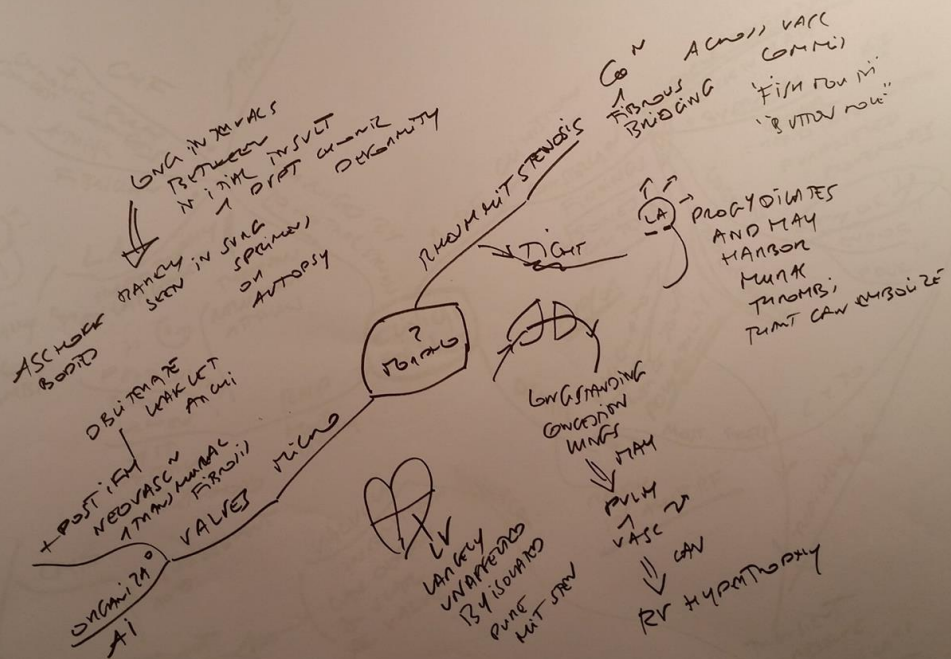
• "DEVELOPED" COUNTRIES
• PANCAARDITIS
ENDO / MYOC / PERIC

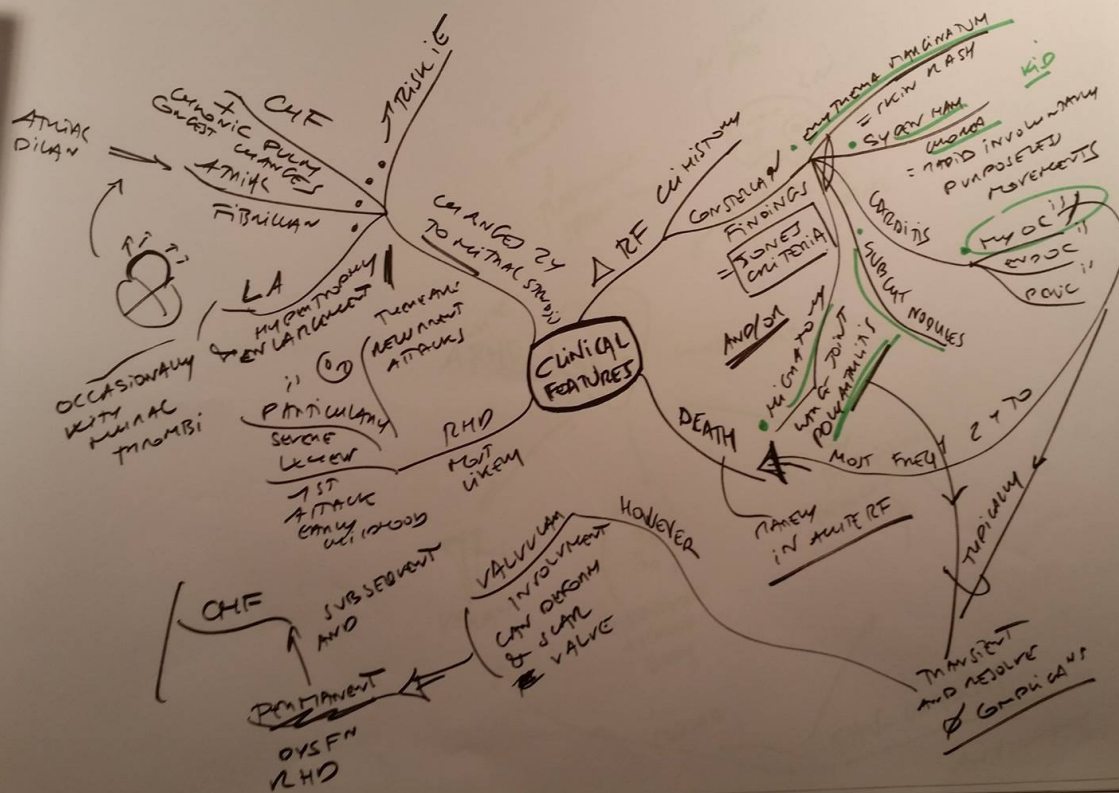


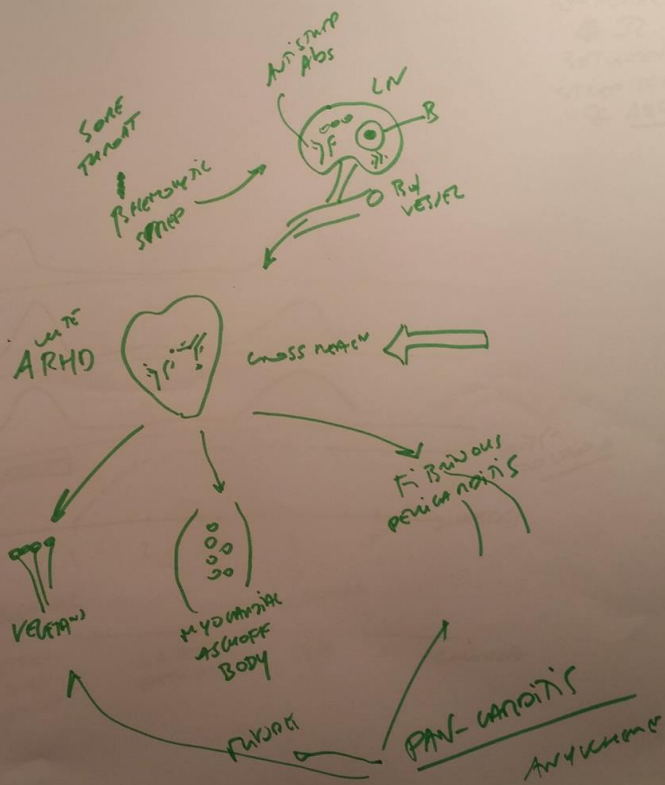
112



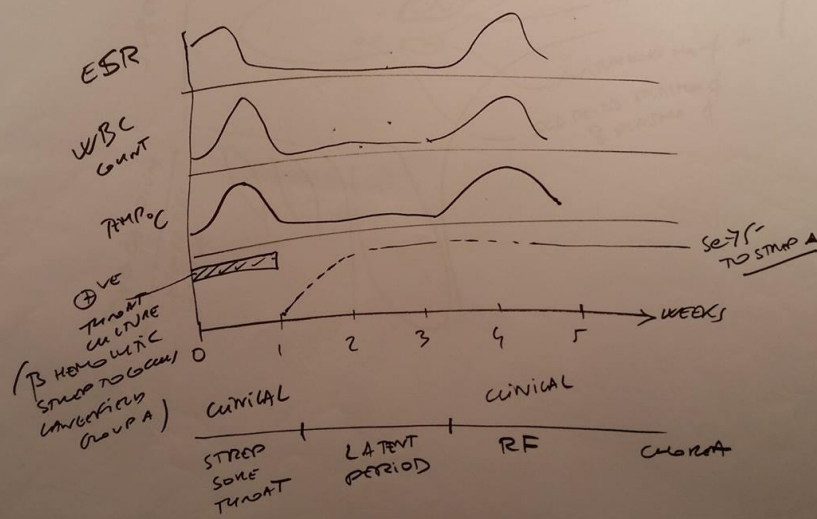


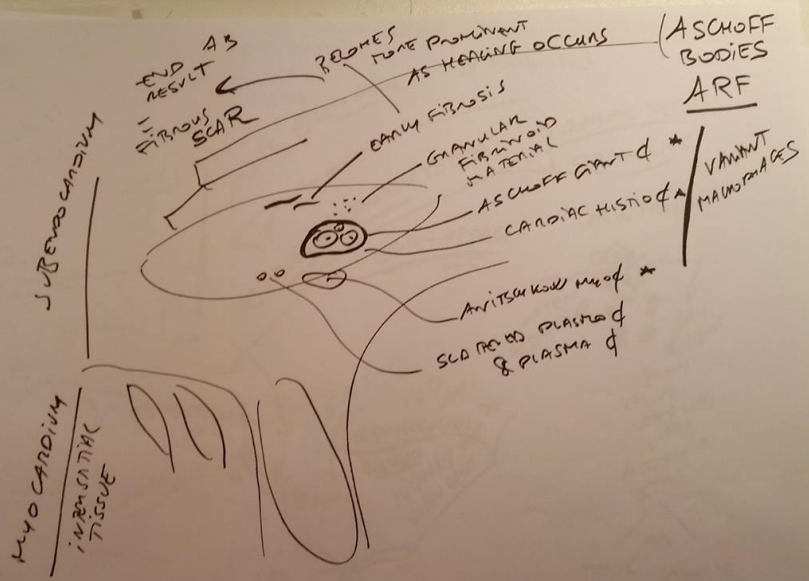






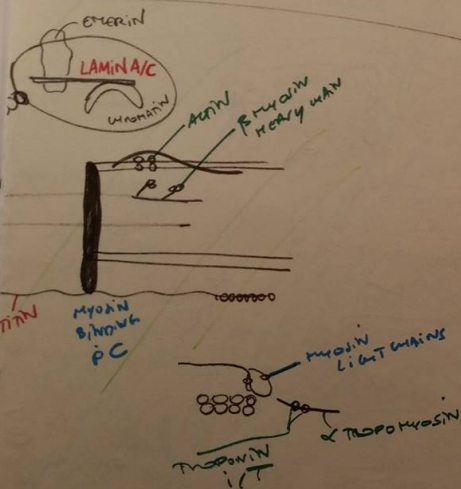
TEMPORAL
RELATIONSHIP
BETWEEN
STREP IDC
& ARF



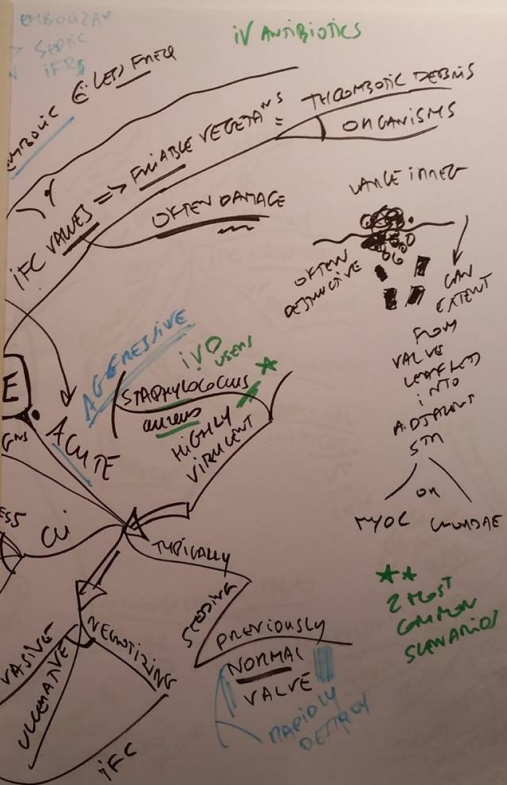


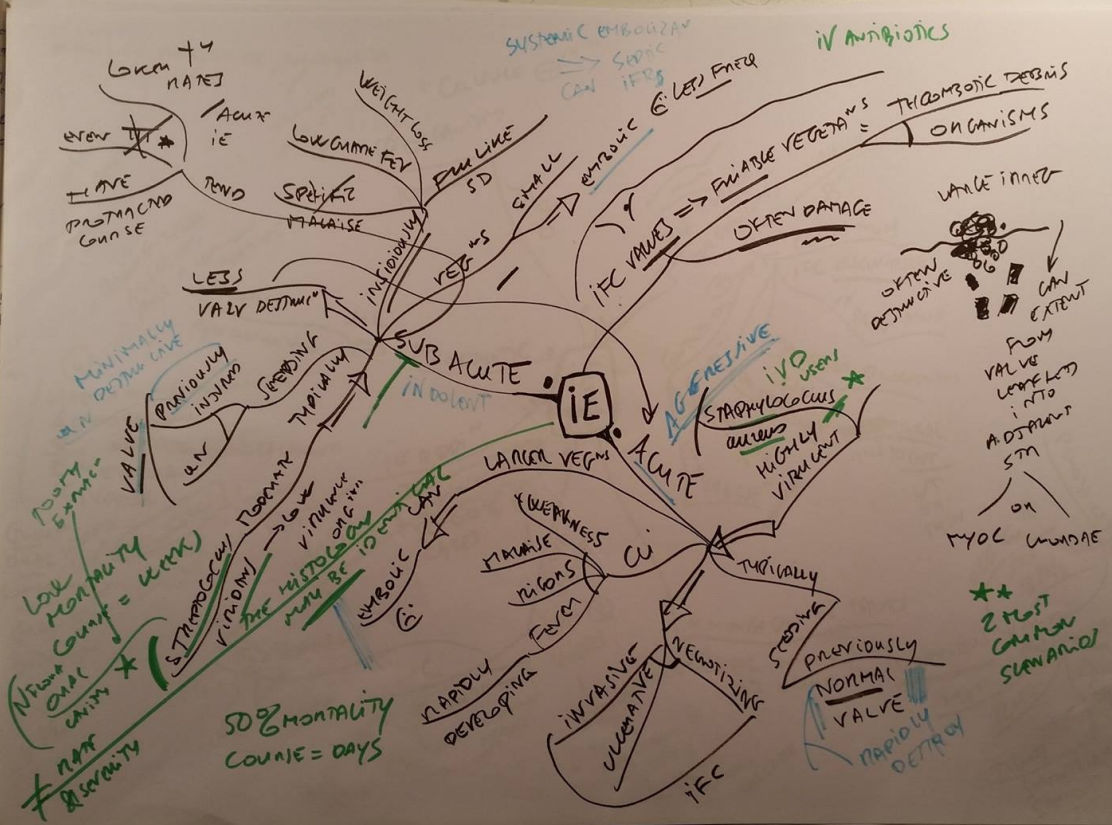
P MUTATED
 DILATED
 HYPERTROPHIC) BOTH
 MYOCHS

ELM



ie





FRANK NICKETTSIDE, GLANDY DIA

14/10/16
POTENTIAL

Culture $\ominus$ "ve"

ORIGIN

10/6

SEVERAL RING
ABSCESSES
FUS + GRAN
STAPH

epidermal

MC

PROSTHETIC

IE
PG

BO BONE
ON ORN (line)

DERIVE FROM
IFC ELSEWHERE

IFC ELSEWHERE

IFC ELSEWHERE

IFC ELSEWHERE

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IFC ELSEWHERE

④
HABITUS
ACQUISITION
EIKENAL
KINGSTON

GROUP
ONAL
CHROMATICS

GROUP
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CHROMATICS

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CHROMATICS

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CHROMATICS

GROUP
ONAL
CHROMATICS

IN ADDITION
TO SINUSITIS
50-60% CASES

USUALLY

RESIDUAL

LEFT IFC

N VALVE

MC

S. aureus

OFF

IVD ②

USERS

ECHOCARDIOGRAPHY

+ FIRM

SEPSIS

+ STAPH

EMUS

W/OUT

ALTHOUGH CAN N VALVE

IFC NON KEY

TO OCCUR IN

PREVIOUS

MVP

RHD

RIGHT

SHORTLY

STEROIDS

+ JERKING

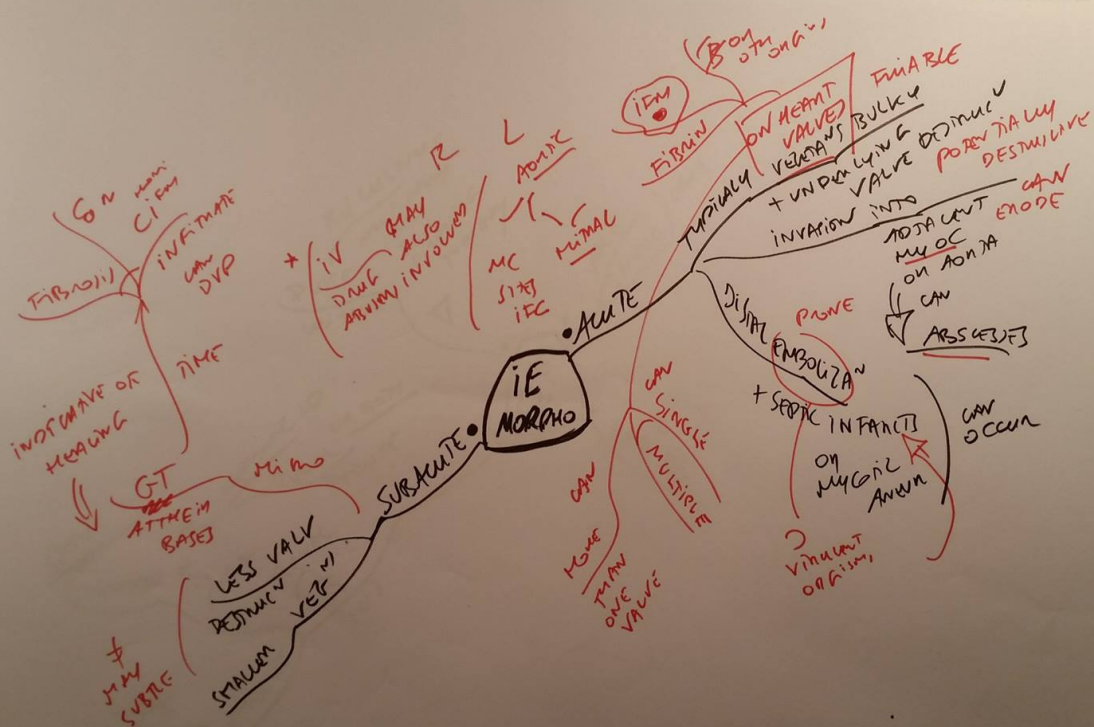
PROSTHETIC

DEE C/IC

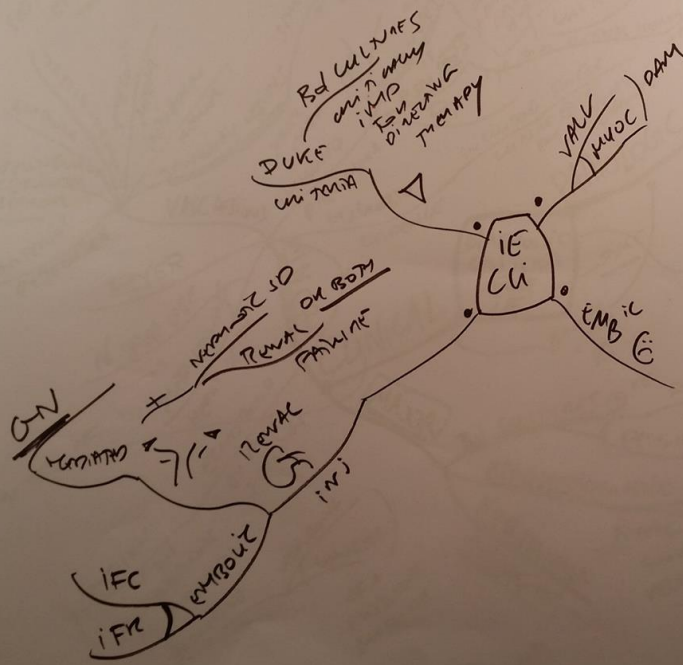
STAY

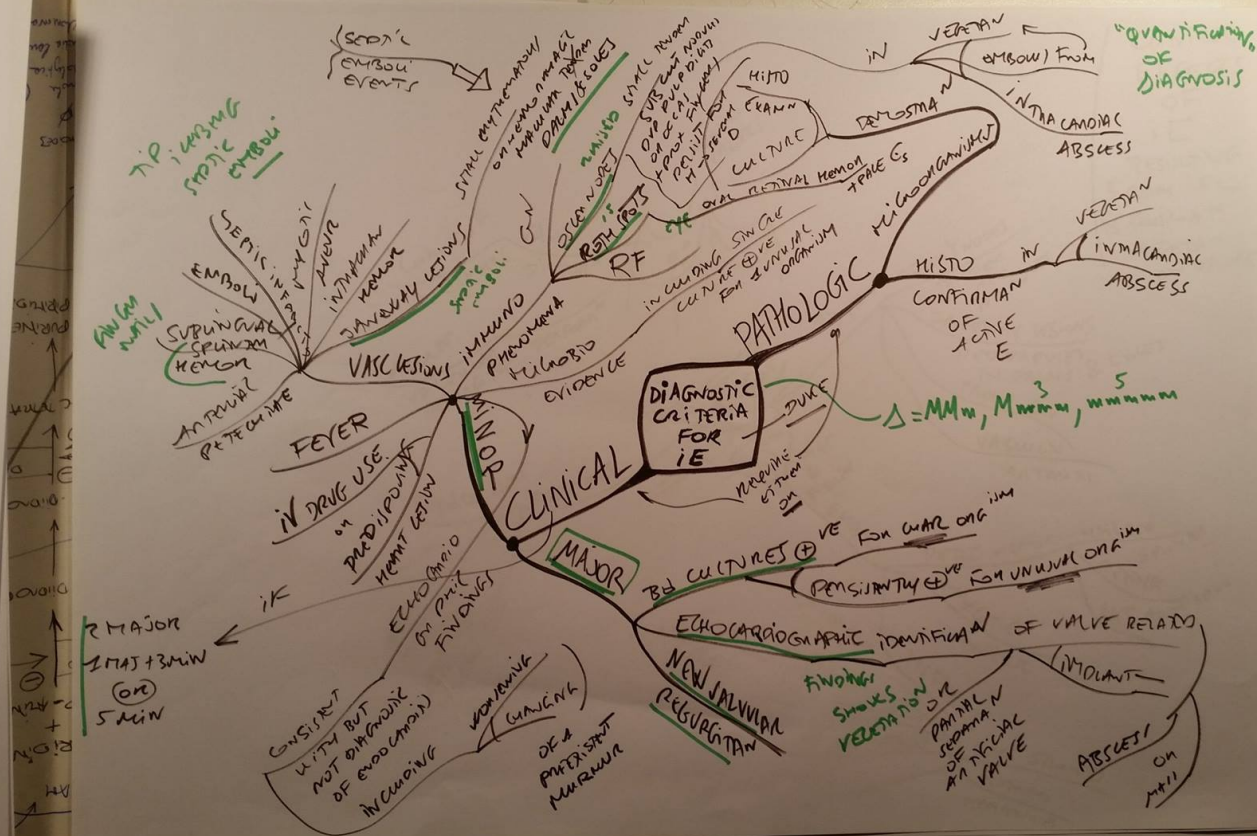
BNS

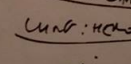
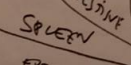
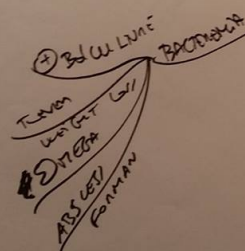
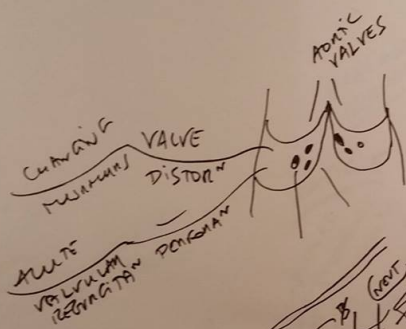
VALVE



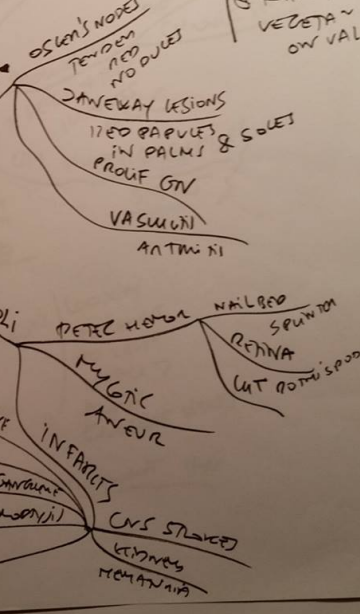
Понравилось

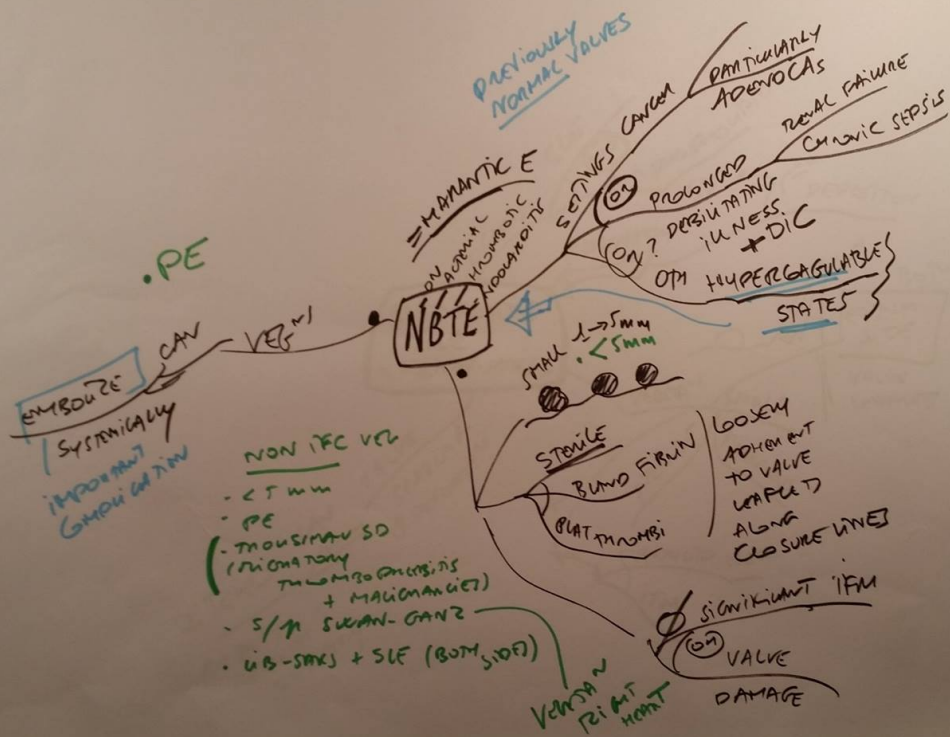
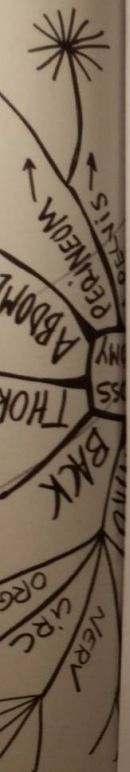


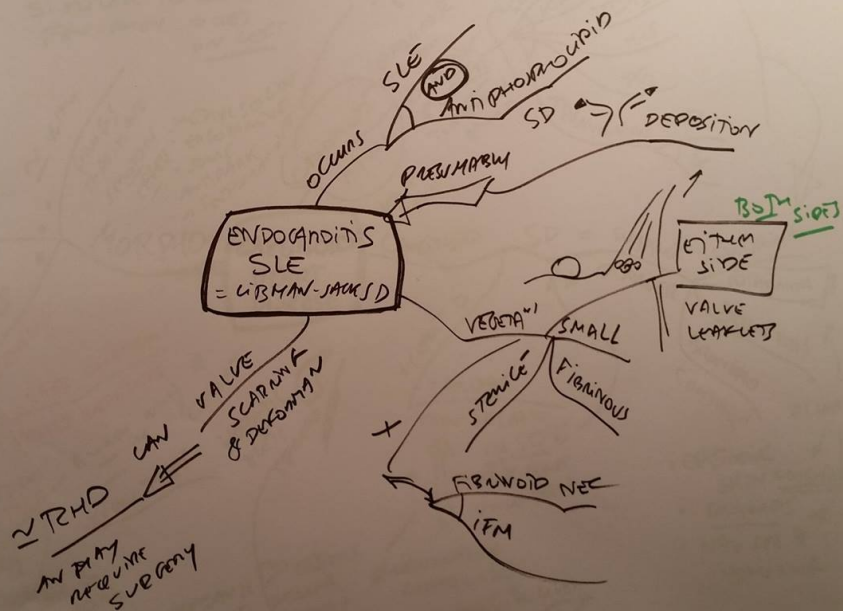


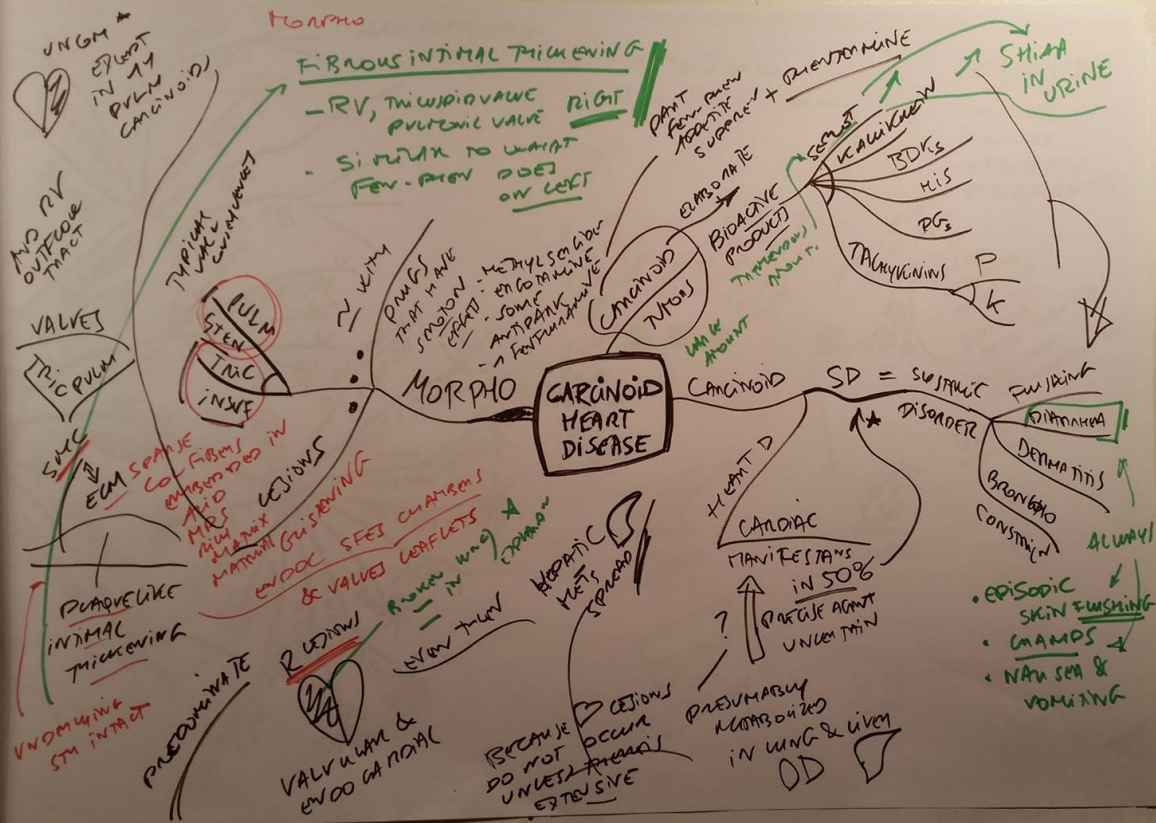


Clinical effects of IE resulting from IE & FEMAN VEGETATION ON VALVE









Blood side

CHORDAE TENDINEAE JUNCTION

AS IE RESOLVES SUBSTANTIAL SCARRING CAN RESULT

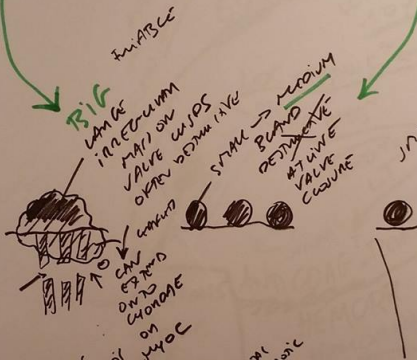
small leaflet vegetation along line closure valve leaflets

FEVER PHASE



RHD

- INFECTIVE > 5mm
- NON-INFECTIVE < 5mm

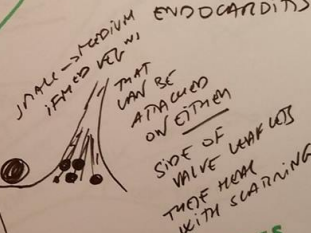


> 5mm ANYWHERE

VBTE

< 5mm

(CA-THE-VAL) SUBVALVULAR IAT TRANSVALVULAR

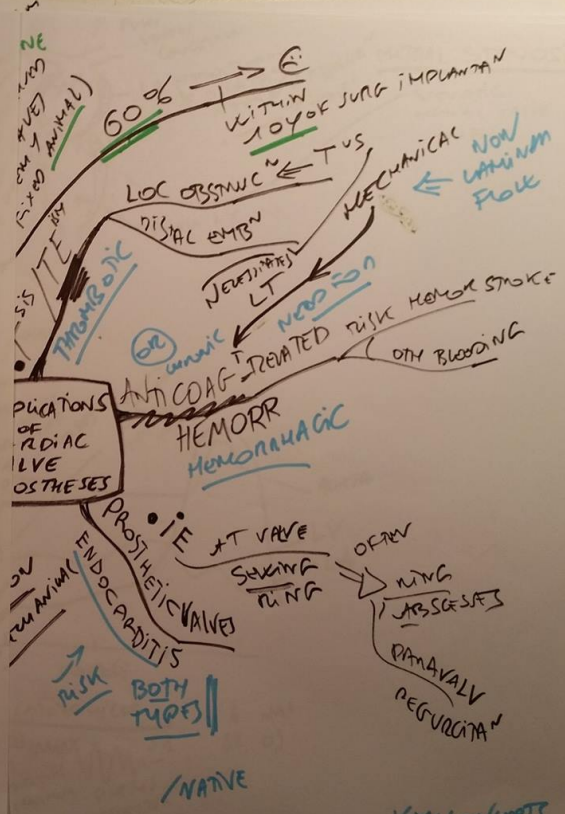


BOTH SIDES

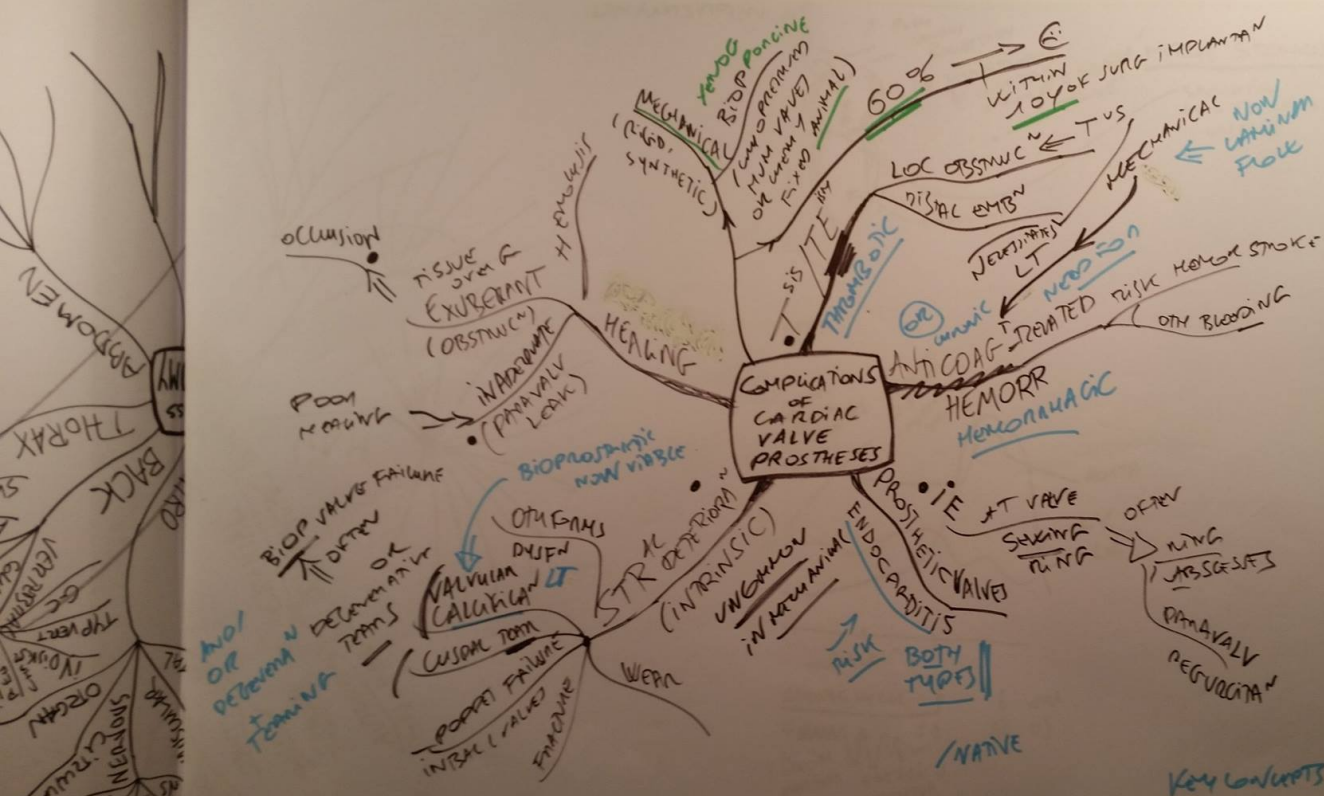
LSE

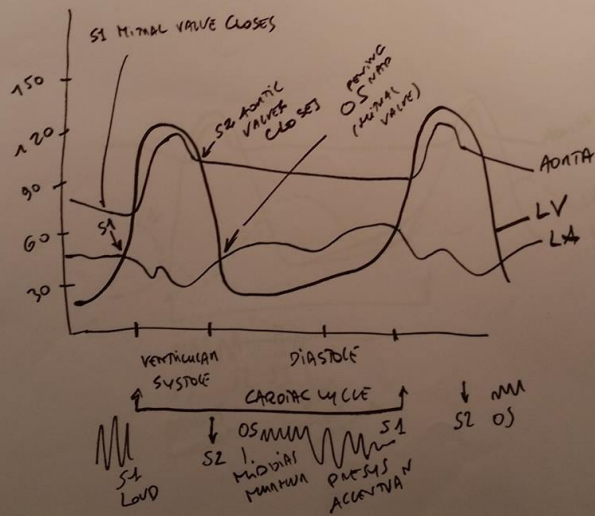
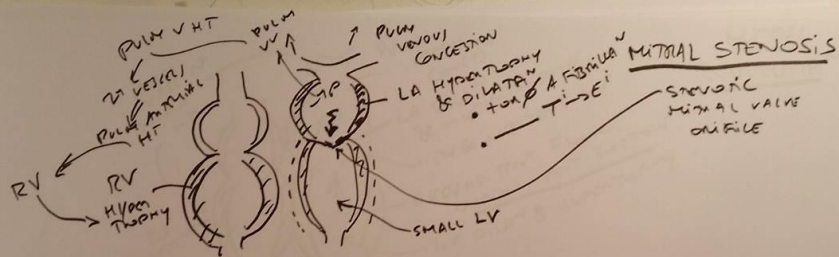
COMPARISON OF 4 MAJOR FORMS VEGETATIVE ENDOCARDITIS

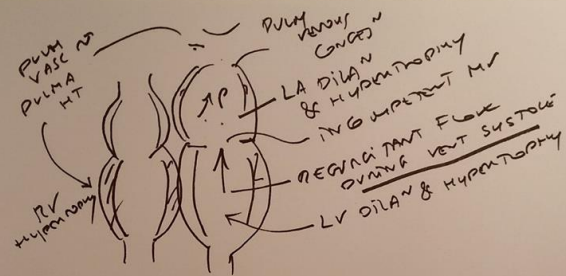
cardiology



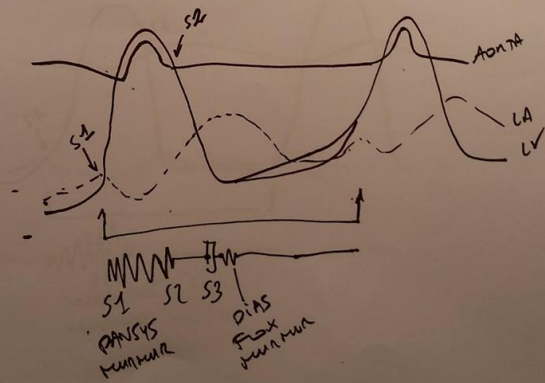
COMPLICATIONS PROSTHETIC VALVES



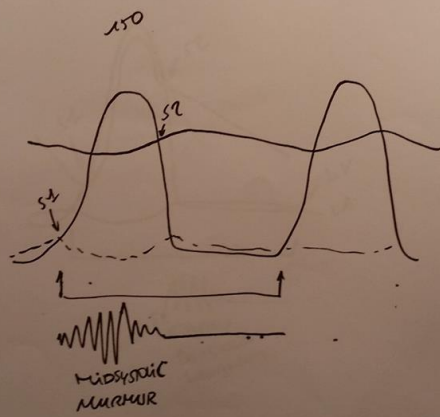




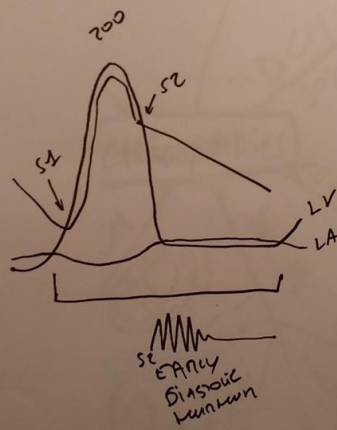
MITRAL
REGURGITATION



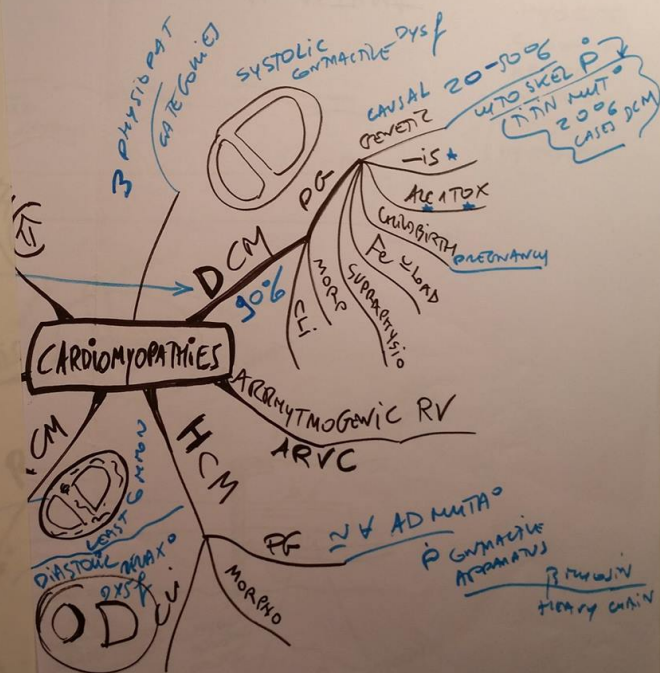
AORTIC
STENOSIS



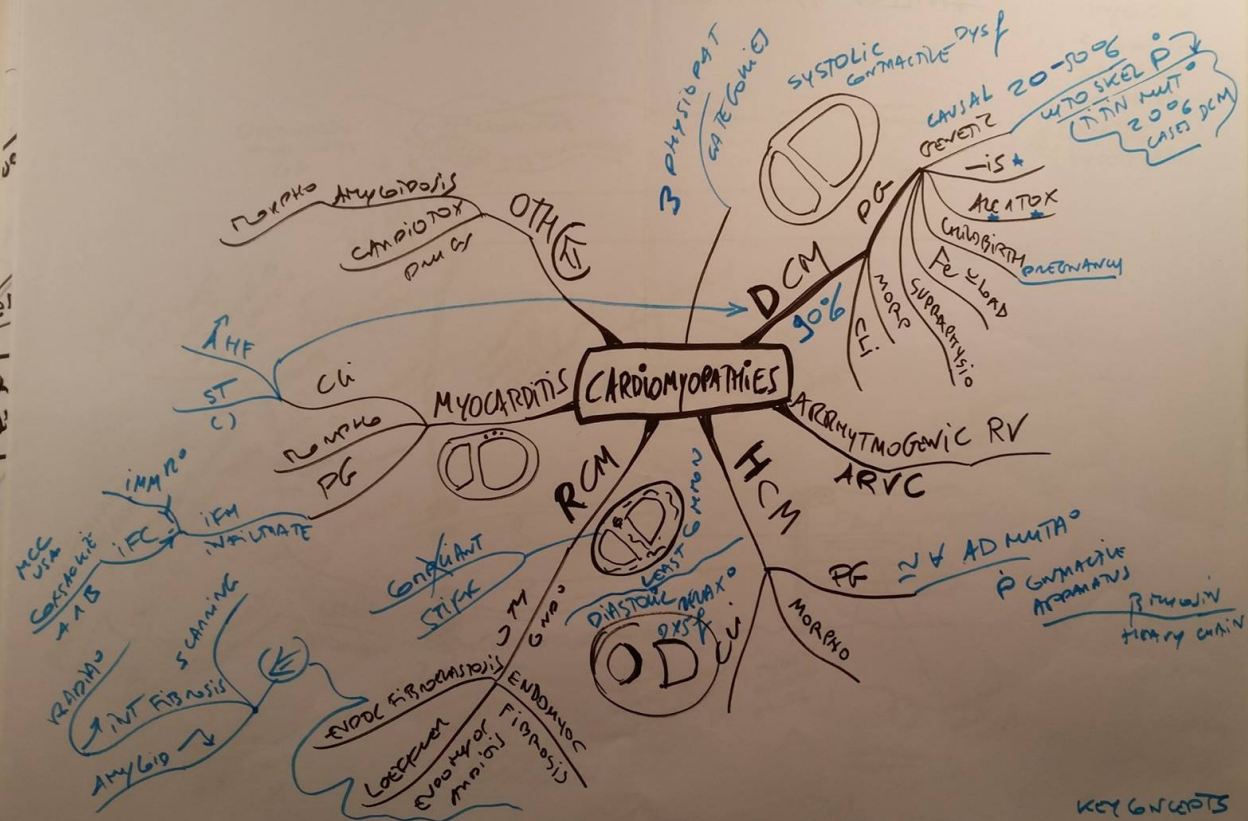
AORTIC
REGURGITATION
(INSUFFICIENCY)

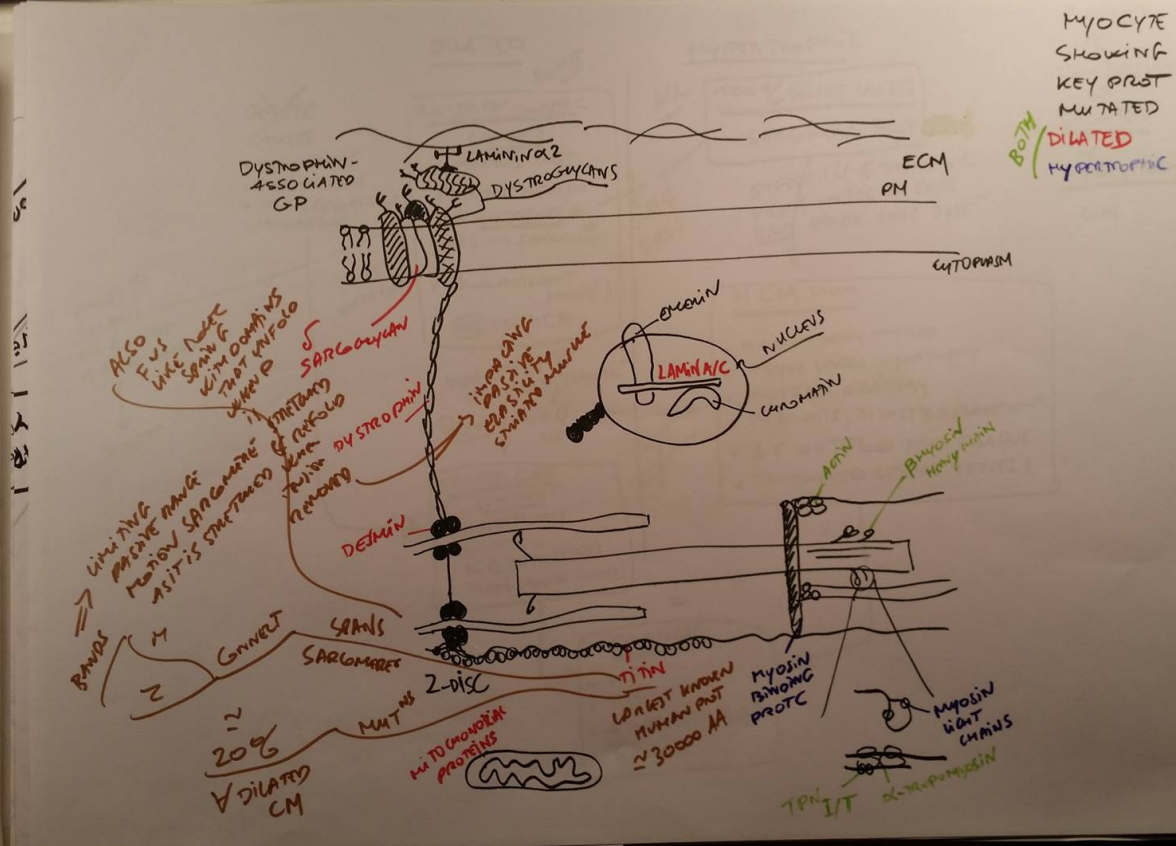


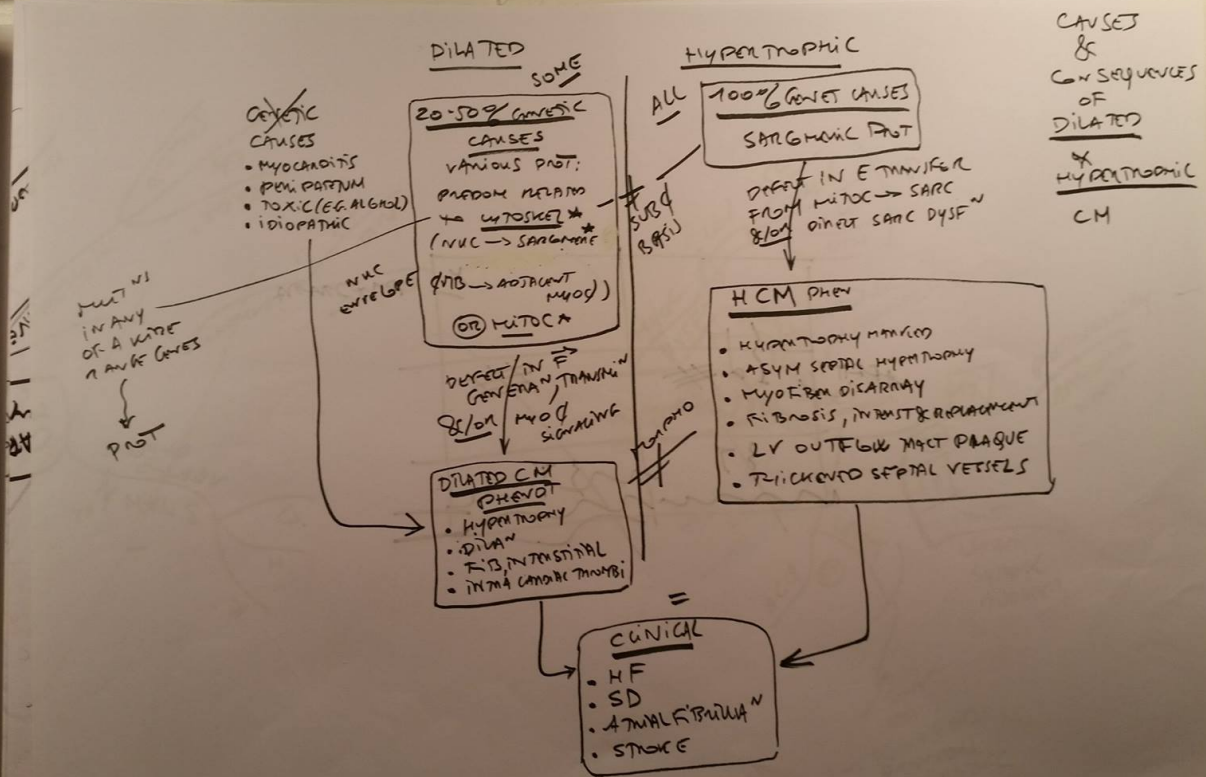
MHD

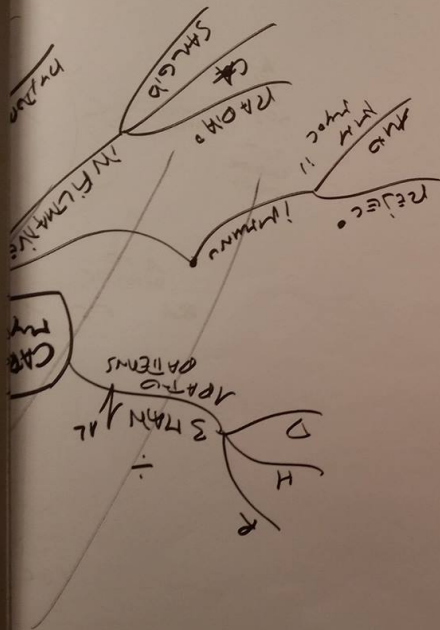


KEY CONCEPTS

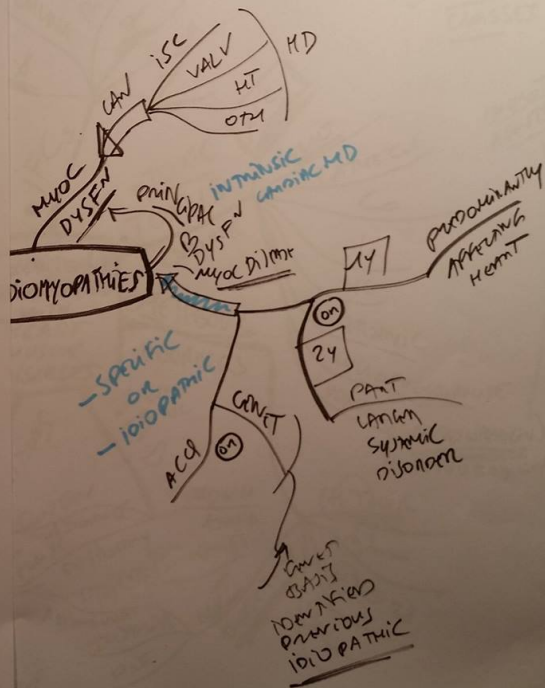


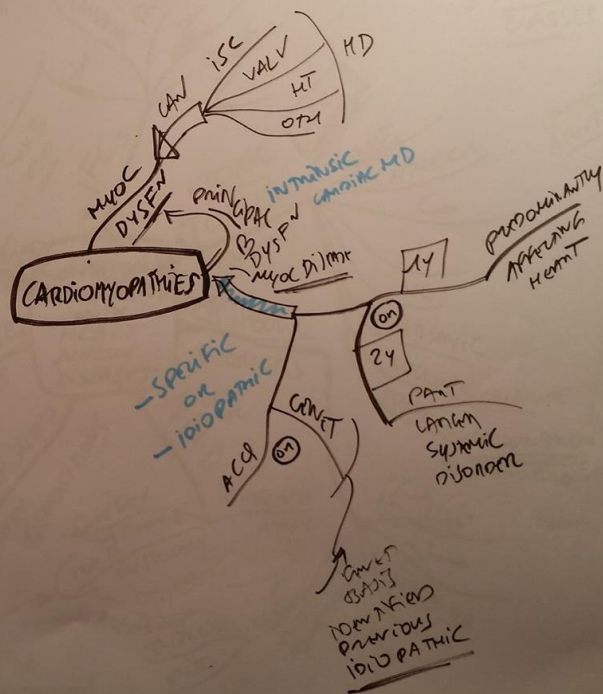


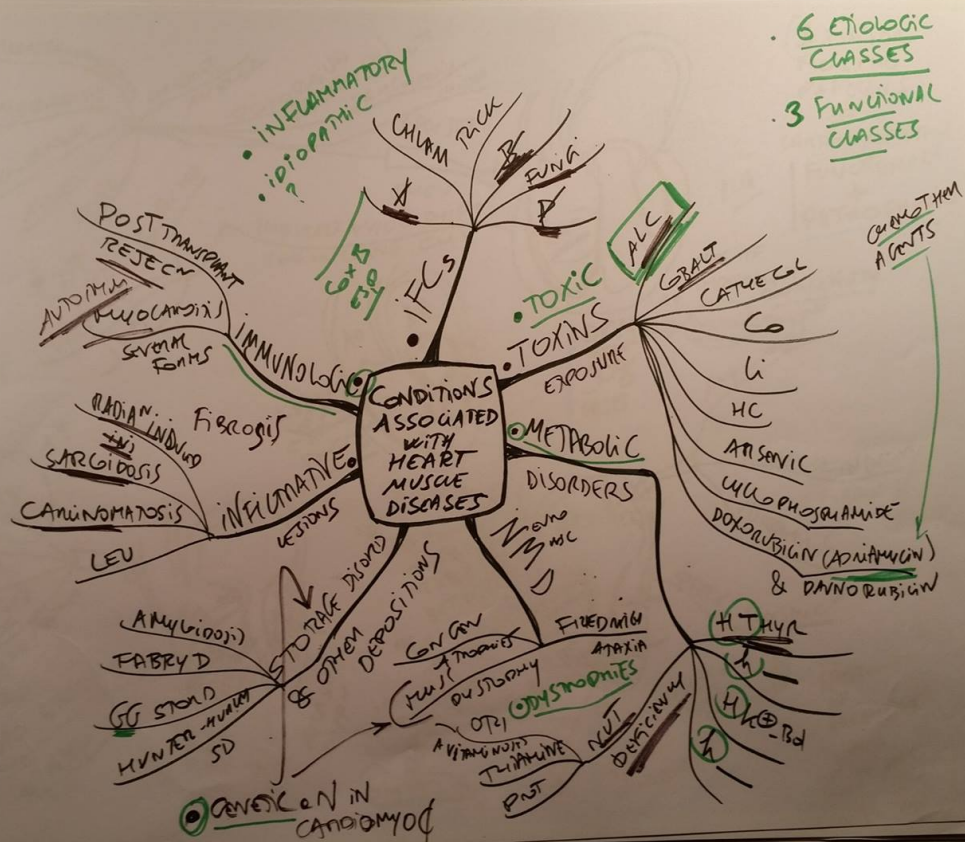




CARDIOMYOPATHIES



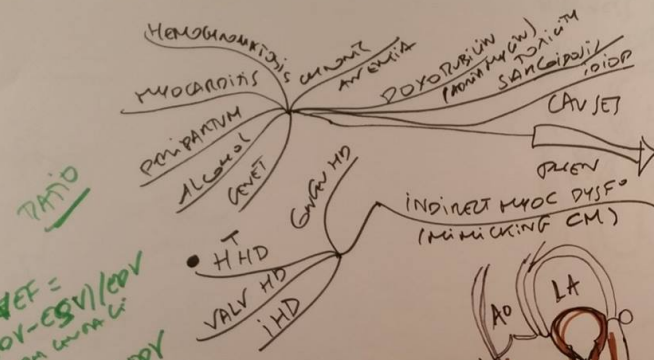




6 ETIOLOGIC CLASSES

3 FUNCTIONAL CLASSES

$EF = \frac{COV - ECV}{COV}$
 on normal Li
 $EF = 1/COV$

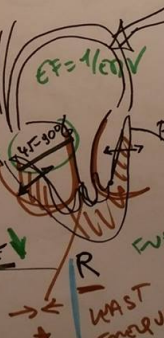


3 MAJOR MORPHO PATTERNS OF CARDIOMYOPATHY
 FUNCTIONAL + PATHOPHYSIO

GENETIC
 FRIEDRICH ATAXIA
 STORAGE DISEASES
 INFANT OF DIABETIC MOTHER



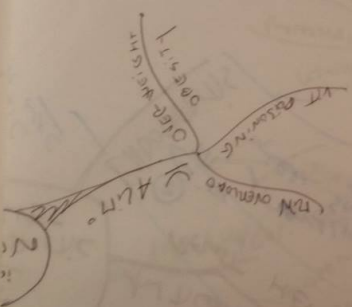
"R" NORMAL
 NOT THICKENED OR DISTENDED
 NERVELY BUT RIGHT
 IN OHS REAR



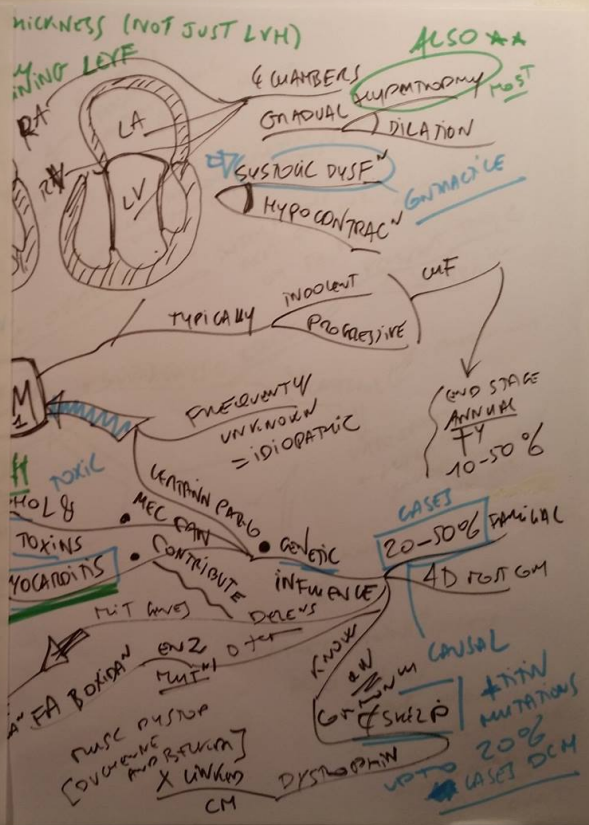
RADIATION-INDUCED FIBROSIS
 IDIOPATHIC
 PERICARDIUM
 SPECTRUM OF SEVERITY AND CLINICAL PAST CAN OVERLAP

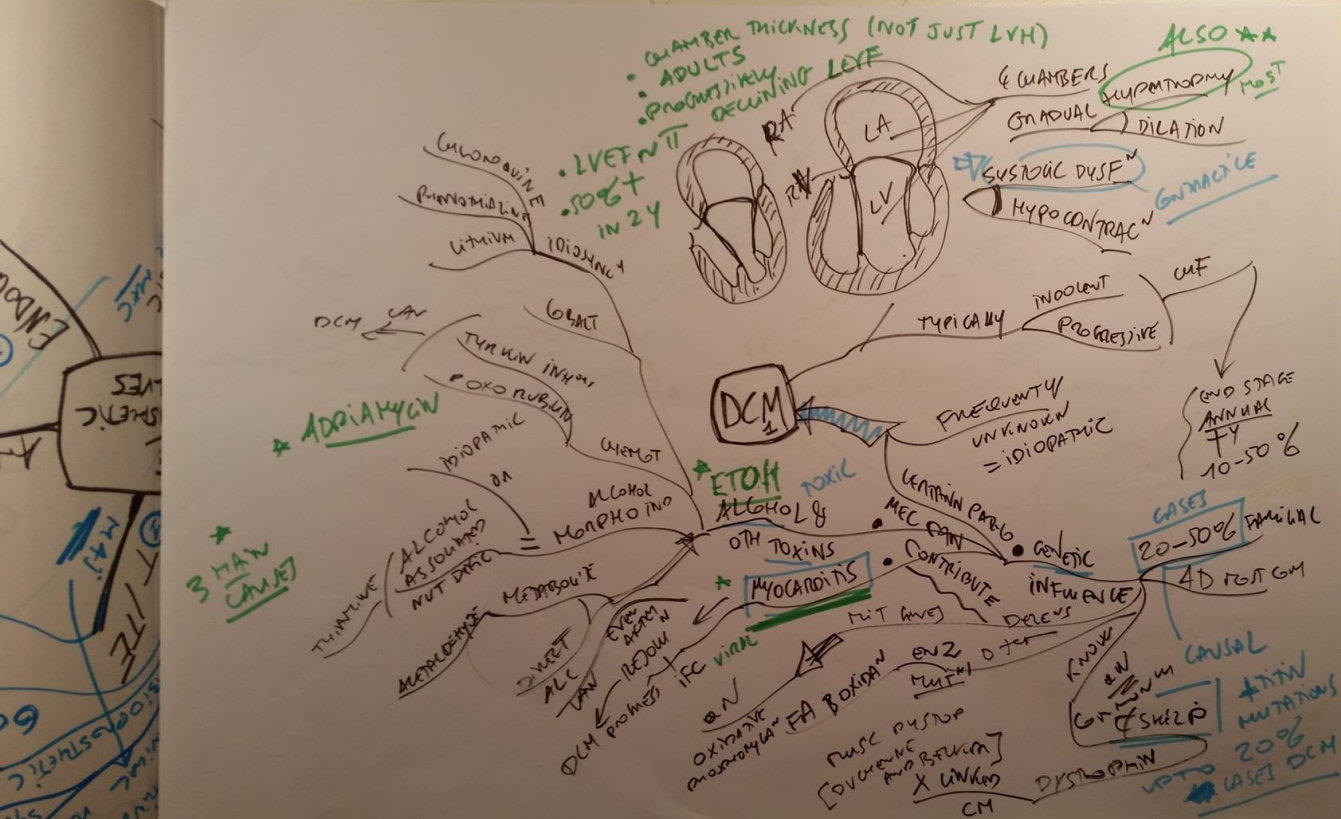
IMPAIRMENT OF COMPLIANCE
 DIASTOLIC DYSFUNCTION

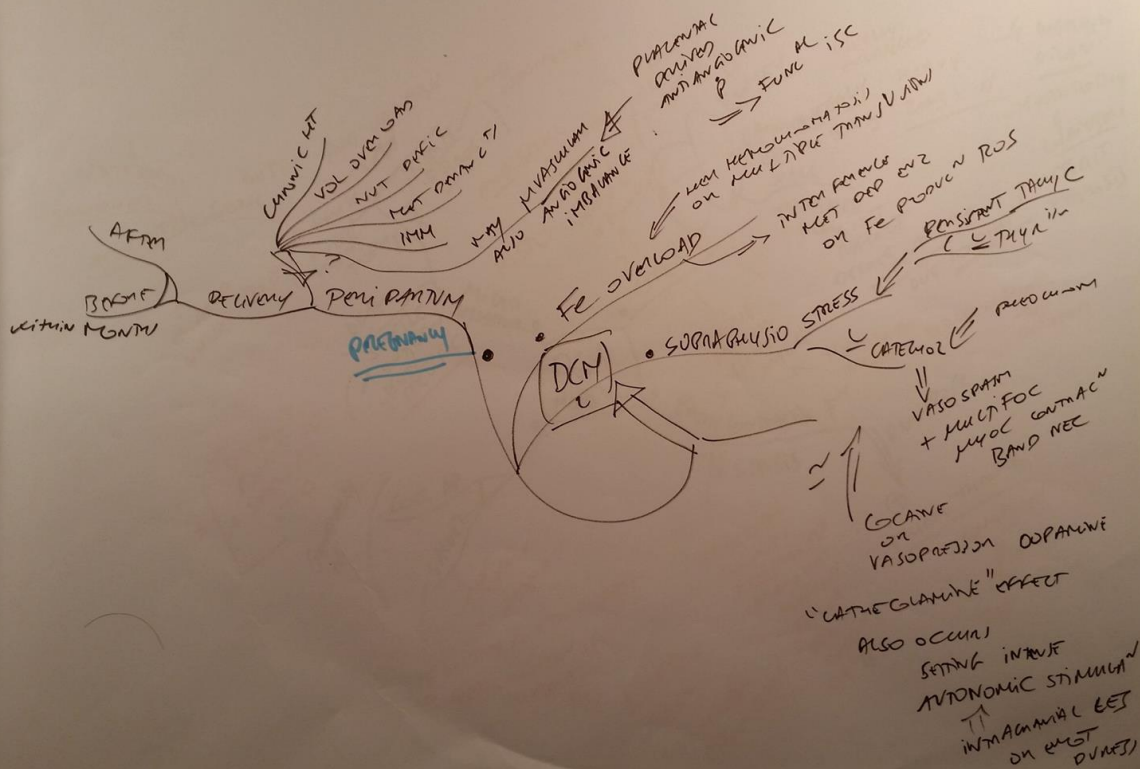
FUNCTIONAL DEFECT
 MOST FREQUENT LES GARN

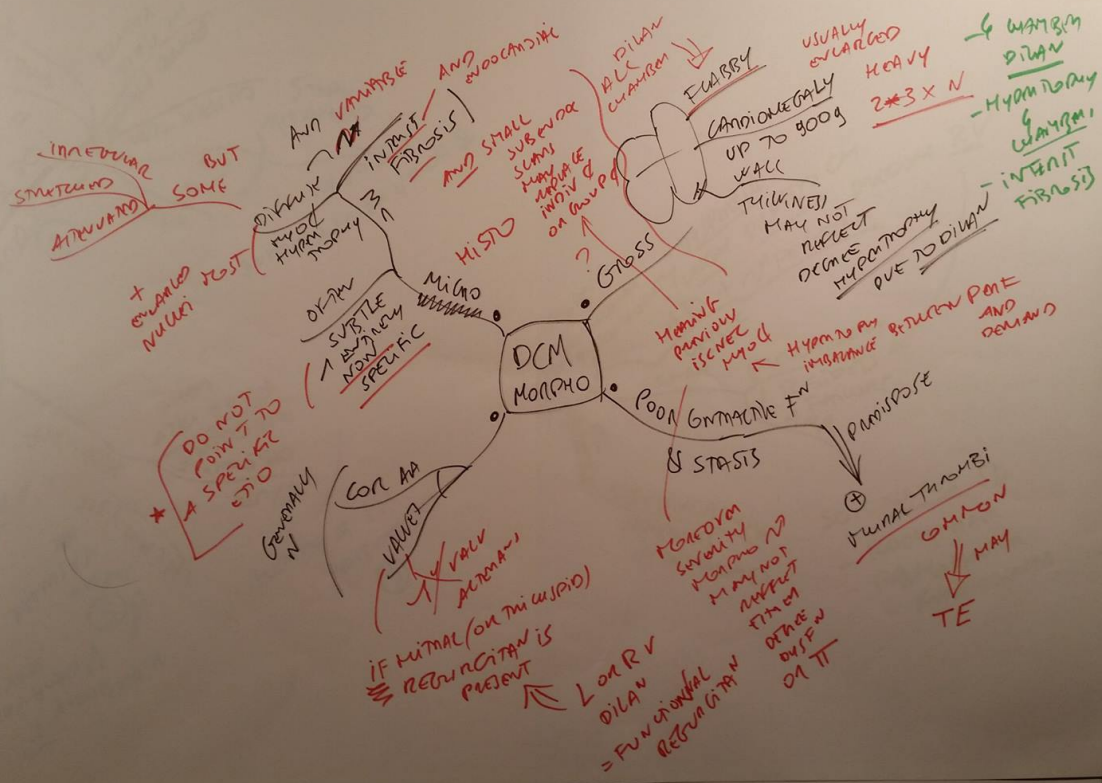


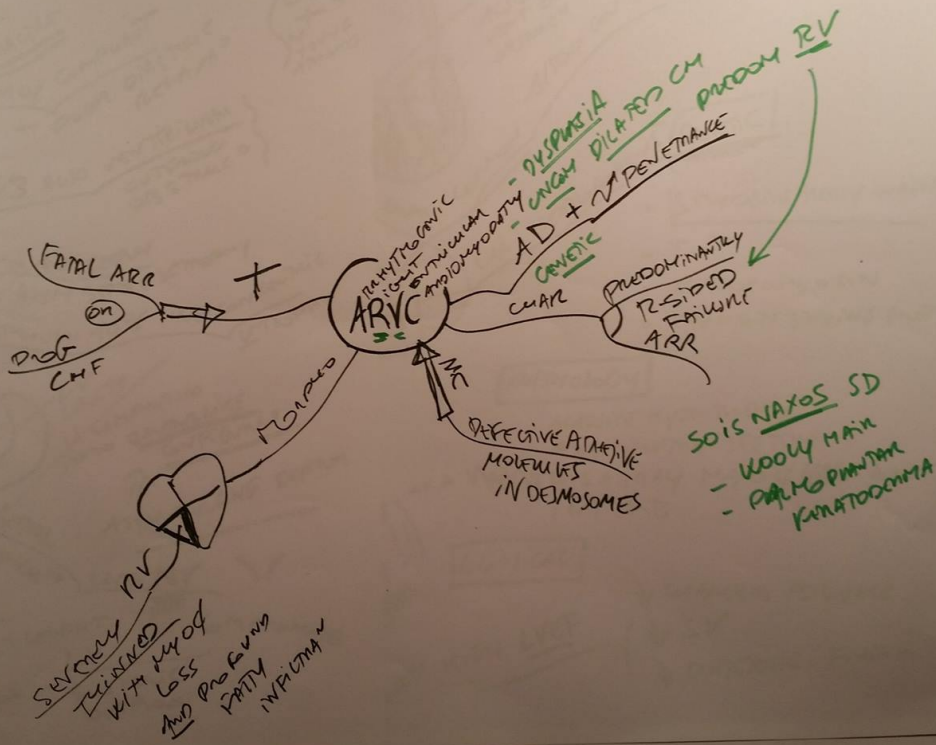
DCM

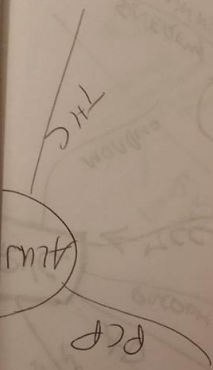




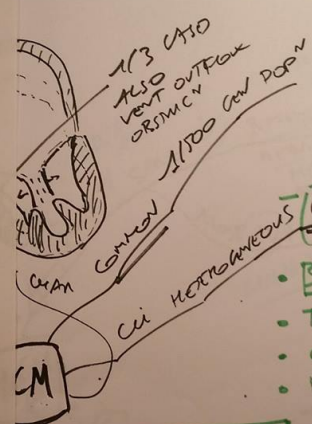








HCM



Genetic

- MYOSIN HEAVY CHAIN
- TnT
- α TROPOMYOSIN
- MYOSIN BINDING PROTC

PATHOLOGY

MASSIVE MYOCARDIOMY
ASYMPTOMATIC SEPTUM

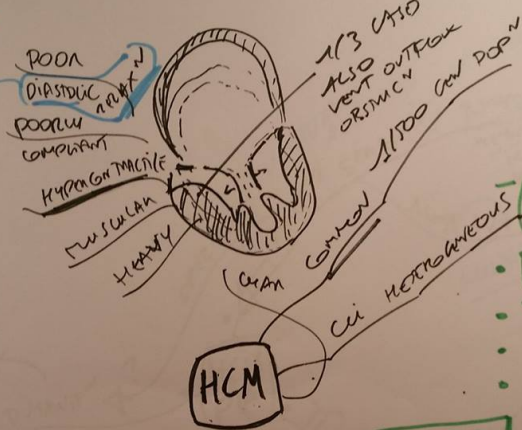
*** Wavy DISARRAY MYO & INT FIB ***

CLINICAL

BUT HIGH LVEF

- ↓ MYOCARDIAL VOLUME
- ↓ SV
- ↓ DIASTOLIC FLOW

* DUSFN



ALSO CALLED

DIABETIC
HSS
HYPERTROPHIC
CARDIOMYOPATHY
US AORTIC
TENDON

WONT
PART
OLD
NAME

GENETIC

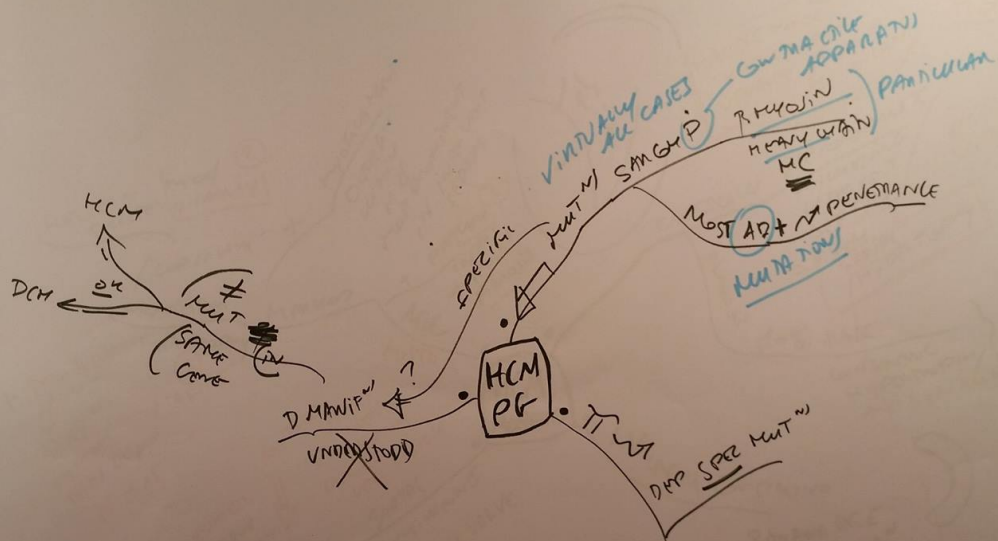
- MYOSIN HEAVY CHAIN
- TnT
- α TROPOMYOSIN
- MYOSIN BINDING PROTC

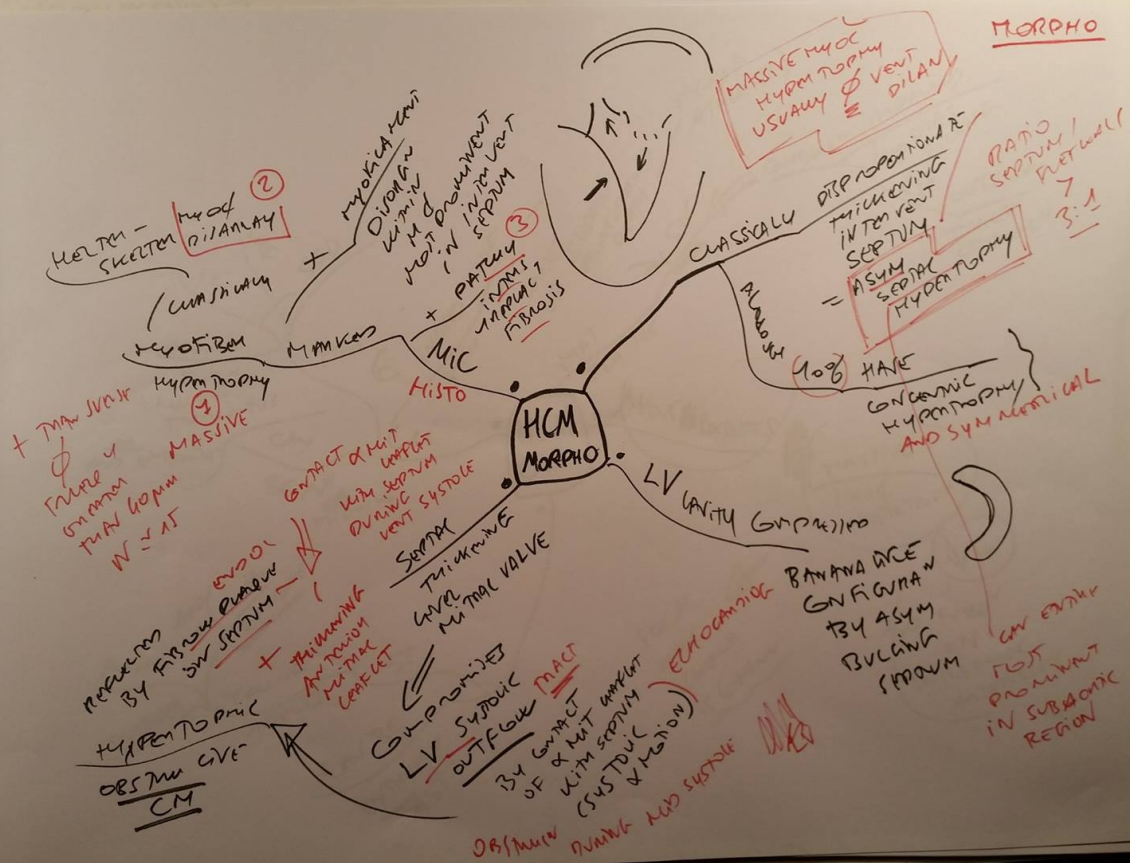
PATHOLOGY

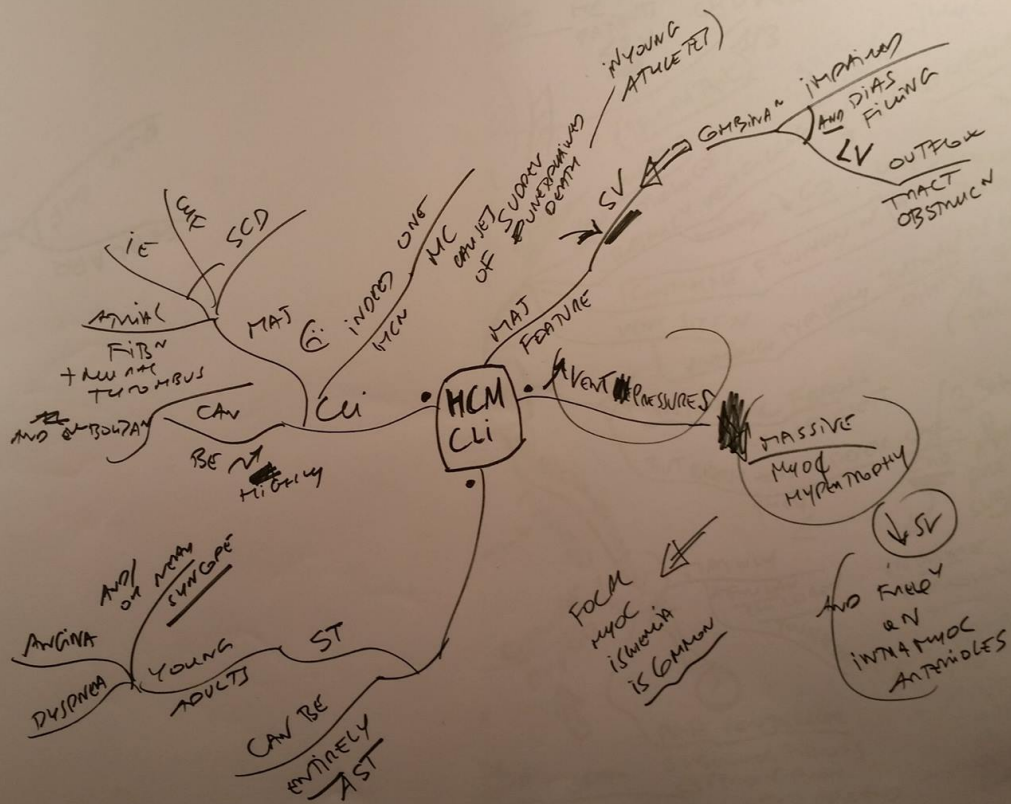
MASSIVE MYOCARDIUM
ASYMMETRIC SEPTUM
*** DISAPPEAR MYO ***
INT FIB

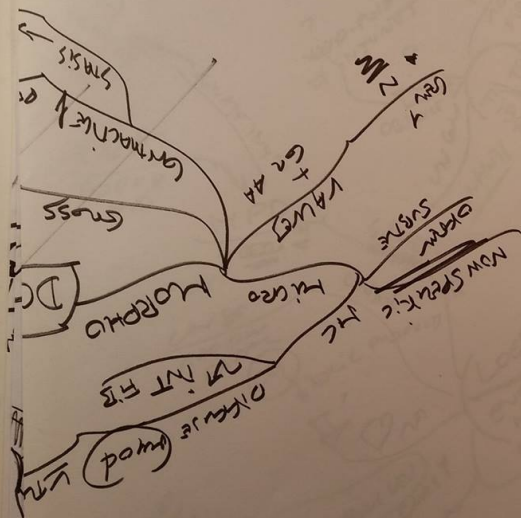
CLINICAL

↓ DISAPPEAR (very thick LV BUT NO HT !!)
BUT HIGH LVEF
↓ CHAMBER VOLUME
↓ SV
↓ DIASTOLIC FILLING

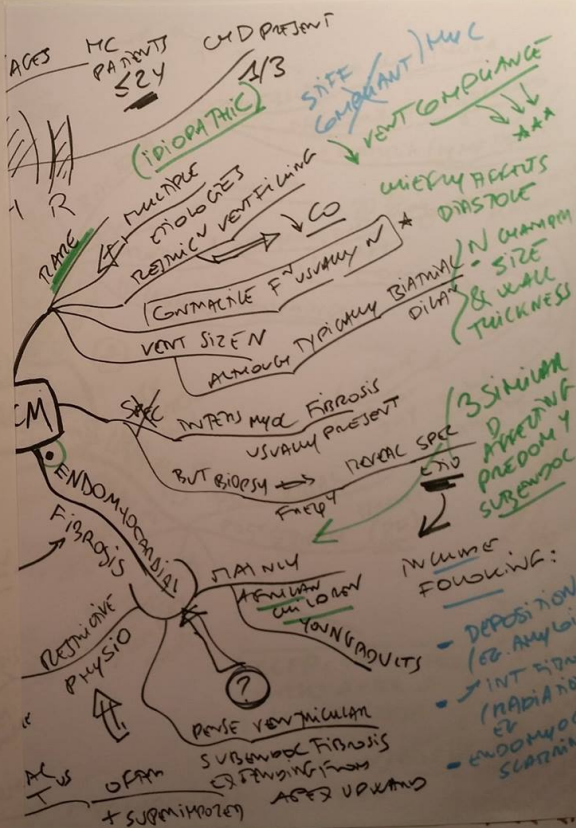




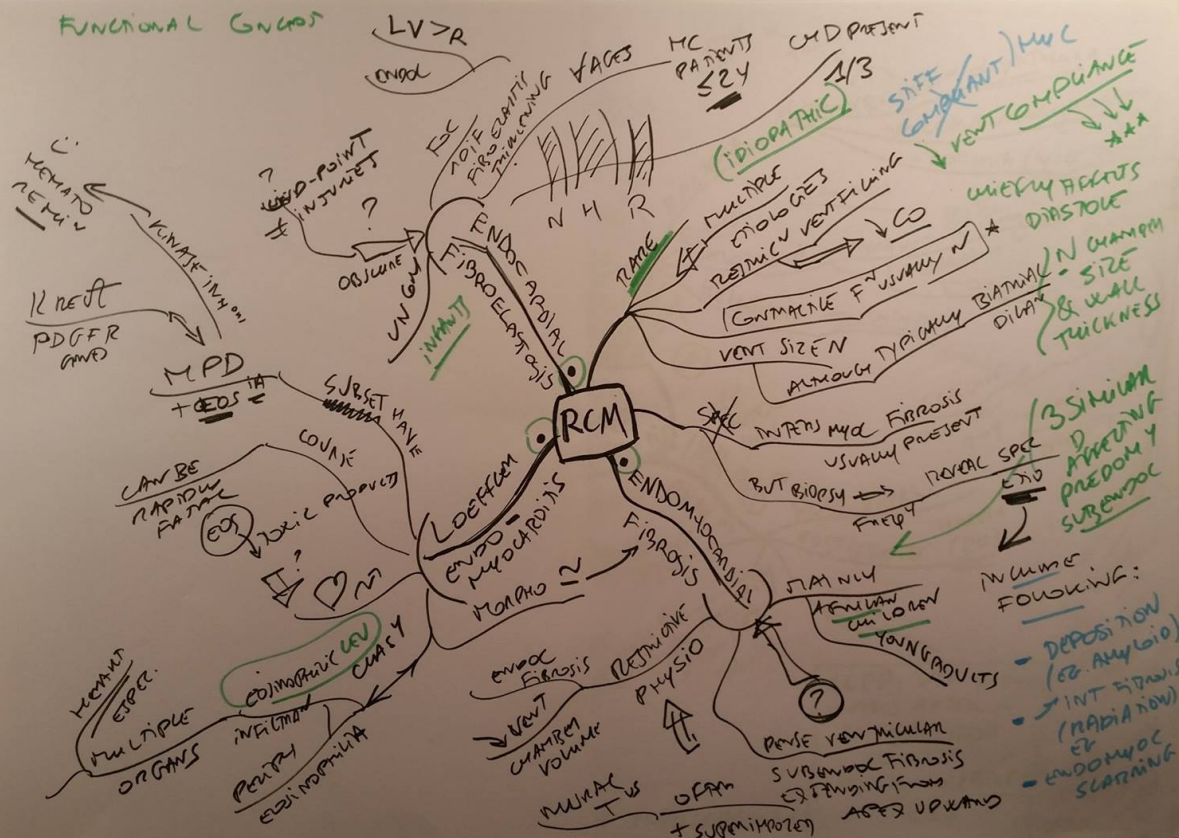




RCM



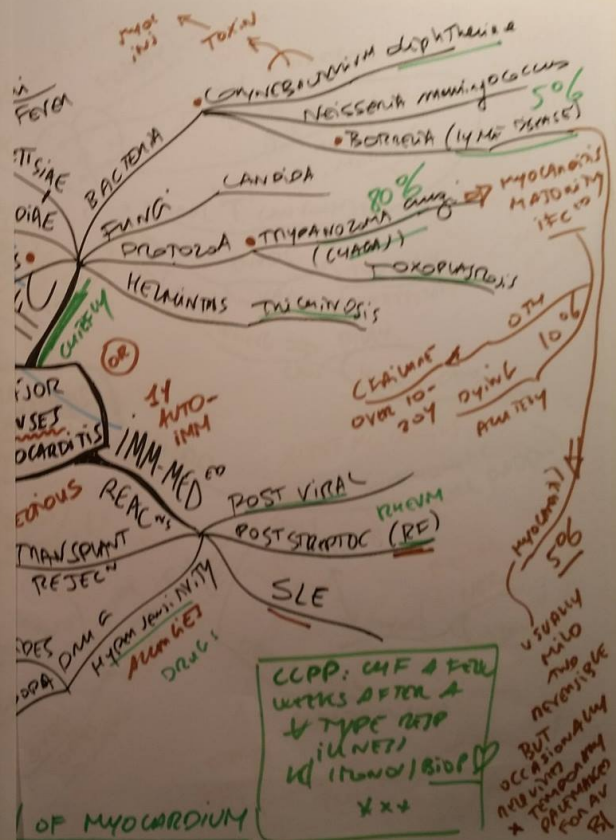
Psychos

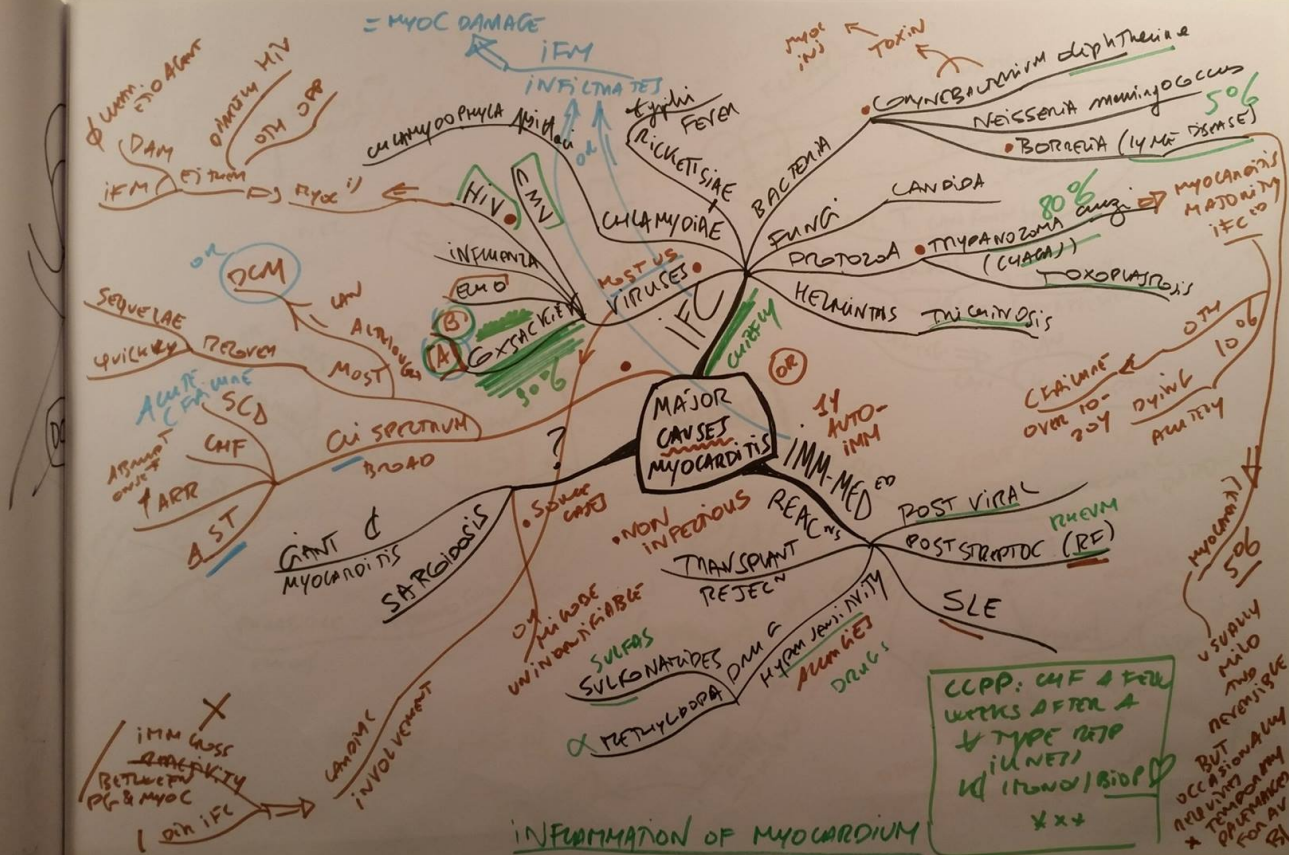


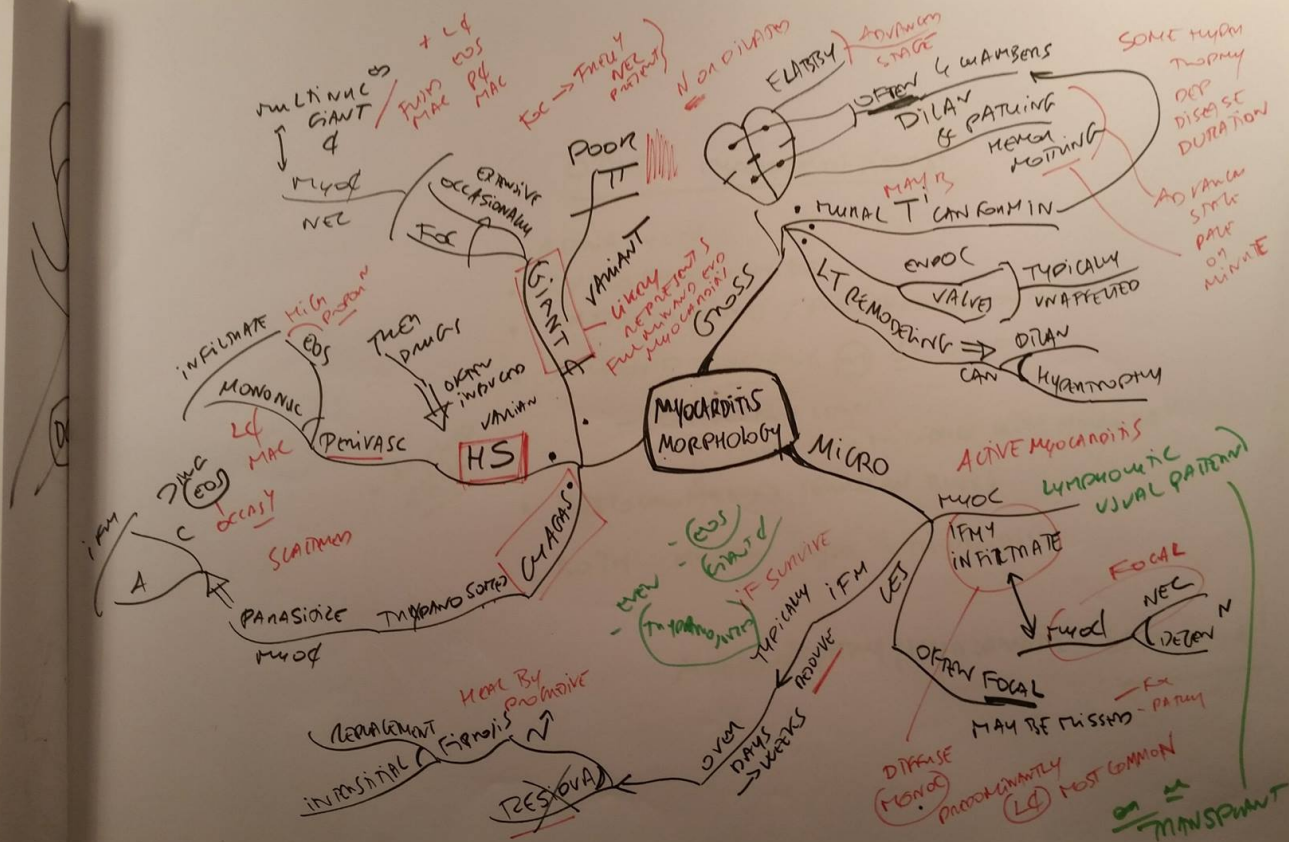
DD



MYOCARDITIS







OTHER MYOCARDIOTIDES

- ADENYLYLATION
- CYCLOPHOSPHAMIDE
- CATECHOLAMINES (PHEDROBOLAMIDES)
- AMYLOID SYSTEMIC $\oplus$ 1 LY CARDIAC

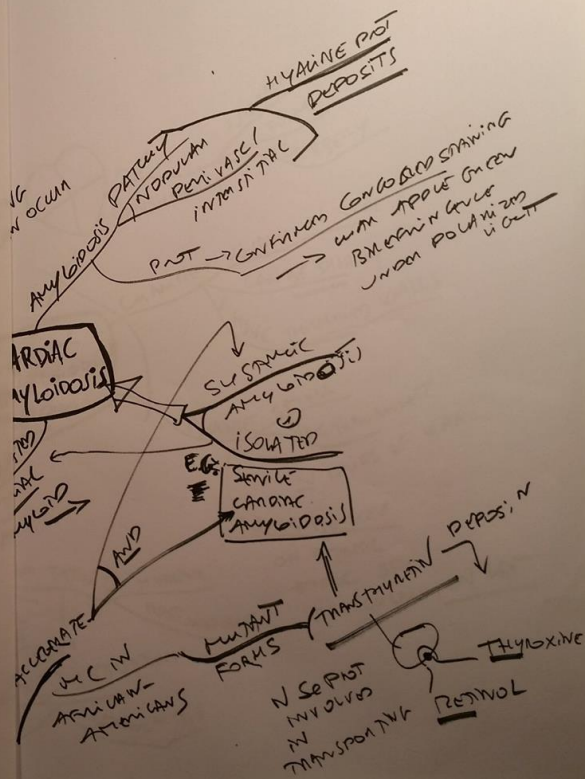
- ON NO RED STAIN:
GREEN BLUE FLUORESCENCE WITH POLARITAN

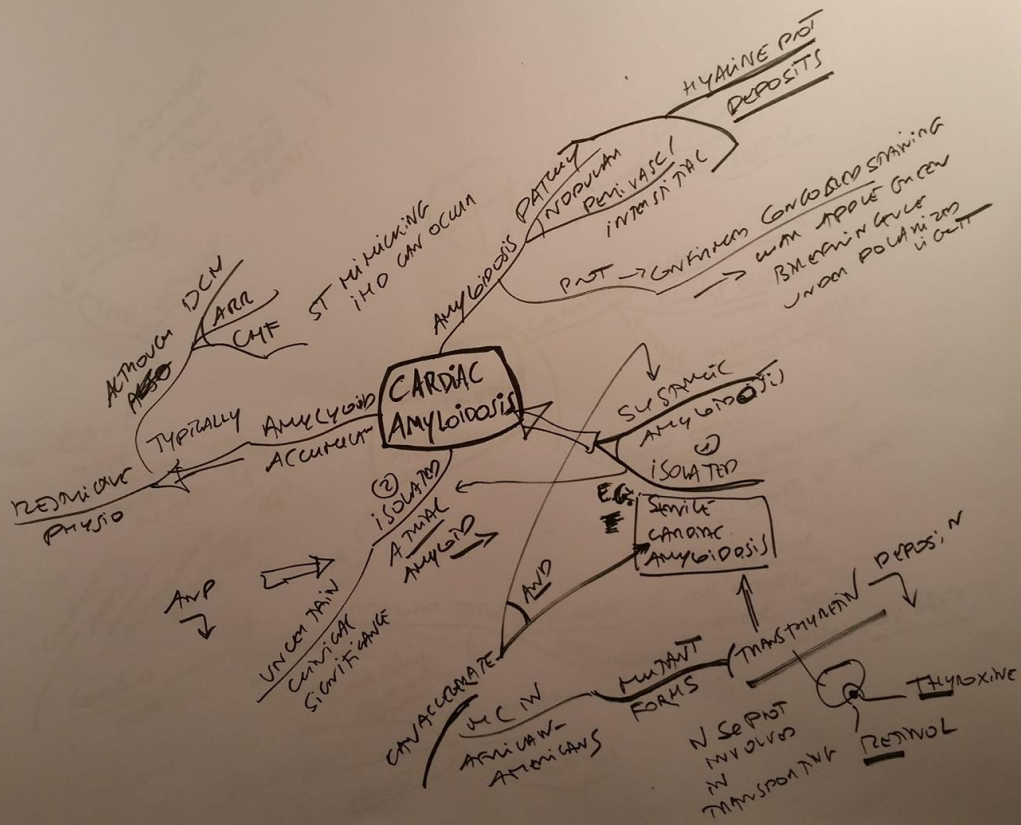
• AMYLOID
AGNA

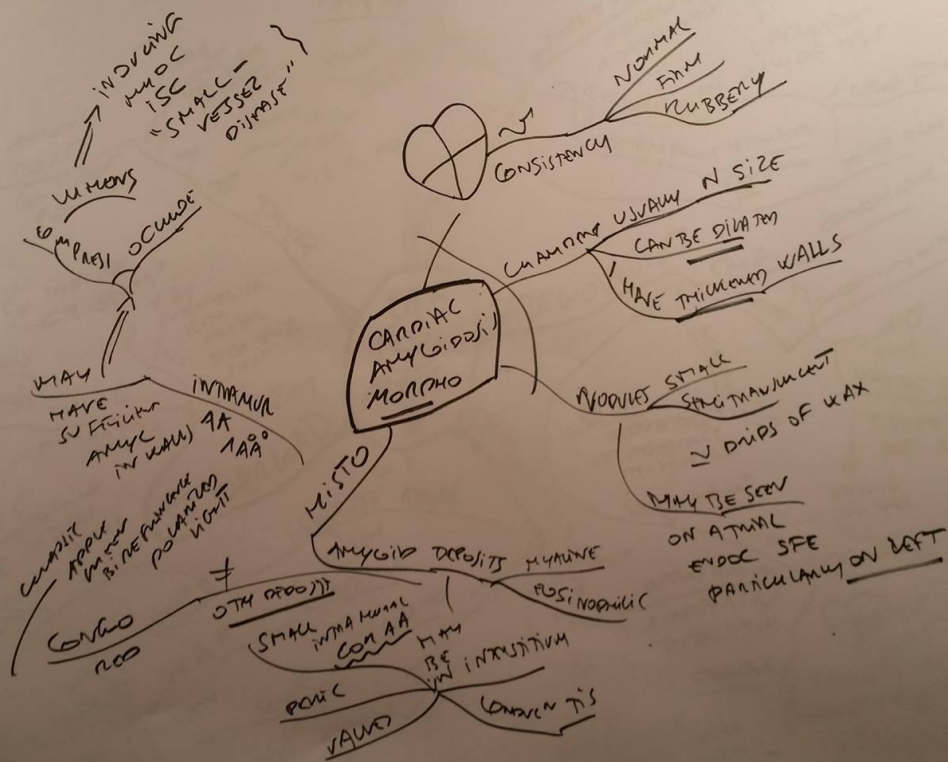
- HEMOGLOBIN (PRUSSIAN BLUE)
- BOTH HYPER - HYPO - THYROIDISM

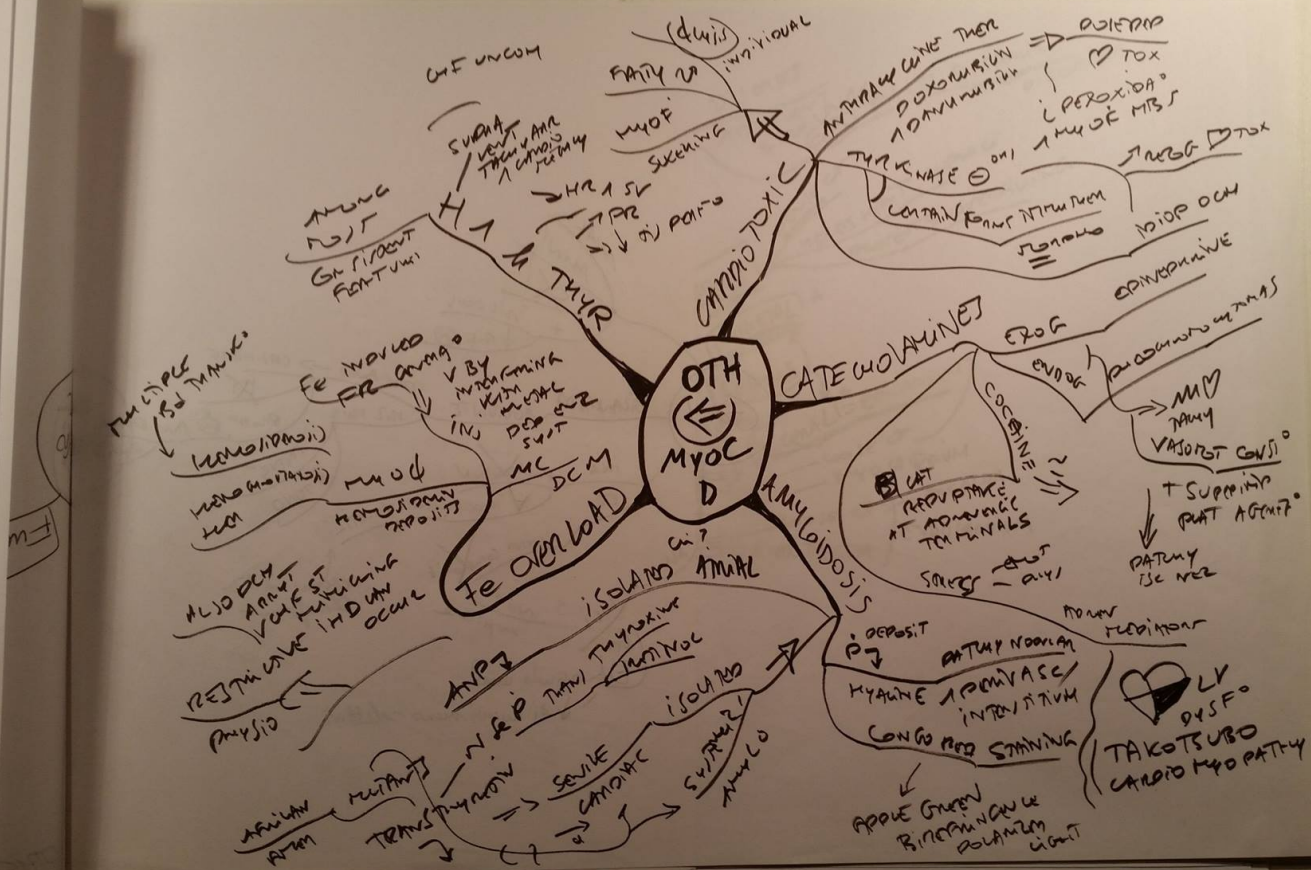
MYOCARDIOPATHY / MYOCARDIAL SPECTRUM!

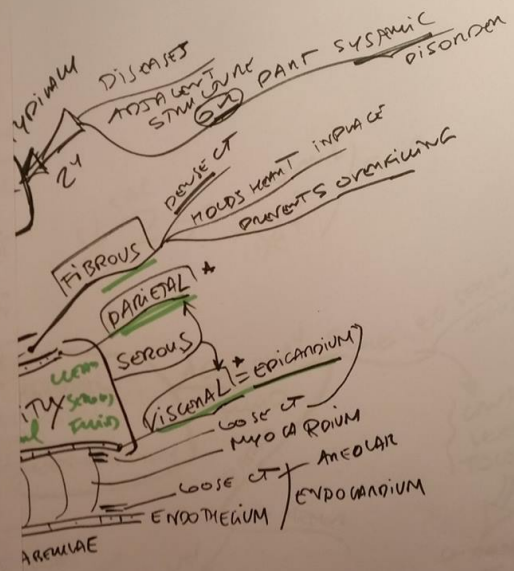
OTHER CAUSES OF MYOC DISEASE



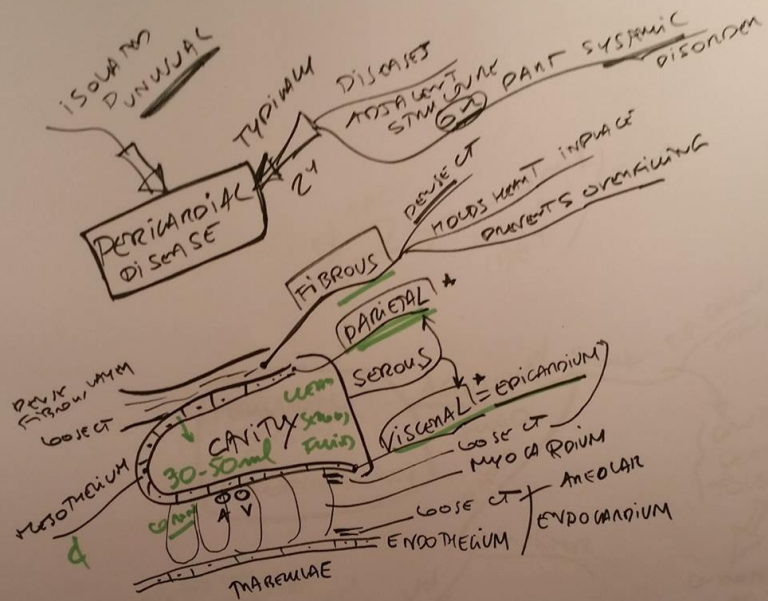








PERIC



NSAC
30-50 ml
serous
effusion

PERICARDIAL
EFFUSION
&
HEMOPERICARDIUM

ACCUMULATION

slow

E.G. SEROUS
EFFUSION

fluid

rapid

E.G. HEMORR

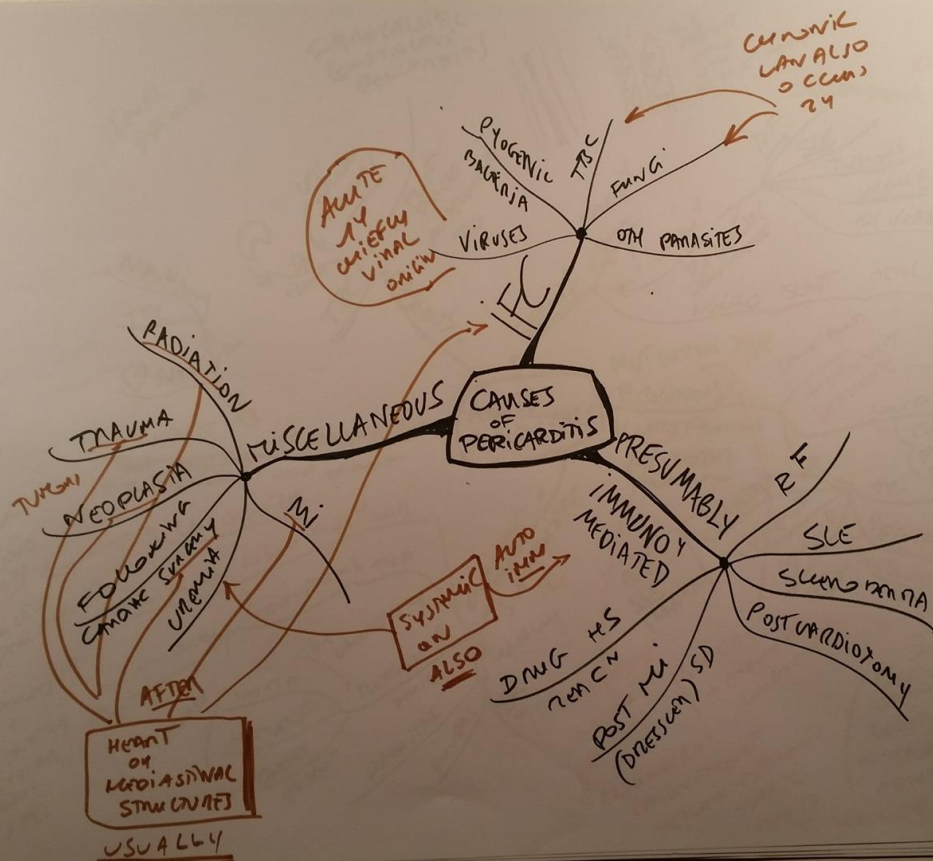
CAN BE
WELL
TOLERATED

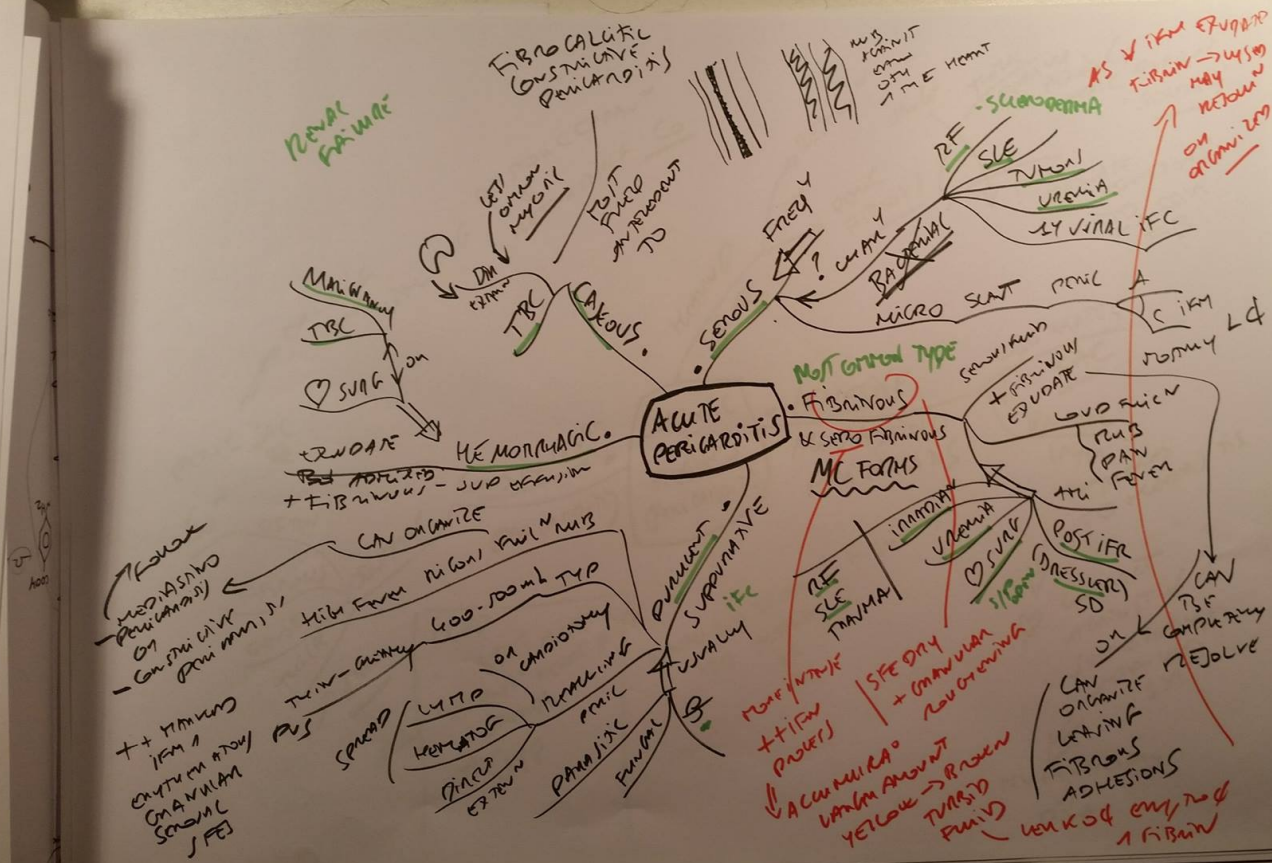
CHRONIC
COURSE

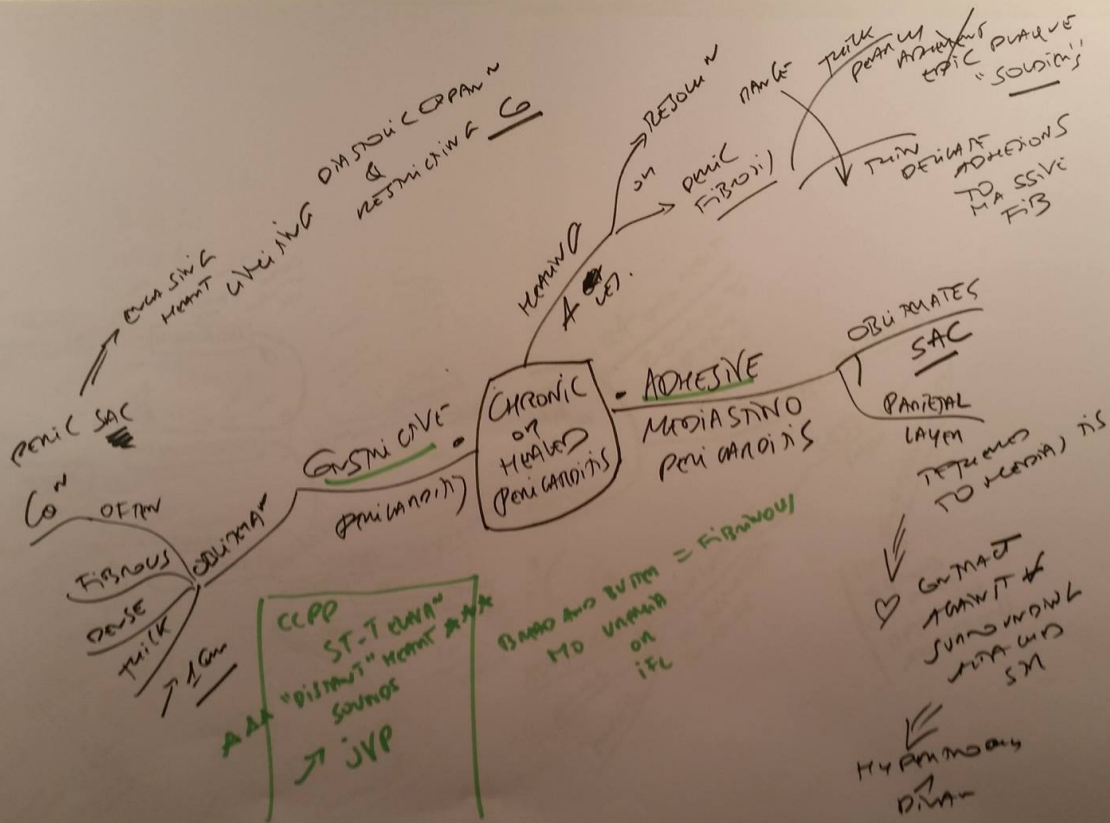
> 500 mL

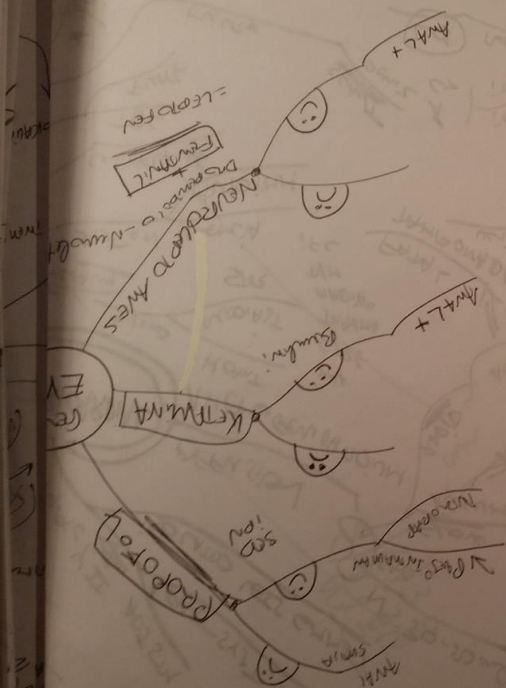
- RUPTURED MI
 - TRAUMATIC PERFORATION
 - RUPTURED AORTA DISSECTION
- Why?

FATAL
IN RESPONSE
WITH AS LITTLE
AS 200 mL

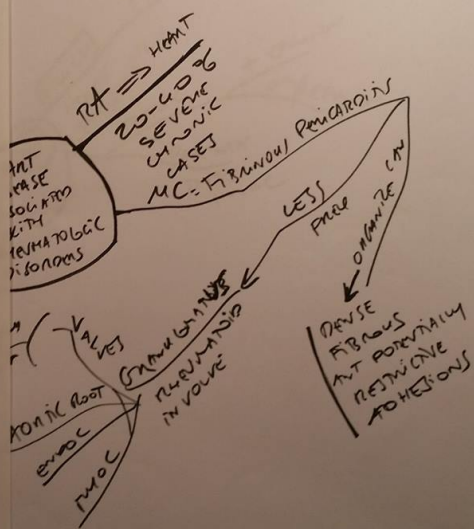


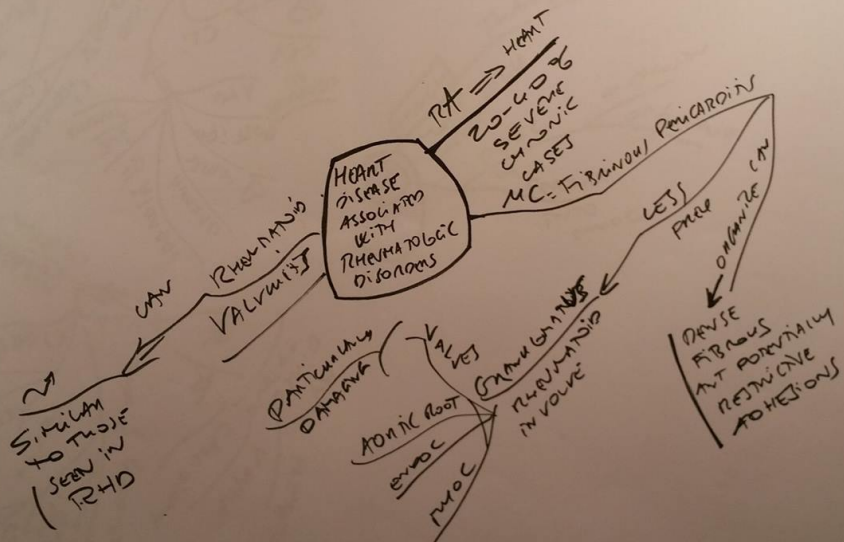


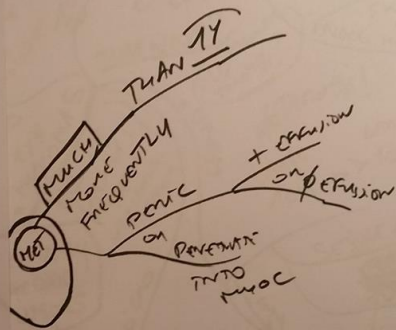




HEART DISORDERS ASSOCIATED WITH RHEUMATOLOGIC DISORDERS





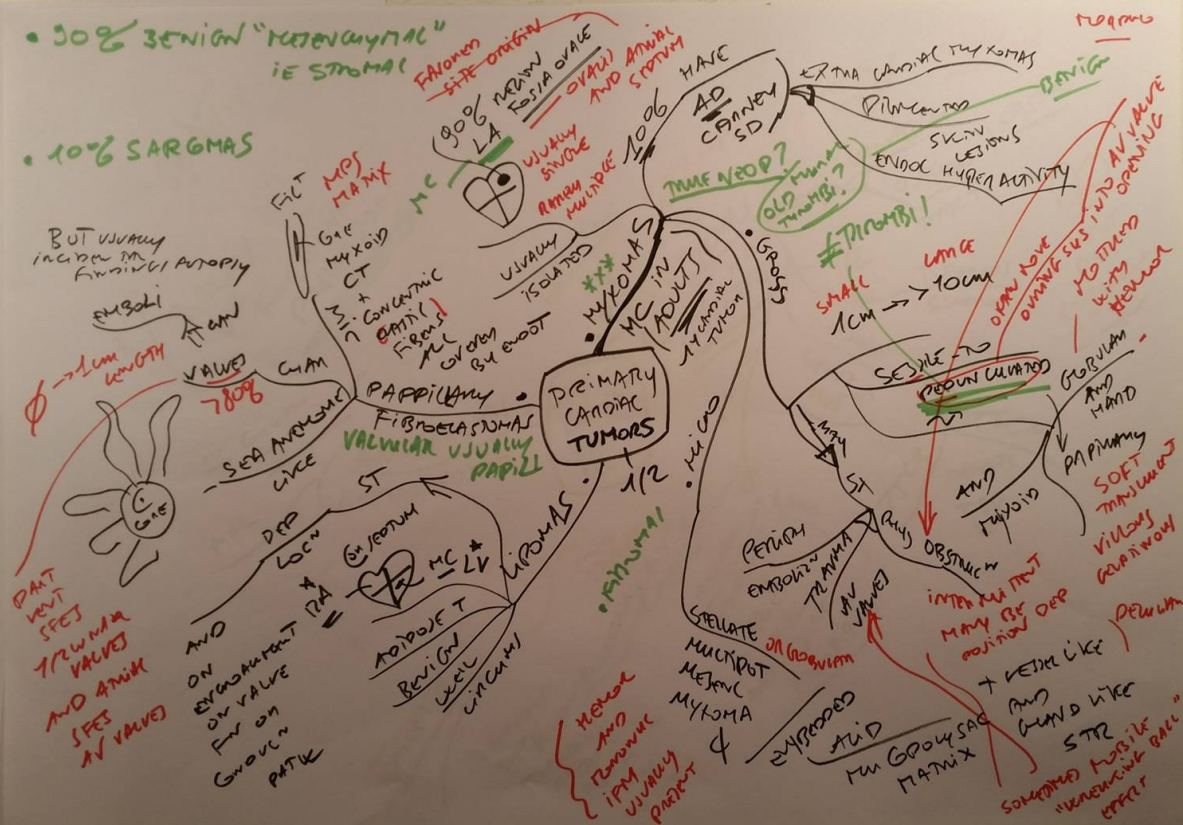


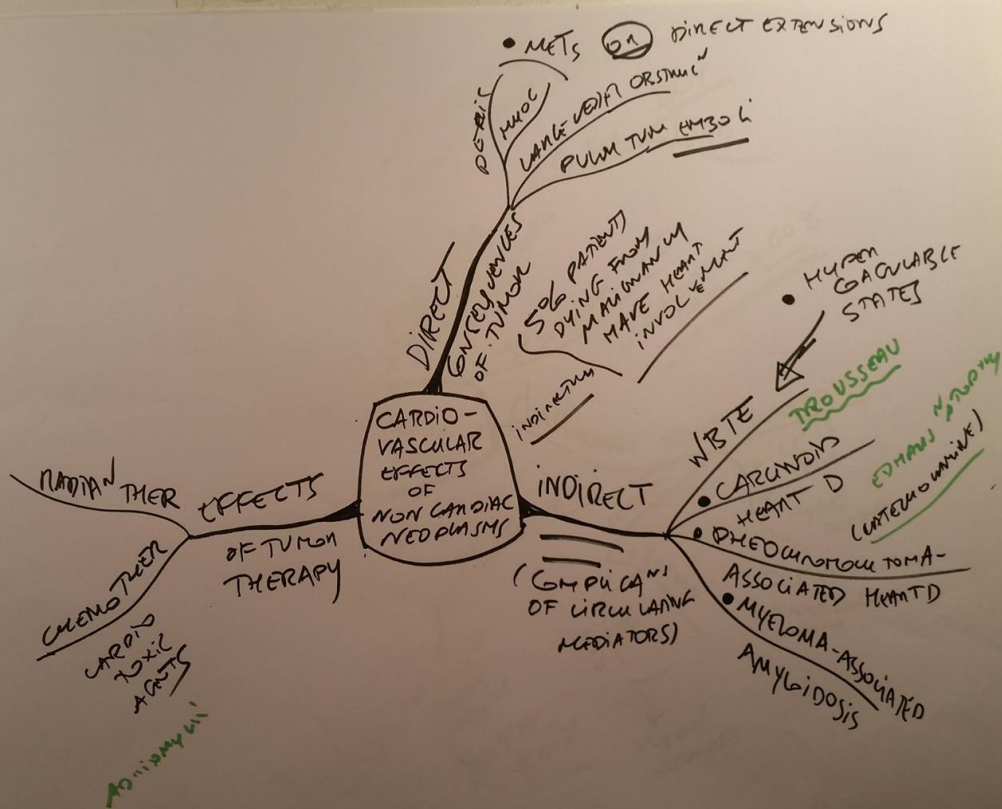
TUMORS

2



- 10% SARGMAS



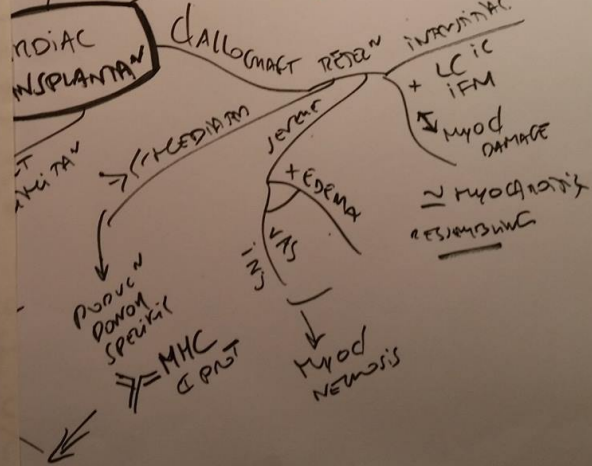


ANTIPROLIFERATIVE
DRUGS → SAIDM 4

SAIDM 4 + RE-ADJUSTING SANGHAI
= 70%
GYNAM 0%

most patients
immunosuppressive

~2000 cases
MCM wire
14 SURVIVAL 90%
+ 54 SURVIVAL 60%



CARDIAC TRANSPLANTATION

Cardio

