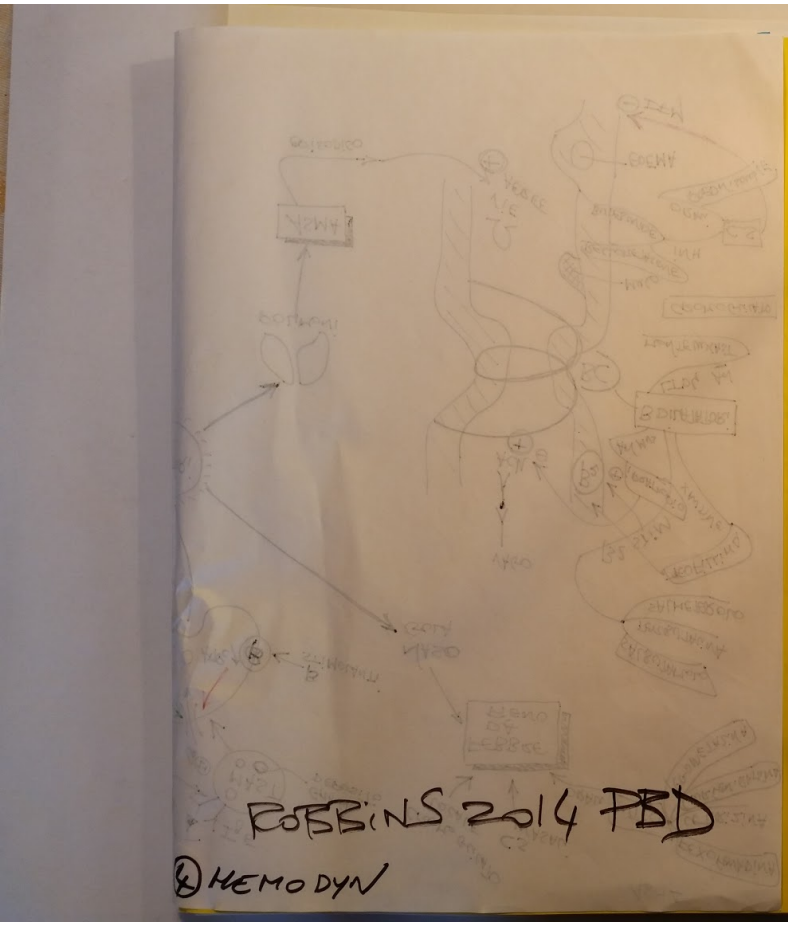
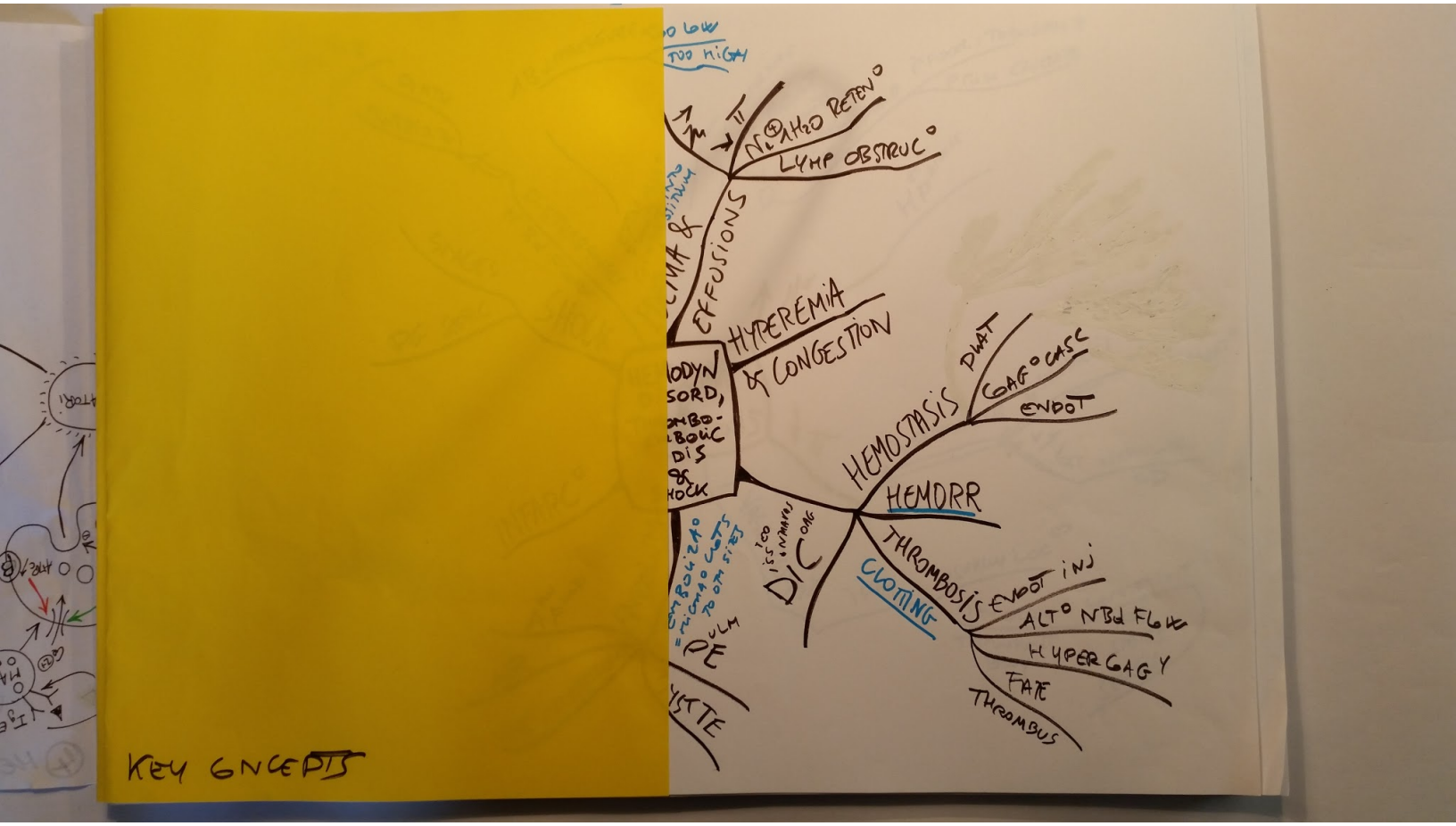
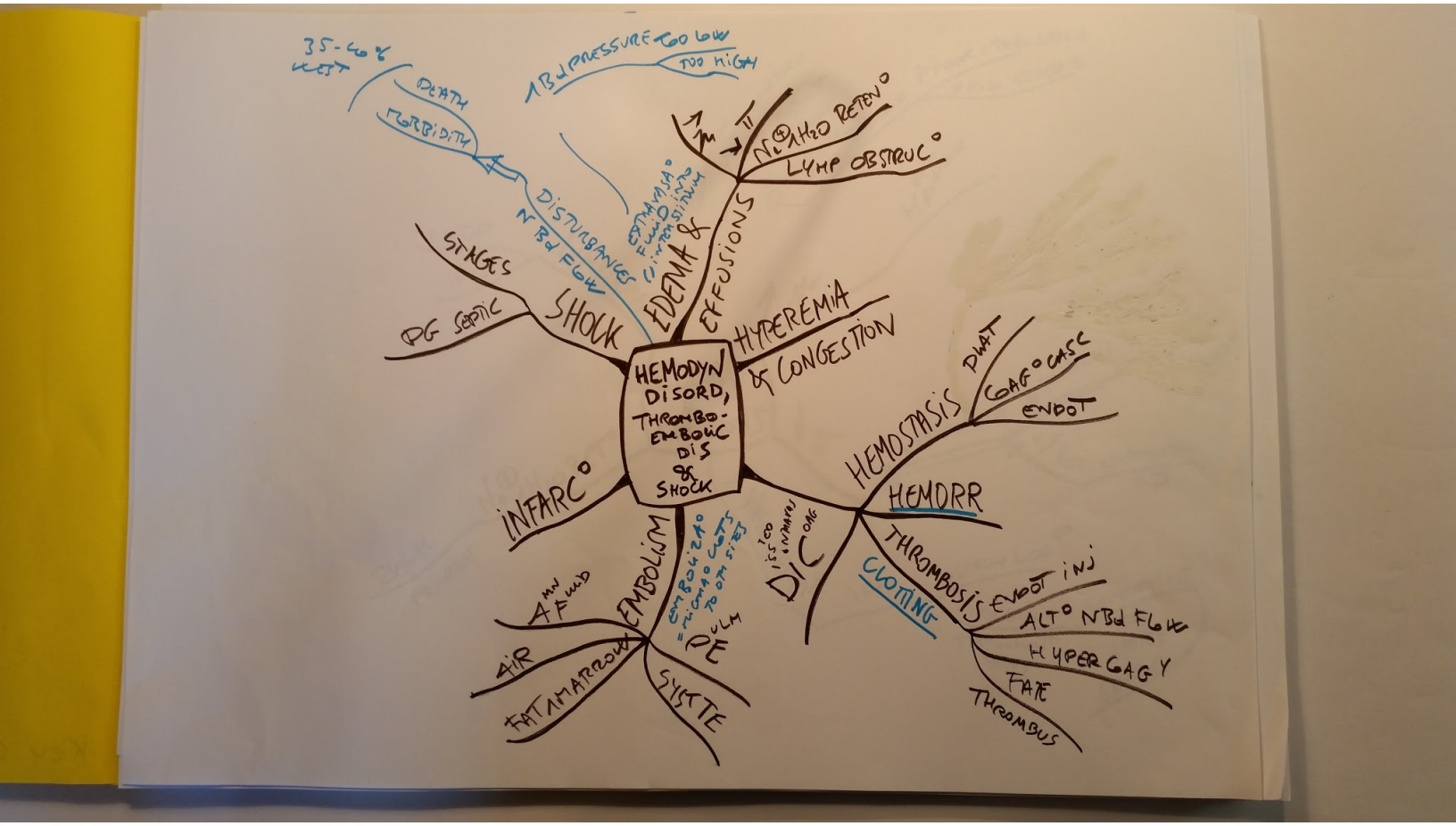
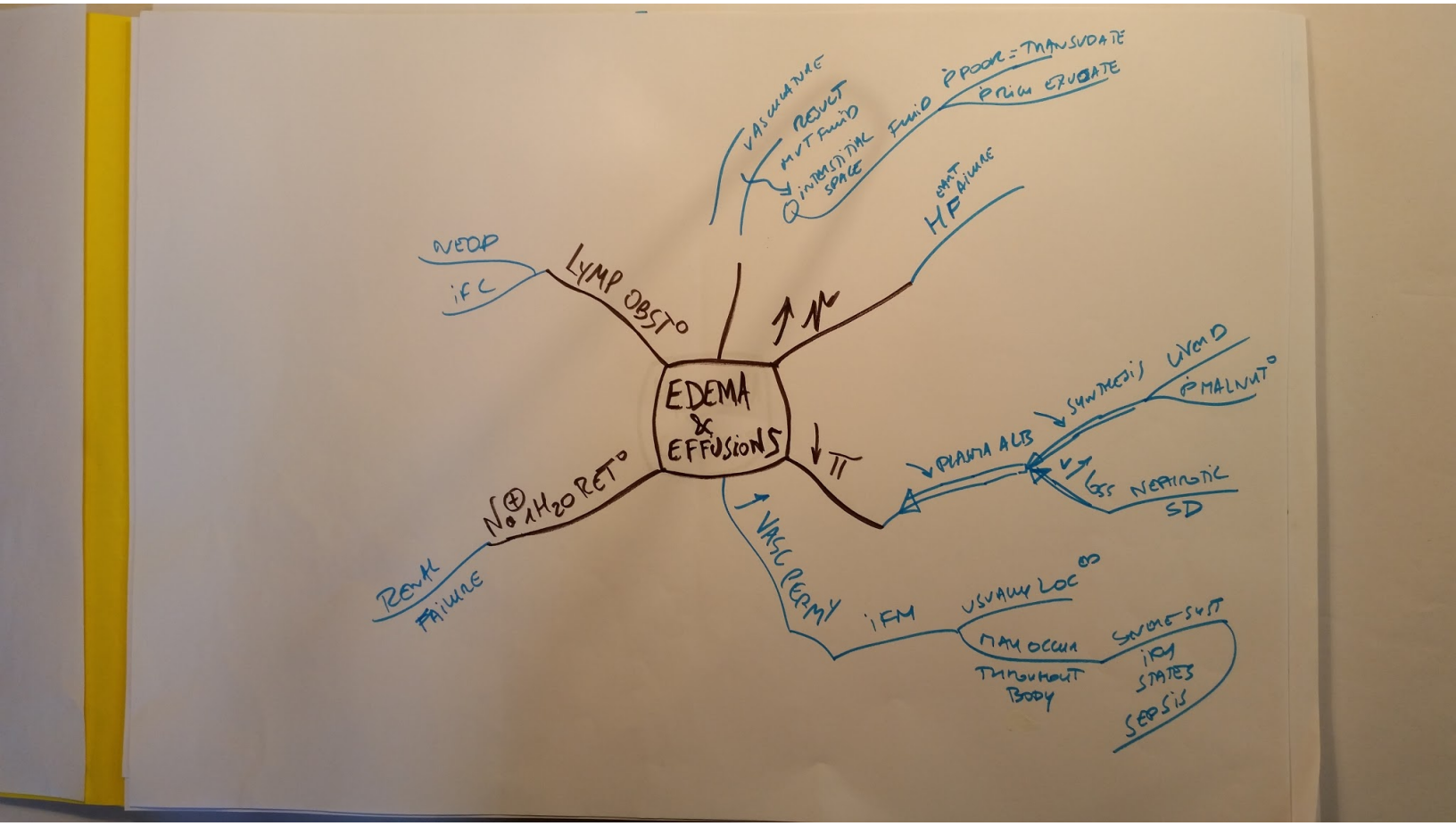


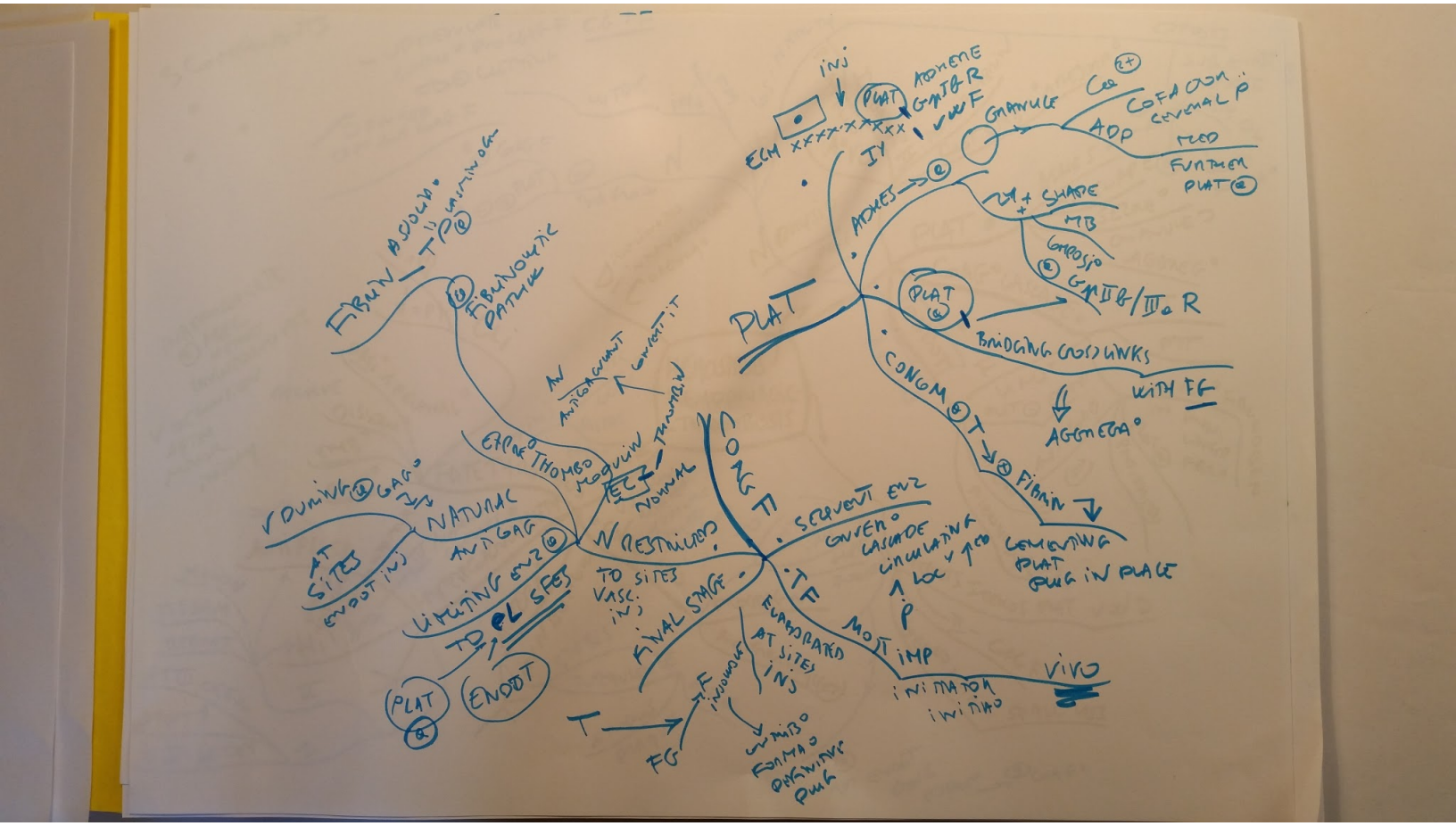
TOO LOW
TOO HIGH
↑M → ↑
N_a H_2O RETEN°
LYMP OBSTRUC°
EFFUSIONS
HYPEREMIA & CONGESTION
HEMOSTASIS
PLAT
GAG° CASC
ENDOT
HEMORR
THROMBOSIS
ENDOT INJ
ALT° NRml FLOW
HYPERGAGY
FATE
THROMBUS
CLOTTING
DIC
PE

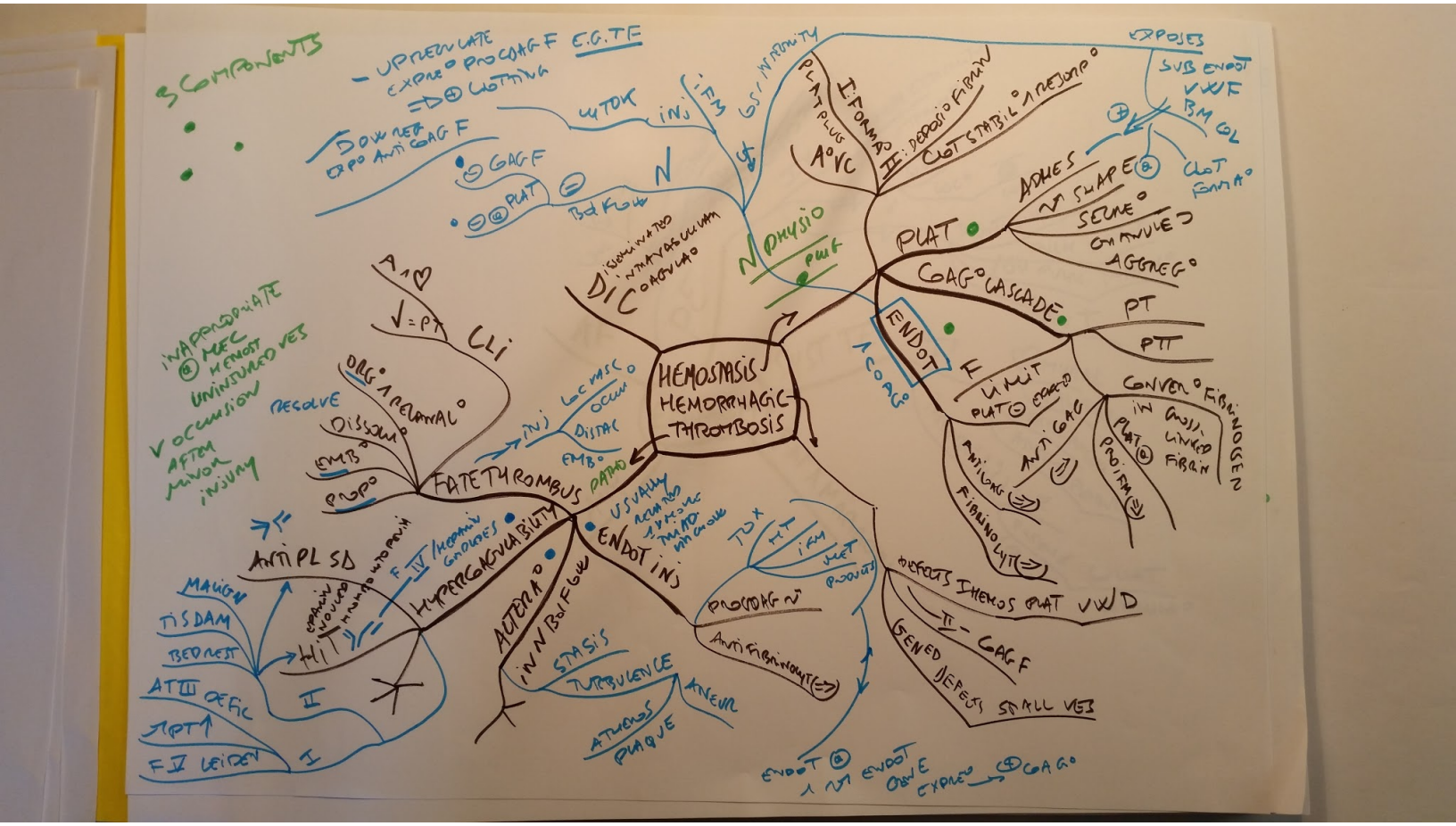

KEY CONCEPTS
EFFUSIONS
HYPEREMIA & CONGESTION
HEMOSTASIS
HEMORR
THROMBOSIS
CLOTTING
DIC
PE
ENDOT INJ
ALT° NRM FLOW
HYPERCOAGY
STASIS
THROMBUS

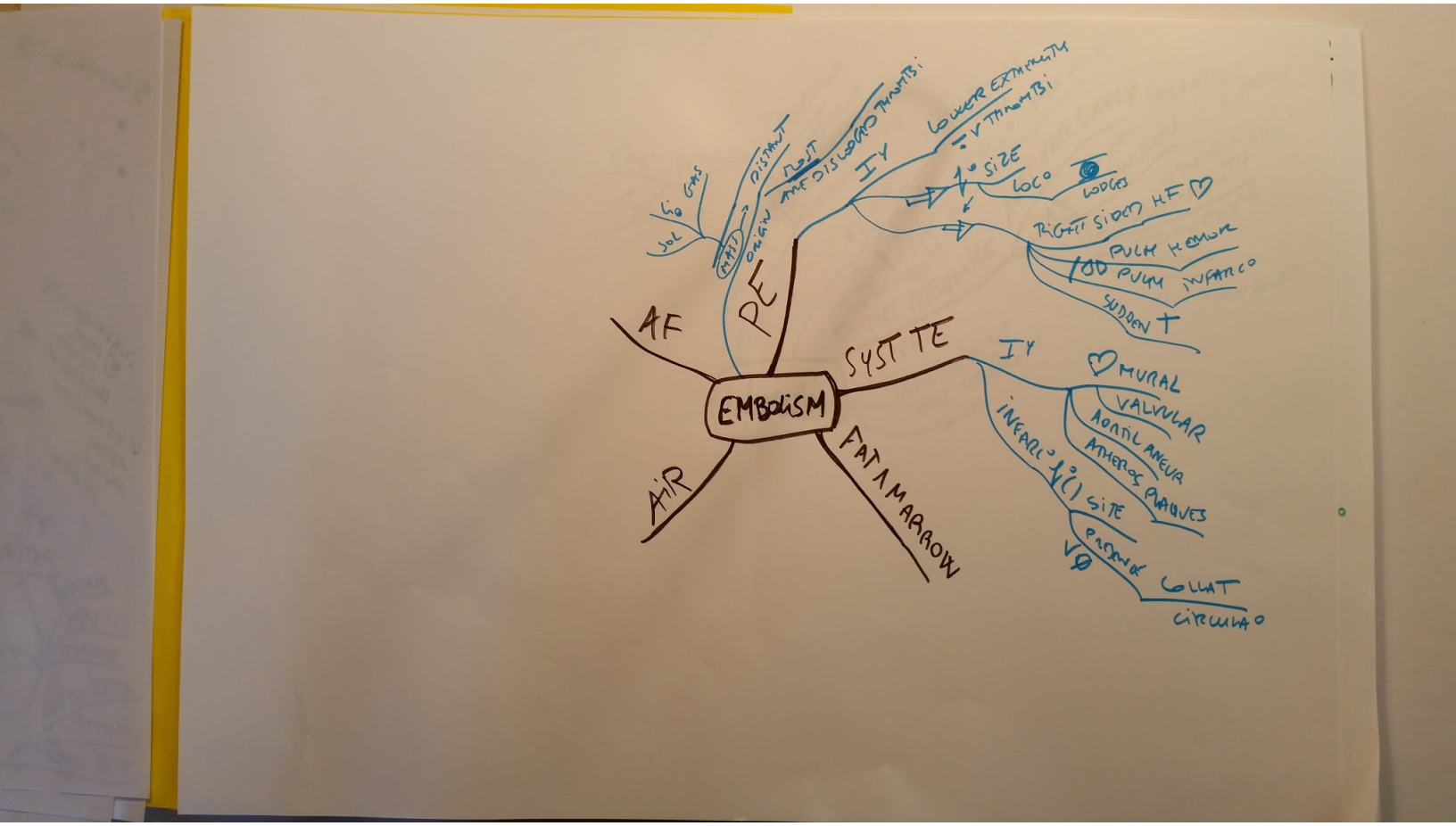

HEMODYN DISORD, THROMBO-EMBOLIC DIS & SHOCK
SHOCK
STAGES
PG SEPTIC
DISTURBANCES N Bd FLOW
DEATH
MORBIDITY
35-40 % MORT
EDEMA & EFFUSIONS
↑M → T
↑BP PRESSURE TOO LOW
TOO HIGH
↑ Na & H2O RETEN°
LYMP OBSTRUC°
HYPEREMIA & CONGESTION
HEMOSTASIS
DIAT
GAG° CASC
ENDOT
HEMORR
THROMBOSIS
ENDOT INJ
ALT° NBd FLOW
HYPERGAGY
FATE
THROMBUS
CLOTTING
DIC
EMBOLISM
AIR
FAT AMARROW
SEPTIC
INFARC°

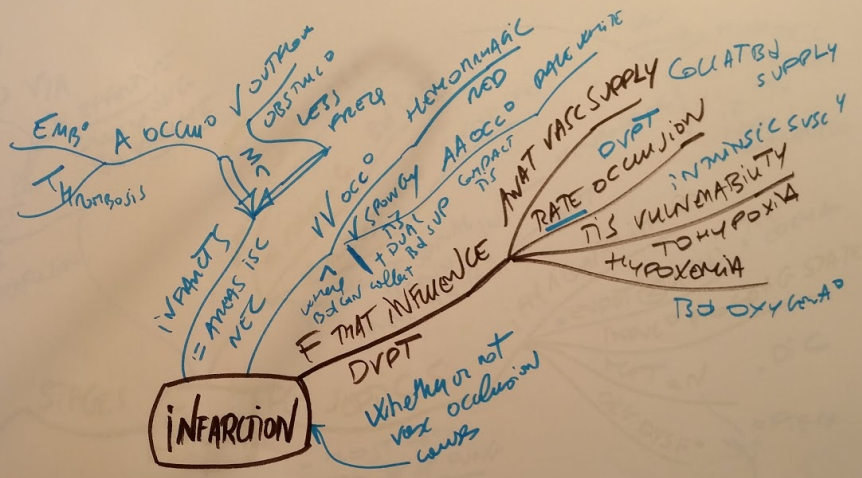

EDEMA & EFFUSIONS
↑ Pc
VASCULITIS
RESIST OUT FLOW
↑ INTERSTITIAL SPACE
↑ FLUID
↑ POOR = TRANSUDATE
↑ PROT EXUDATE
↑ HP
CHRONIC
FAILURE
↓ Pc
NEPH
IVC
LYMP OBST°
↑ Pi
↓ Π
↓ PLASMA ALB
↓ SYNTHESIS
LIVER D
↑ MALNUT°
↑ LOSS
NEPHROTIC SD
↑ Na⊕ H2O RET°
RENAL FAILURE
↑ VASC PERM
↓ FM
USUALLY LOC°
MAY OCCUR THROUGHOUT BODY
SEVERE SEPT
↑ CYT STATES
SEPSIS

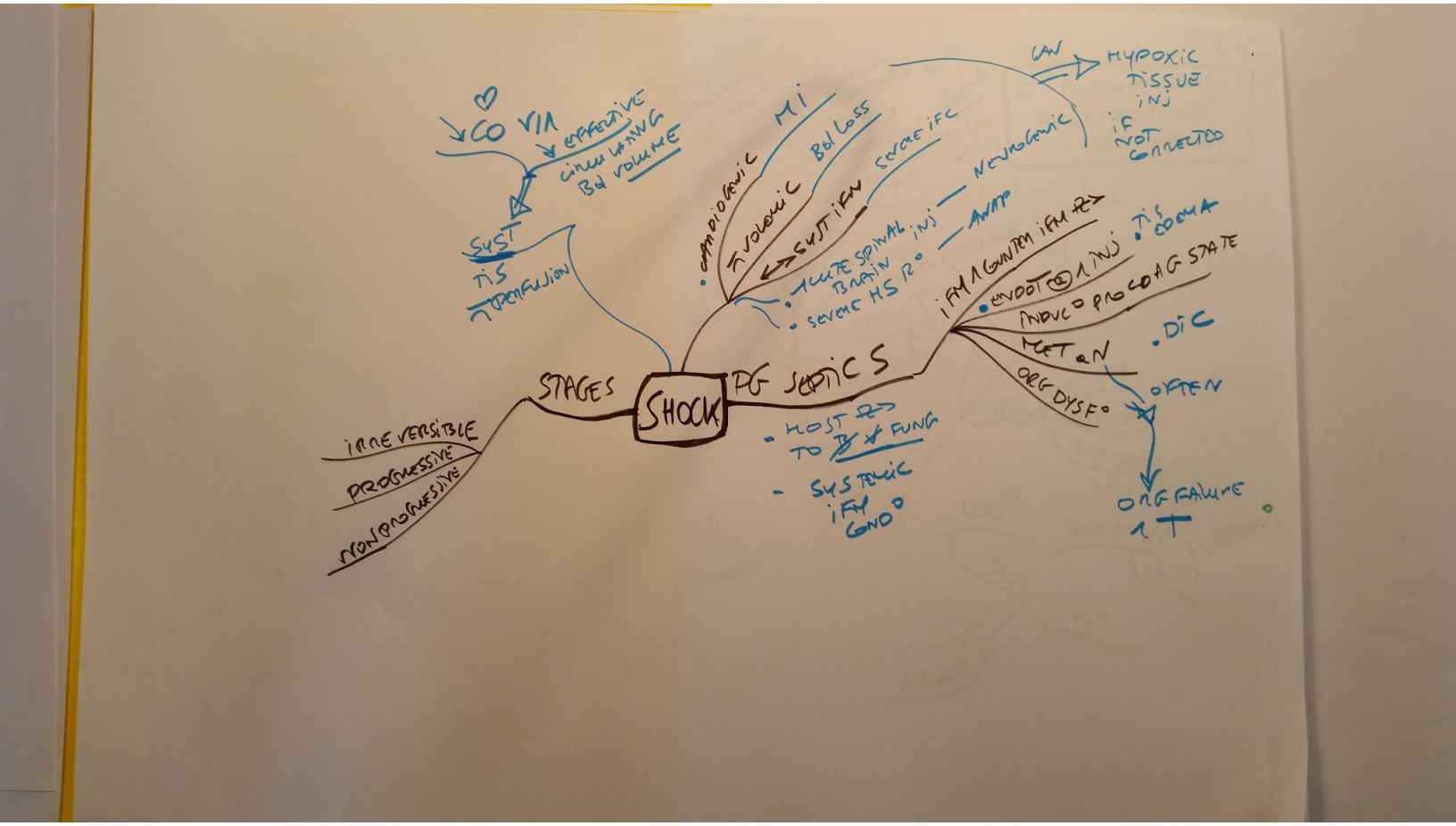
HYPERÆMIA
OR
CONGES°

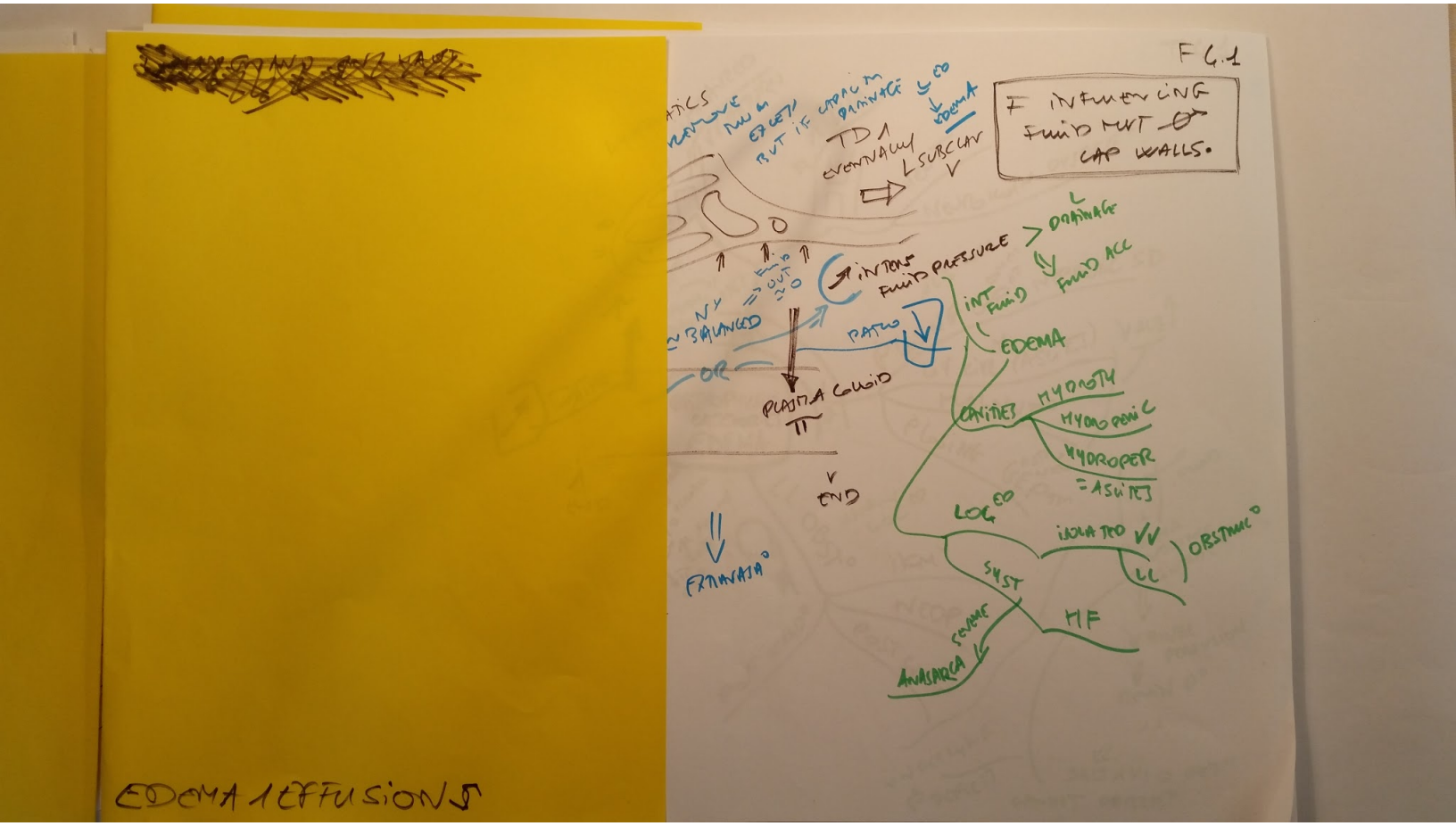

PLAT
INJ
ECM
PLAT
GRANULE
ADP
FURTHER PLAT
ACTIVES
SHAPE
MB
GMPIIB/IIIa R
PLAT
BRIDGING CROSS LINKS
WITH FG
CONGM
FIBRIN
AGGREGATA
FIBRIN
FIBRINOLYTIC PATHS
AN
ANTAGONIST
CONGT
SERVENT ENZ
GIVEN
CASCADE
CIRCULATING
LOC
P
TF
MOST IMP
IN VIVO
INITIATOR
INITIAT
ENHANCED AT SITES INJ
T
FG
FORMS
PERMANENT
PLUG
NATURAL
ANTICOAG
LIMITING ENZ
TO SITES VASC INJ
FINAL STAGE
WRESTRICTED
TO SITES
ENDOT
PLAT
ENDOT


HEMOSTASIS
HEMORRHAGIC
THROMBOSIS
3 COMPONENTS
UPREGULATE
EXPRE° PROCOAG F E.G. TF
=D⊕ CLOTTING
DOWNREG
EXPO ANTICOAG F
VITOK
INJ
TEM
GS / INFAMITY
PLAT PLUG
A°VC
FORMED II: DEPOSI° FIBRIN
CLOT STABIL° / REJUVNO°
EXPOSES
SUB ENDOT
VWF
BM
COL
ADHES
SHAPE
CLOT RETRA°
SECRE°
ON ACTIVE
AGGREG°
PLAT
COAG° CASCADE
PT
PTT
CONVER° FIBRINOGEN
IN CROSS-LINKED
FIBRIN
ANTI COAG
PRO FIBRIN
FIBRINOLYT
ENDOT
ACOAG°
LIMIT
PLAT
DEFECTS INHERIT PLAT VWD
GENED DEFECTS SMALL VES
TF
GAG F
ENDOT
ACTIVE
EXPRES
COAG°
DIC
DISSEMINATED
INTRAVASCULAR
COAGULO°
PHYSIO
PWF
A
PT
CLI
INAPPROPRIATE
MEC
HEMOST
UNINTURBED VES
OCCLUSION
AFTER
MINOR
INJURY
RESOLVE
DISSOLV°
EMB°
PROPO
ORG° / INFLAMM°
FATE
THROMBUS
PATHO
INJ
LOC MISC°
OCCLU
DISTAL
EMB°
ANTIPL SD
MAIGN
TISDAM
BED REST
HIT
HEPARIN INDUCED THROMBOCYTOPENIA
F IV
HEPARIN CAUSES
HYPERCOAGULABILITY
ATIII DEFIC
↗PT↑
F II LEIDEN
I
II
ENDOTINJ
USUALLY ACQUIRED SYSTEMIC INJURY MEDIATED BY CYTOKINES
AUTOM°
IN/N BLOOD FLOW
STASIS
TURBULENCE
ANEUR
ATHEROS
PLAQUE
TUX
HIT
IAM
VHIT
PRODUCTS
PROCOAG↑
ANTIFIBRINOLYT(⇒)

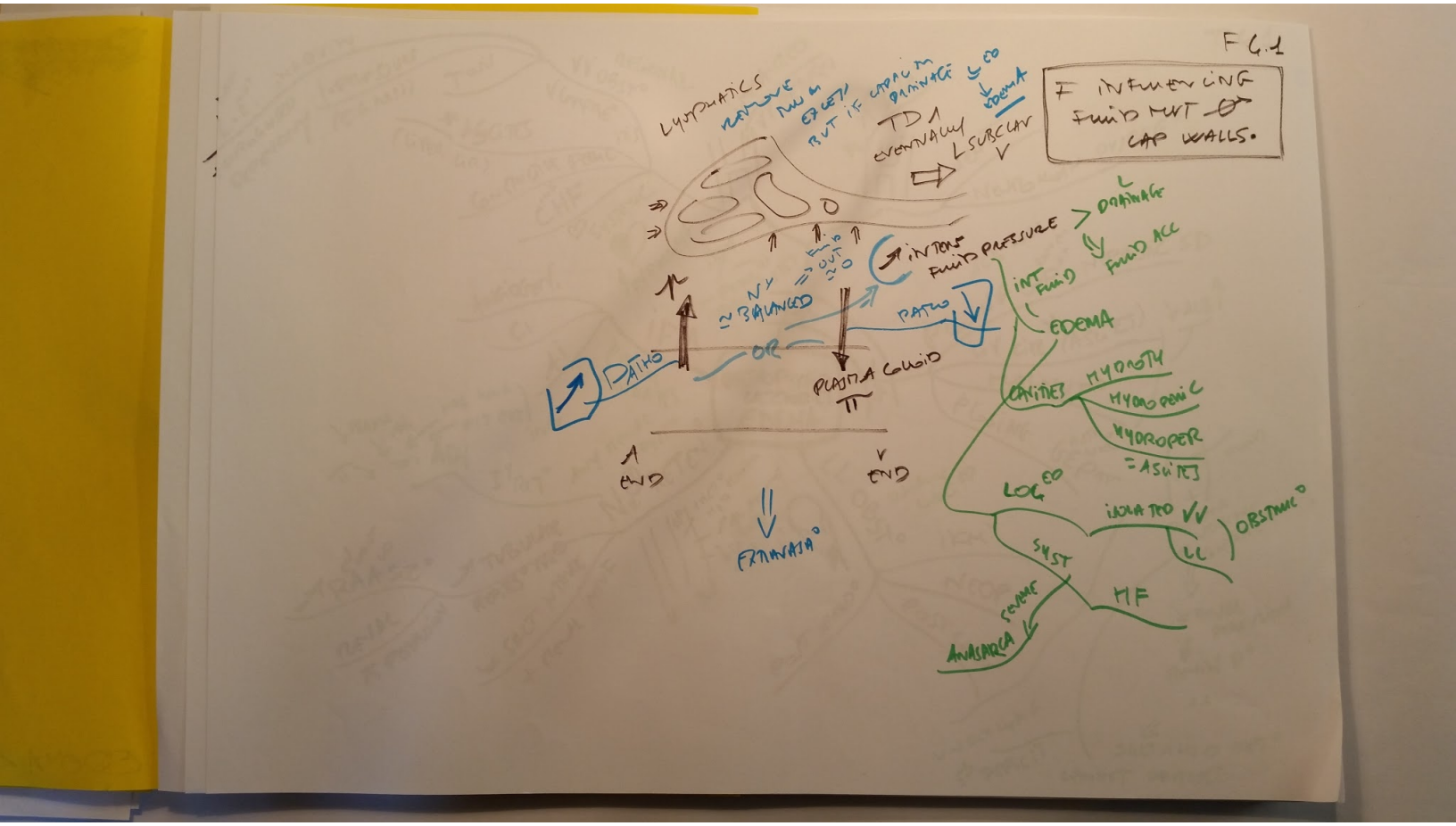

EMBOLISM
AF
PE
SYST TE
AIR
FAT MARROW
IV
LOWER EXTREMITY
↑ VTHROMBI
↓ SIZE
LOC
LODGES
RIGHT SIDED HF
PULM HEMORR
↓↓ PULM INFARCT
SUDDEN ↓
IV
MURAL
VALVULAR
AORTIC ANEUR
ATHEROS PLAQUES
INFARCT 3° ↓ CI SITE
PARADOX
↓ BP
COLLAT
CIRCULAT
RAPID
DISTANT
ORIGIN
LOST
NO DISLODGED THROMBI
VOL
V/Q

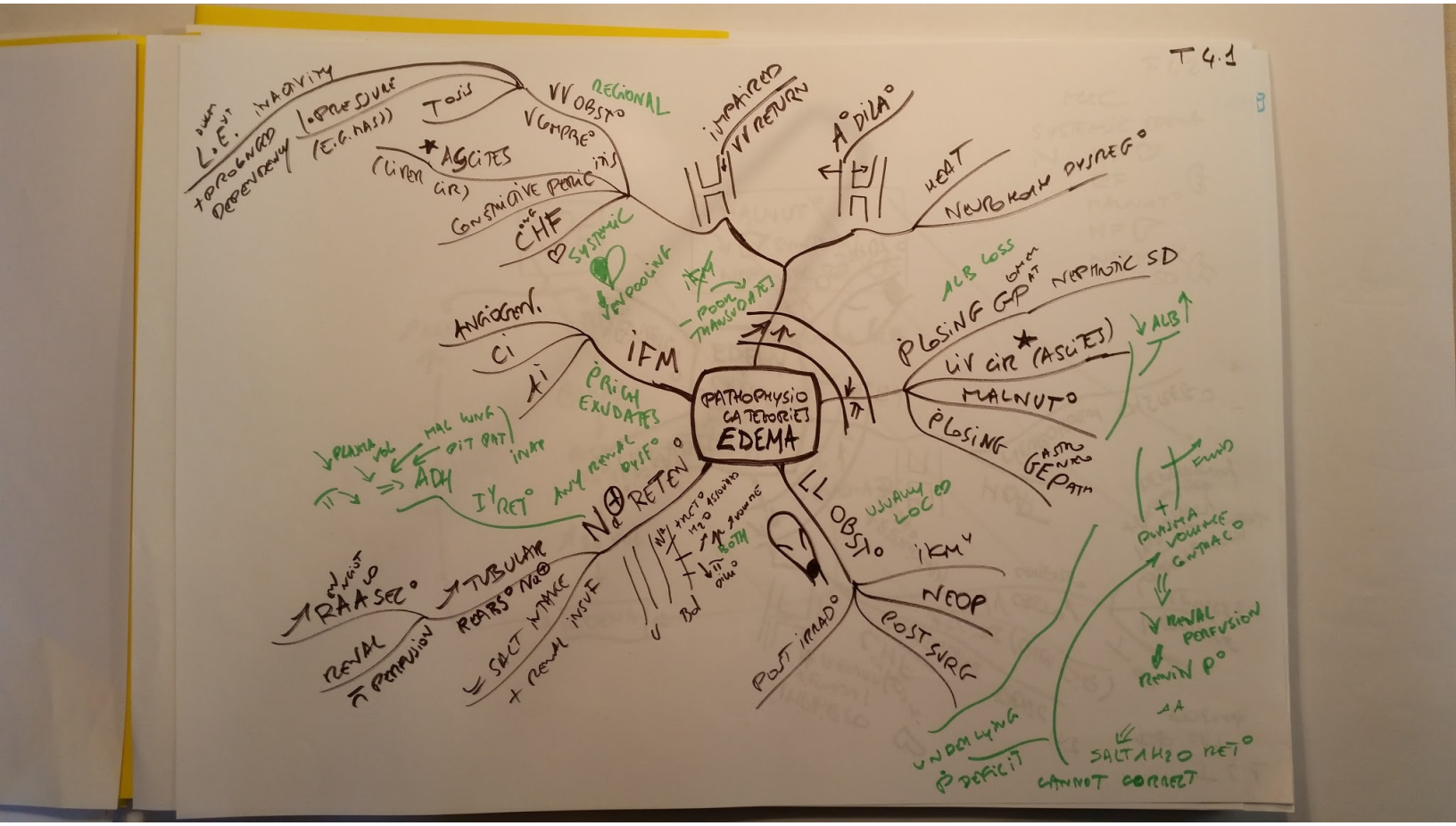

INFARCTION
F THAT INFLUENCE
DVPT
Whether or not vax occlusion causes
ANAT VASC SUPPLY
COLLATRL SUPPLY
DVPT
OCCLUSION
RATE
INTRINSIC SUSC
TIS VULNERABILITY
TO HYPOXIA
HYPOXEMIA
TO OXYGENATED
VV OCC
AA OCC
HEMODYNAMIC NEED
EMB
A OCCLUD
THROMBOSIS
OBSTRUCTED
LESS FREELY
IMPACTS
= ANOXIS ISC NEC


CO
VIA
EFFECTIVE
CIRCULATING
BV VOLUME
SYST
TIS
HYPERFUSION
CARDIOGENIC
MI
HYPOVOLEMIC
BLOOD LOSS
SEPTIC
SEVERE IFC
NEUROGENIC
ACUTE SPINAL INJ
BRAIN
SEVERE HSR
ANTP
CAN
HYPOXIC
TISSUE
INJ
IF
NOT
CORRECTED
STAGES
SHOCK
PATHOPHYSIOLOGY
IRREVERSIBLE
PROGRESSIVE
NONPROGRESSIVE
HOST
TO
FUNG
SYSTEMIC
INFL
GNO
INFLAMM/IMM
ENDOT/INJ
TIS EDEMA
INDUC PROCOAG STATE
MET AN
DIC
ORG DYSF
OFTEN
ORG FAILURE
↑ T



EDEMA / EFFUSIONS


F G.1
LYMPHATICS
REMOVE
PUMP
EFFECT
BUT IF
TD
EVENTUALLY
SUBCLAV
F INFLUENCING
FLUID MVT
CAP WALLS.
INTERS
FLUID PRESSURE
INT
FLUID
EDEMA
PATHO
OR
PLASMA COLLOID
END
END
FIBROSIS
CAVITIES
MYOSTY
HYDROPER
ASCITES
ISOLATED
OBSTRUC
SYST
HF
ANASARCA

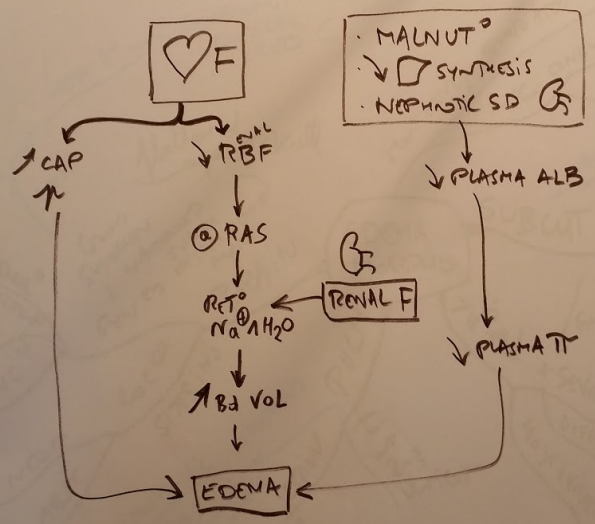

T 4.1
PATHOPHYSIO CATEGORIES EDEMA
IFM
NO RETEN
LL OBST°
A° DILA°
PLOSING GP
PLOSING GEPATH
MALNUT°
LIV CIR (ASCITES)
NEPHROTIC SD
ALB LOSS
ALB
ANGIOGRAM
CI
AI
PRICH EXUDATES
ADH
RAAS
RENAL
TUBULAR
SALT INTAKE
RENAL INSUF
IKM
NEOP
POST SURG
POST IMMAD°
HEAT
NEUROGEN DYSREG°
CHF
CIRRH CIR
ASCITES
TOXIN
VV OBST°
VCOMPRE°
CONSTRICTIVE PERIC
SYSTEMIC
VENOCONSTR
POOR TRANSDUCT°
RENAL PERFUSION
RENIN
SALT & H2O RET°
CANNOT CORRECT
UNDERLYING DEFICIT

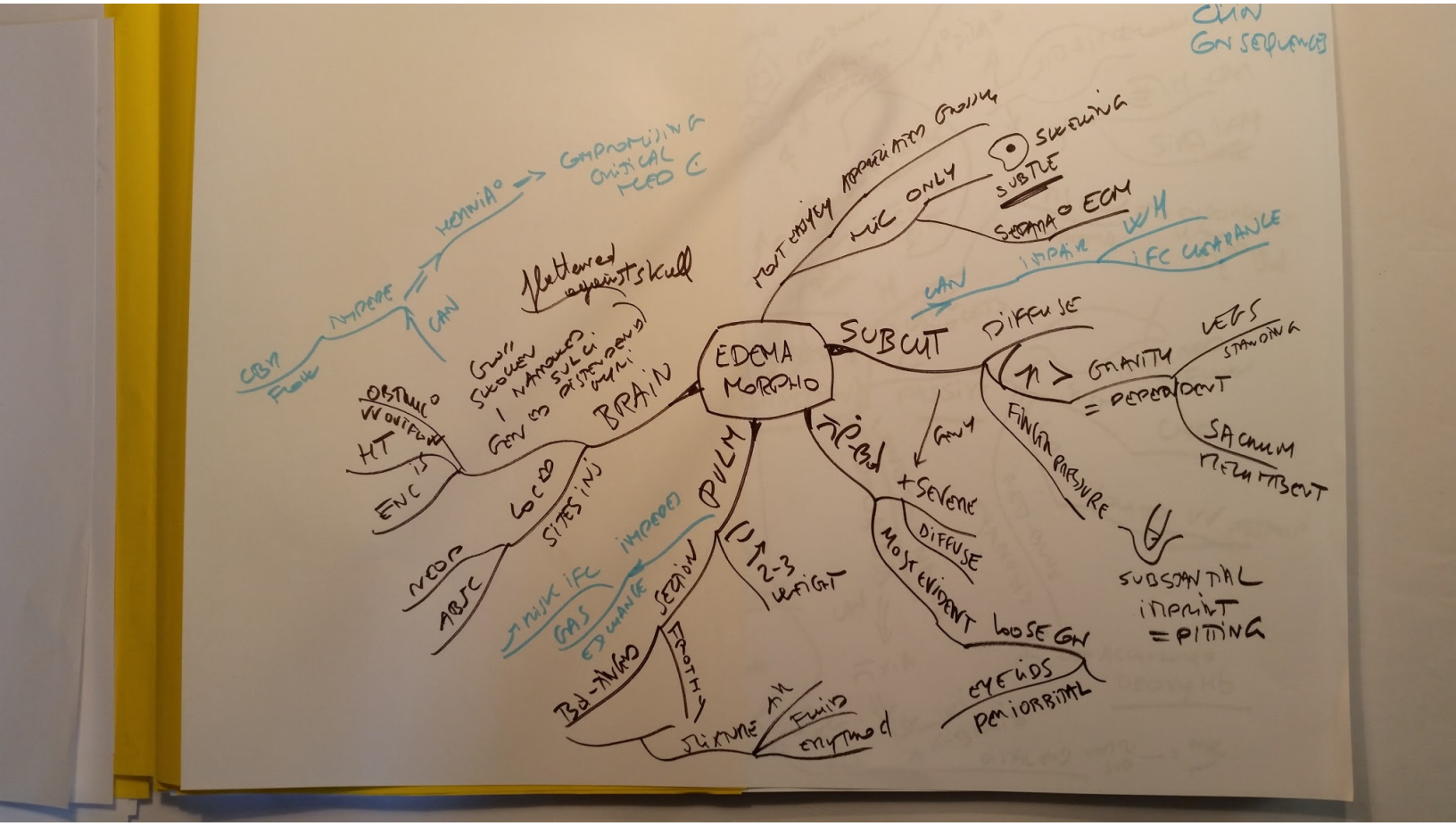
F 4.2

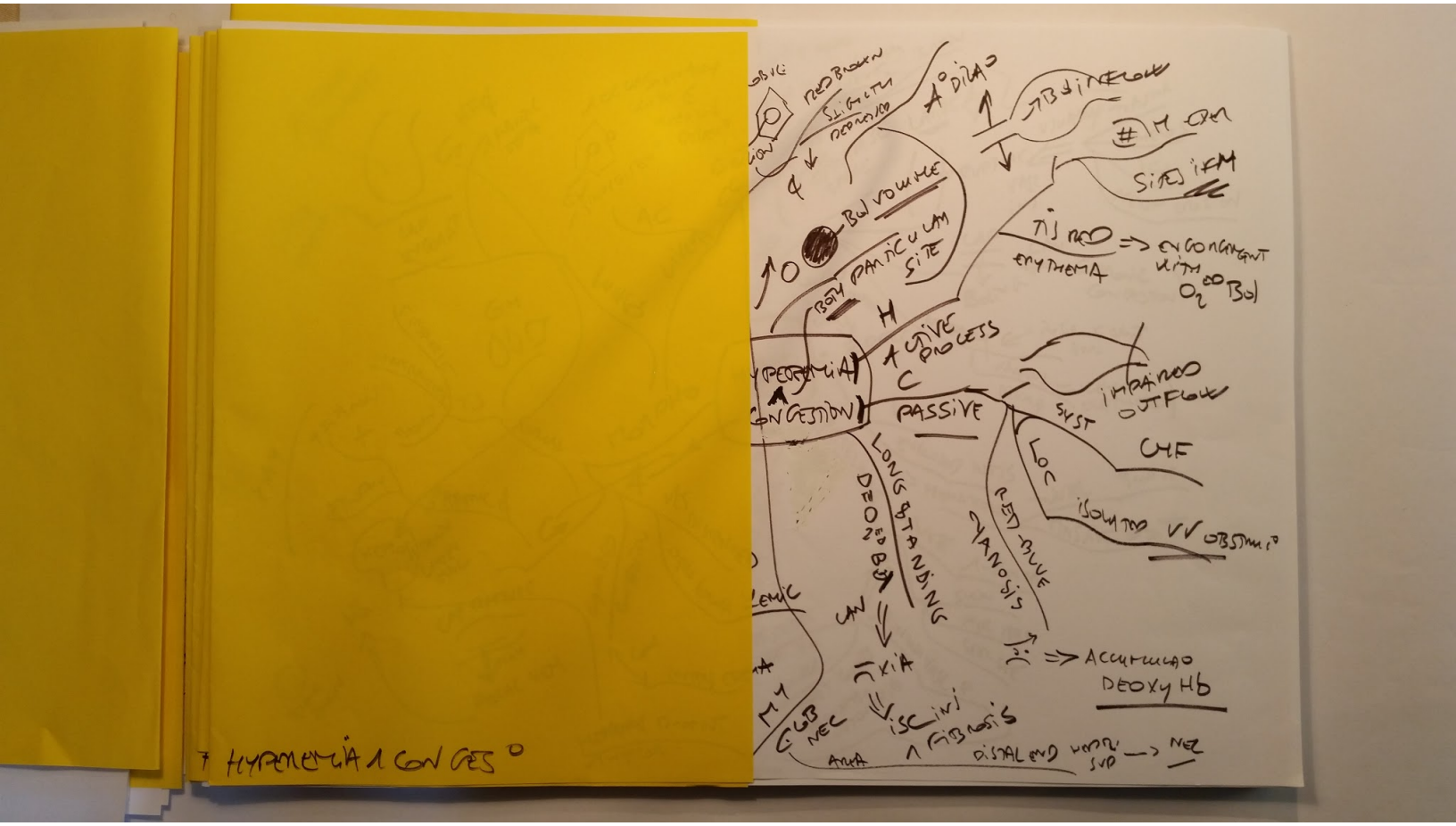
MEC

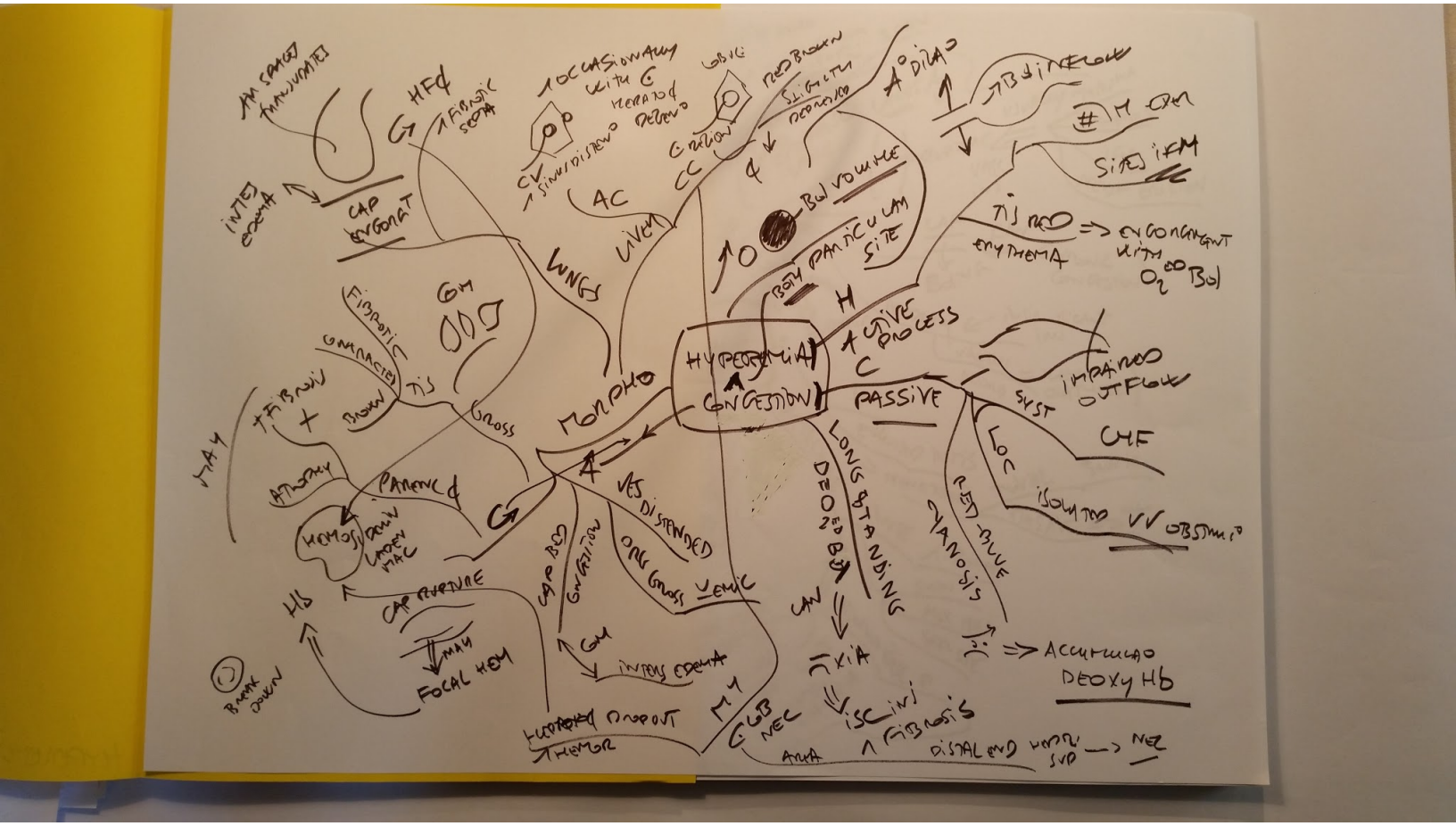
SYSTEMIC OEDEMA

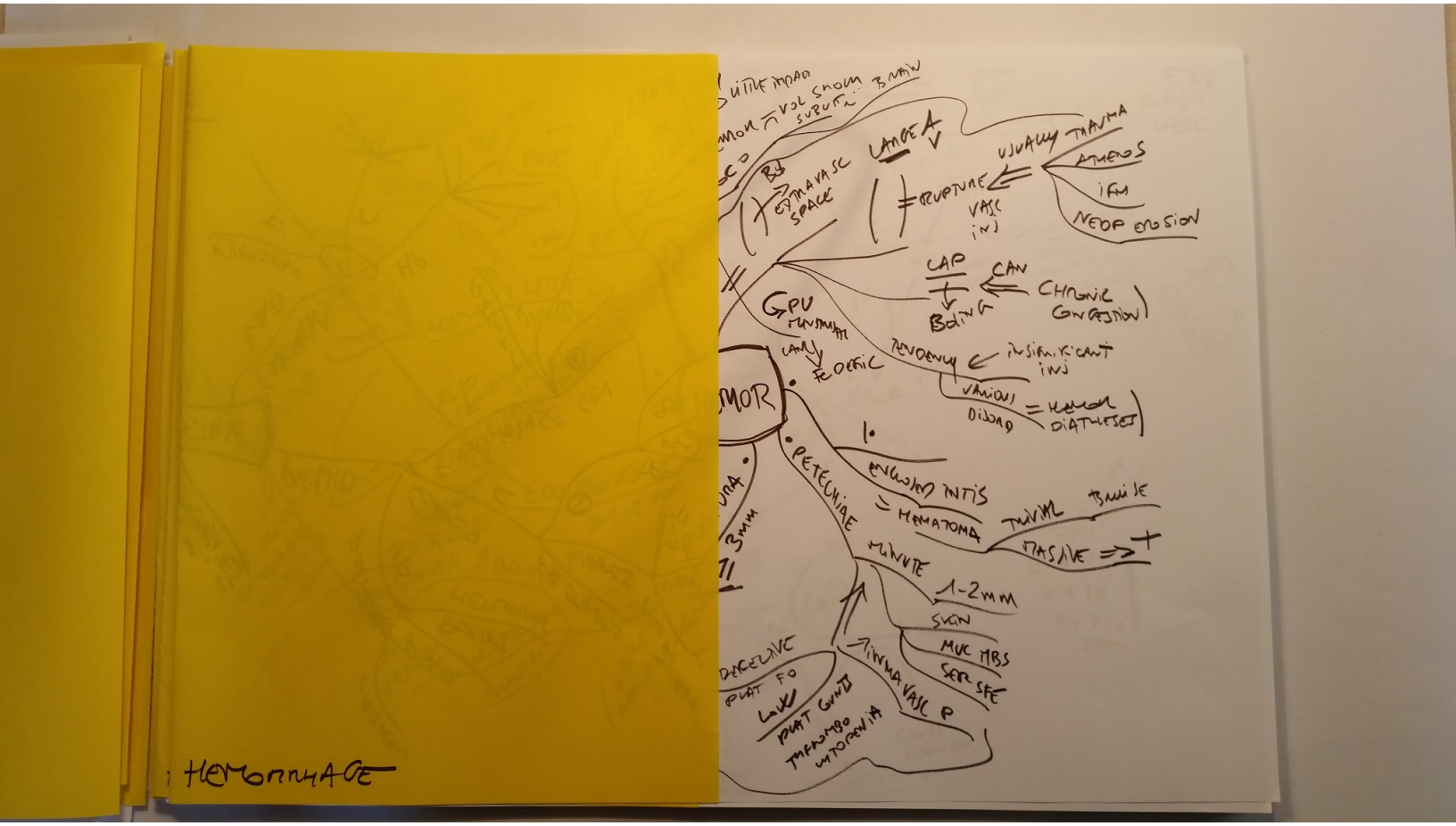
IN HF
RF
MALNUT°
HF
NEPHROTIC SD

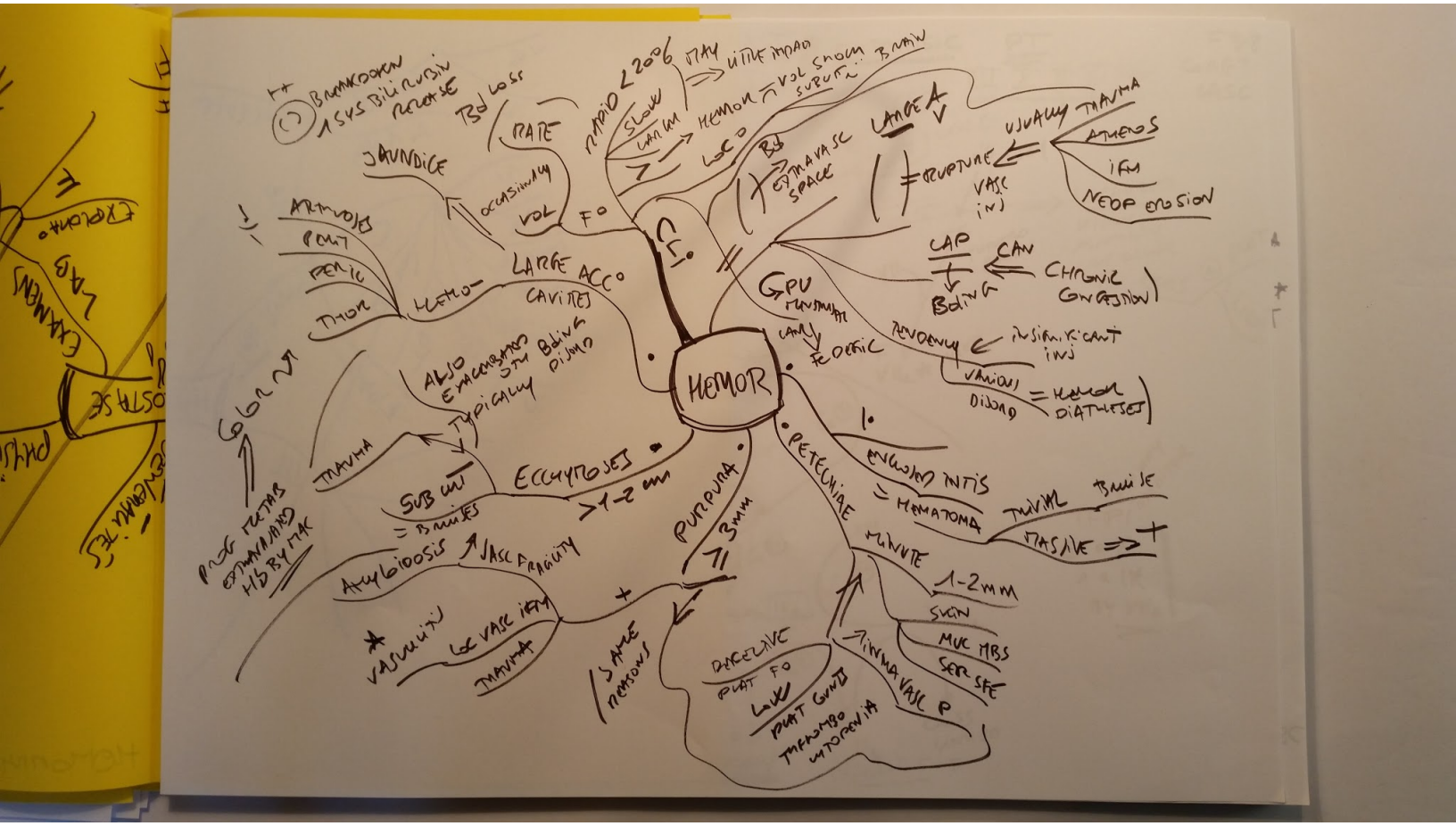


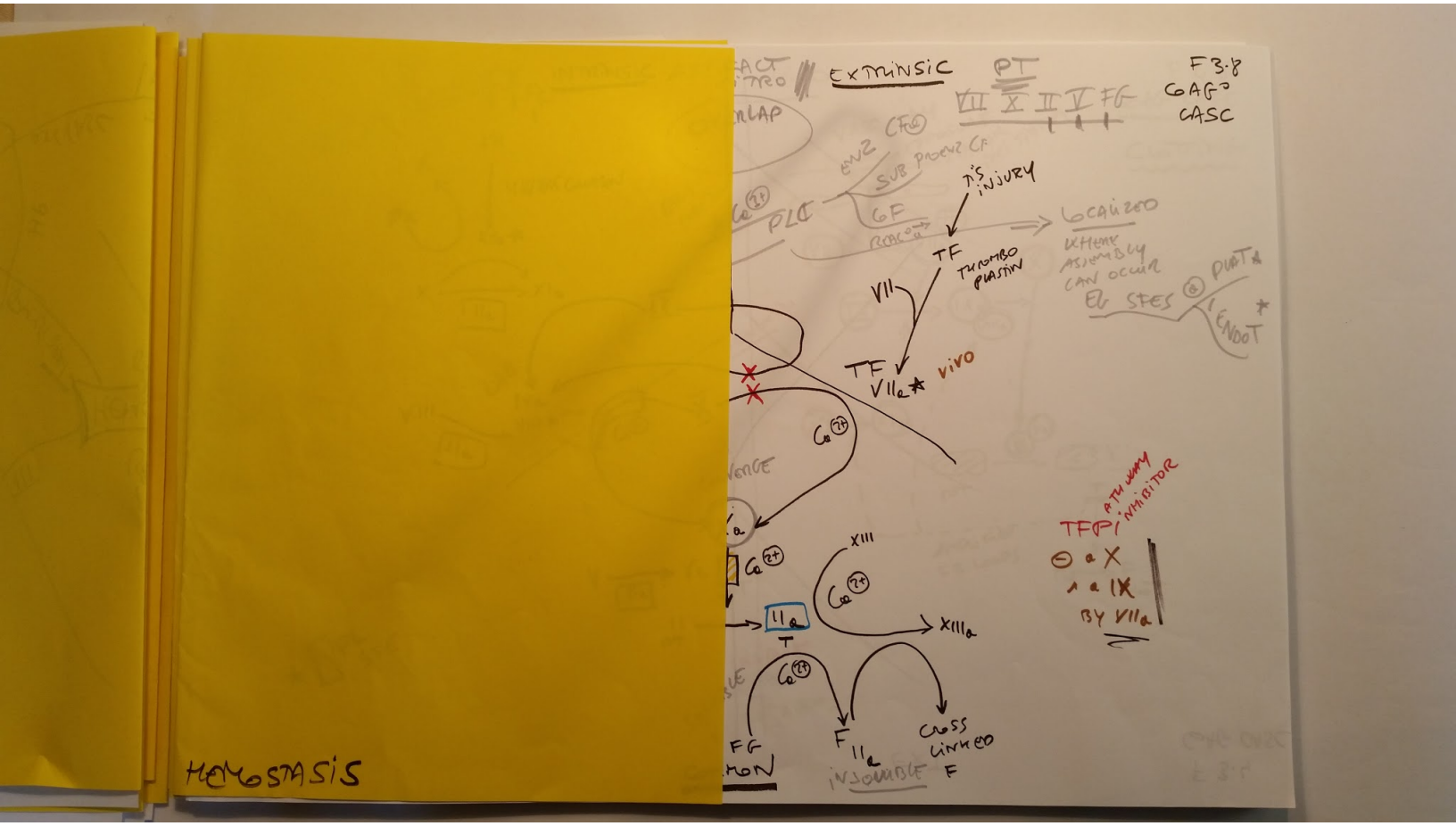

EDEMA MORPHO
BRAIN
SUBCUT
PULM
CBV
CBF
HERNIA
COMPROMISING CRITICAL NEO
CAN
flattened agains skull
GW SHIFT
MIDLINE
SULCI
OBSTRUCTIVE HYDROCEPH
HT
ENC
LOCAL SITES INJ
NEOP
ABTC
MOST EVIDENT
SUBTLE
SEVERE
DIFFUSE
LOOSE CT
EYELIDS
PERIORBITAL
SUBSTANTIAL IMPRINT
=PITTING
FINGER PRESSURE
LEGS
SACRUM
BI-NARD
FROTH
FLUID
ERYTHEMA
IMPAIR
IFC CLEARANCE
GRAVITY
DEPENDENT

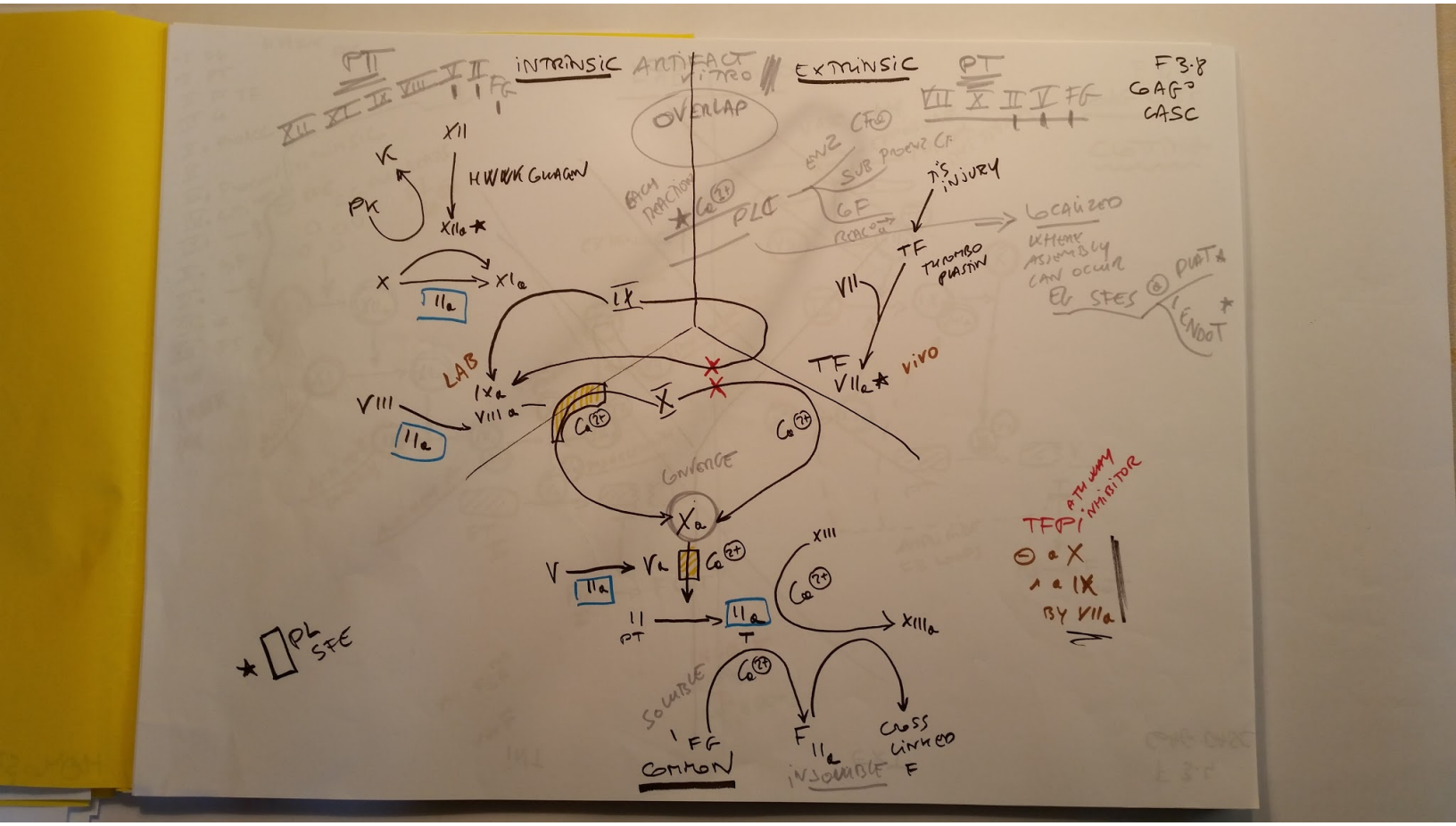

↑ HYPERAEMIA / CONGES°
RED BROWN
SLIGHTLY
DEOXY Hb
↑ BV VOLUME
↑ INTERSTITIAL SITE
H
ACTIVE PROCESS
C
PASSIVE
SYST
CHF
LOC
ISOLATED
LONG STANDING
DEOXY Hb
CAN
ANOXIA
ISCHAEMIA
ACCUMULATED
DEOXY Hb
DISTAL END
NEC

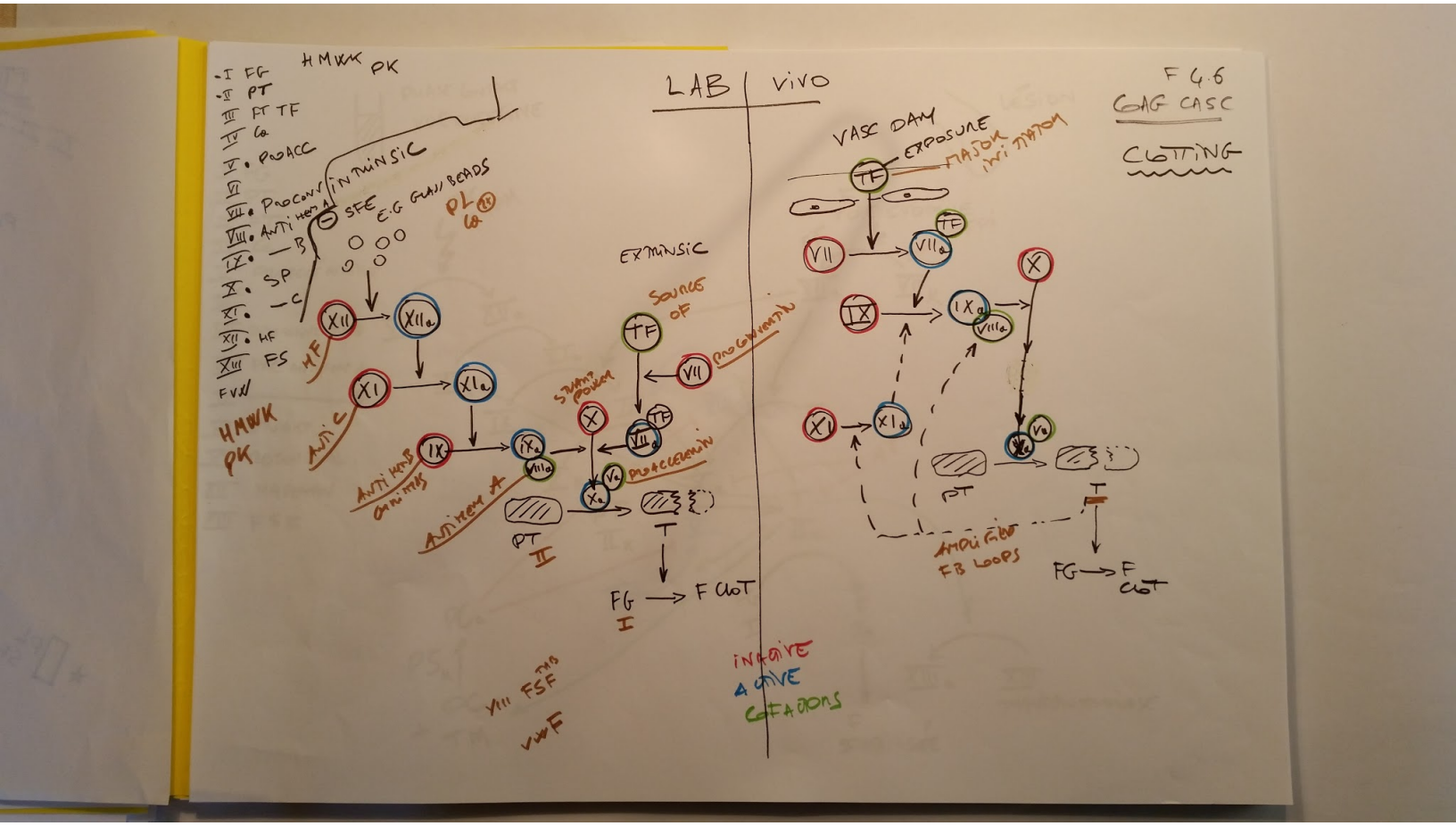

HYPERTHERMIA
CONGESTION
ACTIVE PROCESS
PASSIVE
MORPHO
WNL
CAP BVGDAT
INTER ECCHY
HFd
FIBROTIC SEPTA
OCCASIONALLY WITH C
CV
SINUSOIDAL
AC
CC
LOBIC
RED BROWN
SLIGHTLY
DIAD
BLOOD VOLUME
PERIPHERAL SITE
TIJ INCR
ERYTHEMA
ENCOURAGEMENT
WITH O2
SITES IEM
IMPAIRED OUTFLOW
SYST
CHF
LOC
ISOLATED
VV OBSTRUCT
LONG STANDING
DEOXY Hb
RETROGRADE
VANOSIS
ACCUMULATED
DEOXY Hb
FIBROTIC
CONNECTED
TIJ
BROWN
GROSS
PATENCY
HOMOGENOUS
LACEY PATC
CAP PERVIOUS
FOCAL HEM
VES DISTENDED
ONG GROSS
CAP BED
CONGESTION
GM
INTRA EDEMA
HAEMORRHAGIC INFARCT
THROMBUS
CLOT
NEC
AREA
FIBROSIS
DISTAL END
NEC
BLOOD

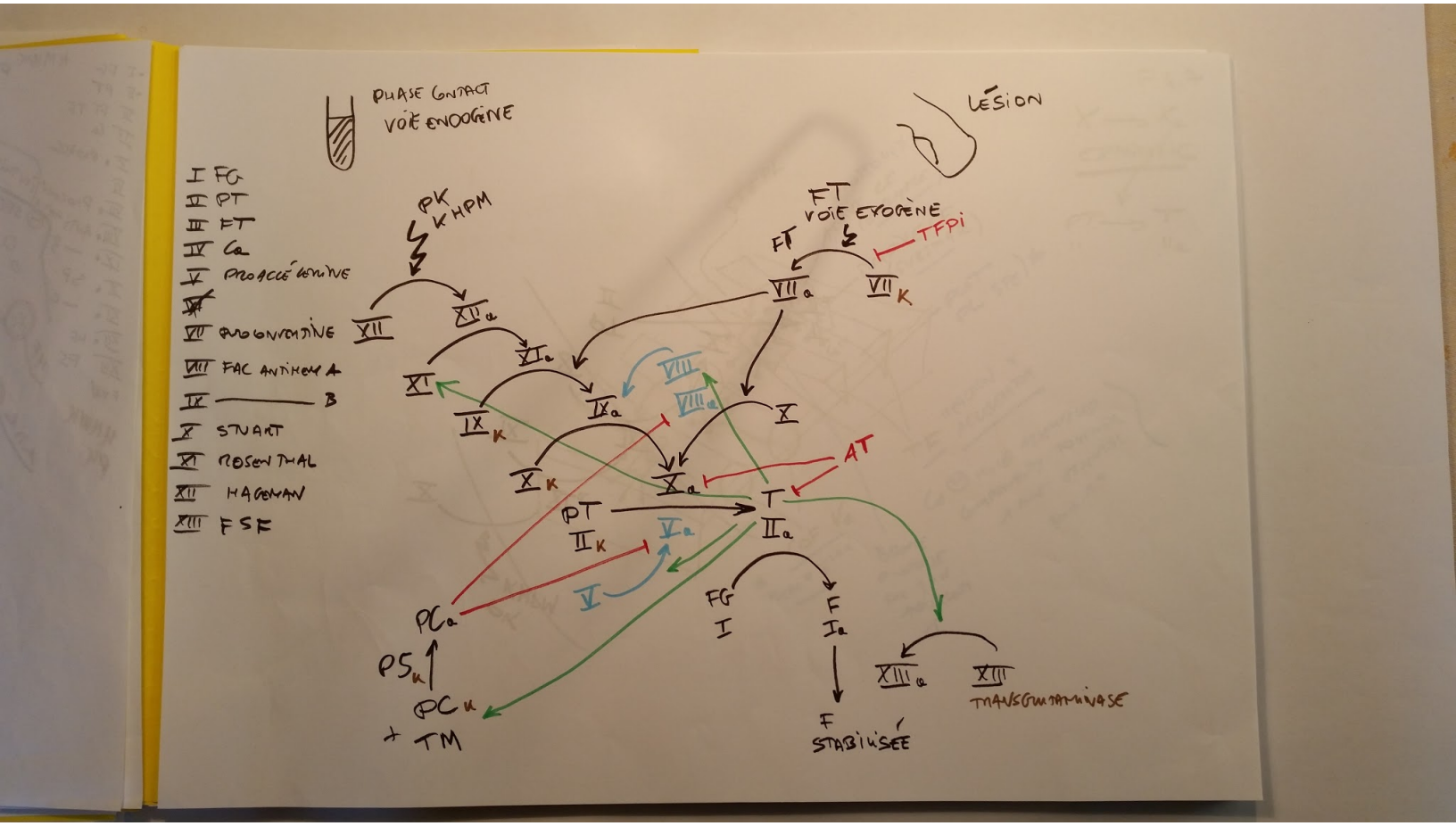

HEMORRHAGE
LARGE
ATHEROS
IFM
CHRONIC
TRAUMA
MASSIVE
1-2mm
SKIN
MUC MBS
SER SEE

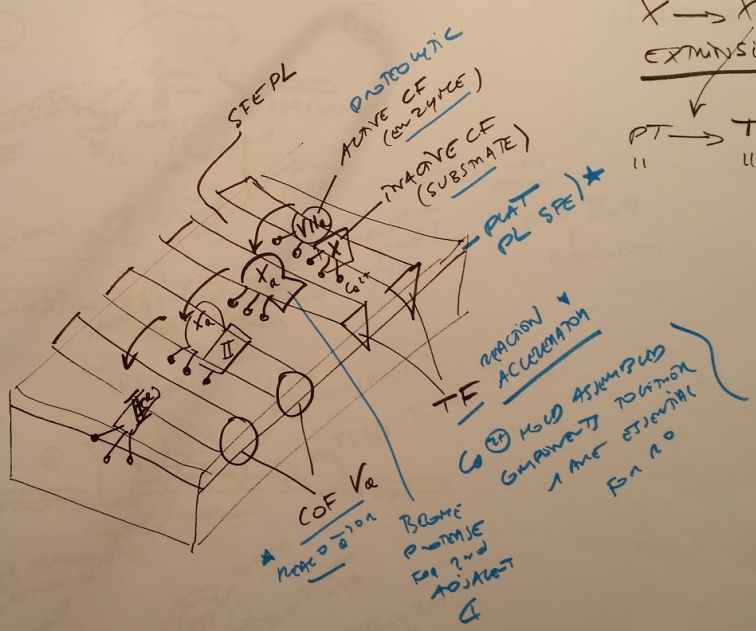

HEMOR
RATE
RAPID
SLOW
JAUNDICE
LARGE ACC
CAVITIES
TRAUMA
ECCHYMOSES
PURPURA
PETECHIAE
HEMATOMA
SUBCUT
GPU
CAP
CAN
CHRONIC CONGESTION
USUALLY TRAUMA
ATHEROS
IFM
NEOP EROSION
RUPTURE
LARGE
LOC
FO
VOL
ATHEROSES
PCM7
PERIC
THOR
HEMOL
ALSO EXTRAVASATED STH BEING DIAMS
TYPICALLY
ATHEROSCLEROSIS
VASCULITIS
LOC VASC INFM
TRAUMA
PLAT FO
LOW
PLAT GUNS
THROMBOCYTOPENIA
SMALL
MEDIUM
1-2MM
SKIN
MUC MBS
SER SFC
INFLAMM VASC P
MINUTE
MASSIVE
TRAUMA
ENCLOSED INTIS
RUPTURE
VASC INJ
SIGNIFICANT INJ
REVOENUM
VARIOUS DIBNDS
= HEMOR DIATHESES
CARDIAC FEMORIC
BRAIN
LIVER
SPLEEN
BONE MARROW
BLOOD LOSS
BRAINSTEM
1 SYS BILIRUBIN
RELEASE
MAY
LITTLE IMPACT
SHOCK
SUBCUT
= BRUISES
>1-2 cm
>3mm
PROC TESTS
ESTIMATIONS
HISTOLOGY


HEMOSTASIS
EXTRINSIC
PT
VII X II V FG
F3.8
GAG°
CASC
PLC
TF
THROMBOPLASTIN
VII
TF VIIa*
VIVO
LOCALIZED
ENDOT
XIII
XIIIa
IIa
THROMBIN
FIIa
CROSS LINKED F
INSOLUBLE
TFPI
INHIBITOR
BY VIIa


PT
INTRINSIC
ANTIFACT in vitro
EXTRINSIC
PT
F 3.8
GAG
CASC
OVERLAP
XII
HMWK GLUCAGON
PK
XIIa
X
Xa
IIa
IX
LAB
IXa
VIII
VIIIa
IIa
Ca2+
CONVERGE
Xa
V
Va
IIa
II
PT
IIa
T
XIII
XIIIa
SOLUBLE
FG
COMMON
FIIa
INSOLUBLE
CROSS LINKED F
ENZ CF
SUB PHOSPH CF
PLC
GF
TF
TISSUE INJURY
THROMBOPLASTIN
VII
TF VIIa
VIVO
LOCALIZED
HEMOST ASSEMBLY CAN OCCUR
ES SITES
PLAT
ENDOT
TFPI
ANTHROMBIN INHIBITOR
a X
a IX
BY VIIa
PL SITE

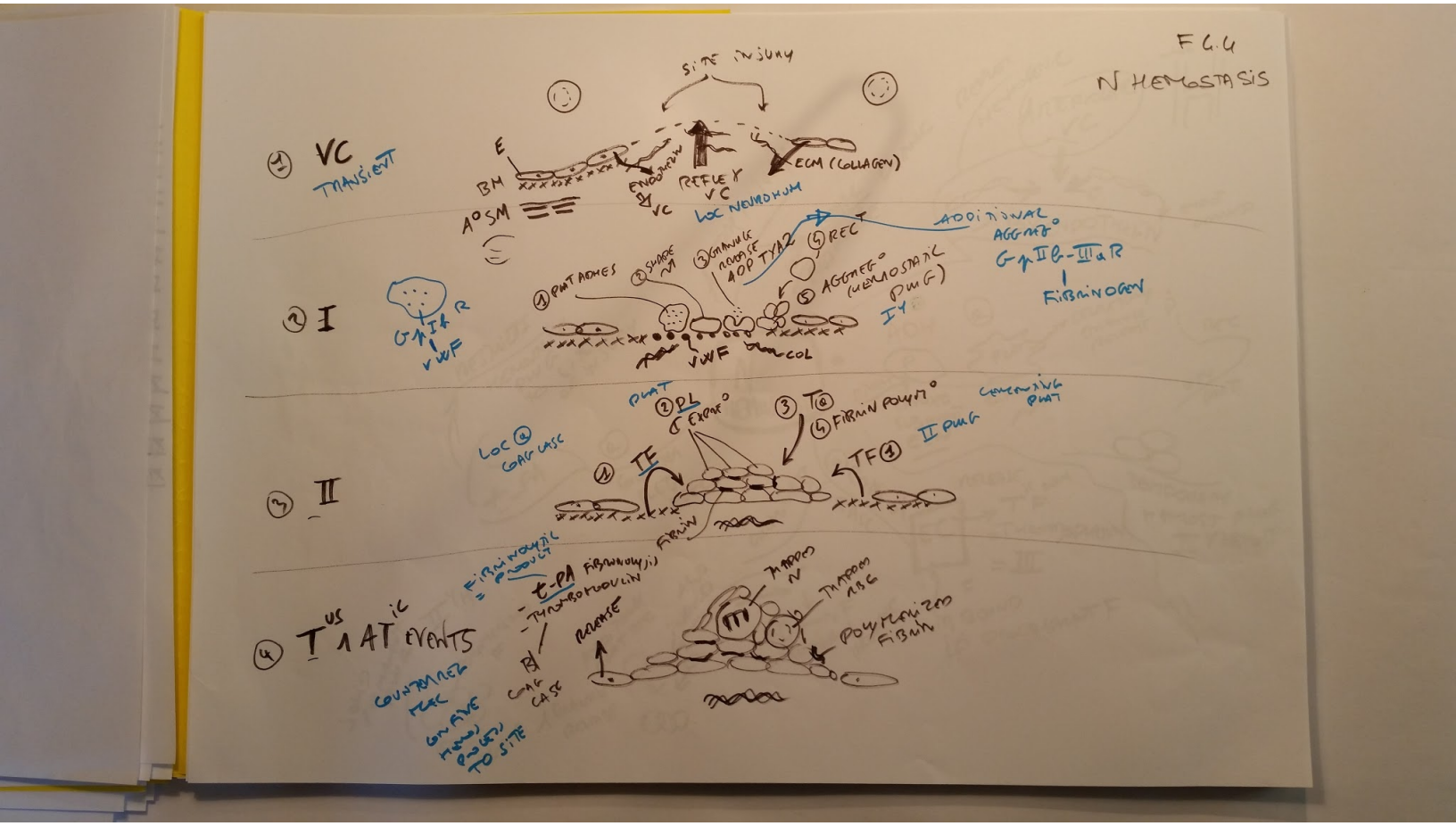

HMWK PK
LAB | VIVO
F 4.6
CAG CASC
CLOTTING
I FG
II PT
III FT TF
IV Ca
V • ProACC
VI
VII • ProConv
VIII • Anti Hem A
IX • — B
X • SP
XI • — C
XII • HF
XIII FS
FVW
HMWK PK
INTRINSIC
SEE
E.G GLASS BEADS
PL Ca
XII
XIIa
HF
XI
XIa
Anti C
IX
Anti HMWK Chemistry
IXa
VIIIa
Anti Hem A
PT II
EXTRINSIC
Source of
TF
VII
Pro Conversion
Strong Positive
X
VIIa
Va
Pro Acceleration
Xa
T
FG I
F CLOT
VIII FSF
vWF
INHIBITIVE & DRIVE CASCADES
VASC DAM
EXPOSURE
MAJOR INFLAMMATION
TF
VII
VIIa
IX
IXa
VIIIa
X
XI
XIa
Va
PT
T
AMPLIFIER FB LOOPS
FG
F CLOT

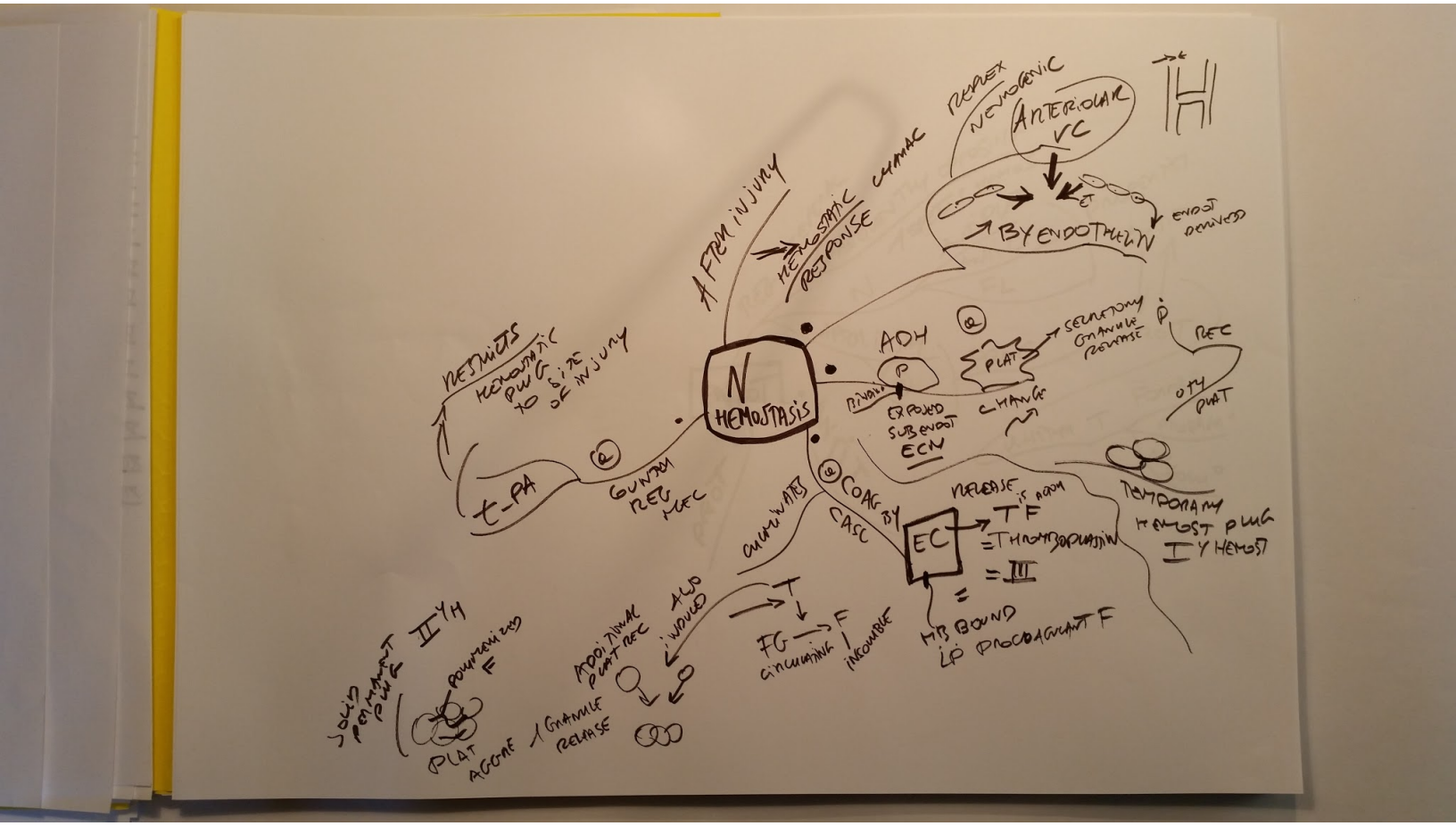

PHASE CONTACT
VOIE ENDOGÈNE
LÉSION
I FG
II PT
III FT
IV Ca
V PROACCÉLÉRINE
VII PROCONVERTINE
VIII FAC ANTIHEM A
IX ——— B
X STUART
XI ROSENTHAL
XII HAGEMAN
XIII FSF
PK
K HPM
XII
XIIa
XIa
XI
IXa
IX K
VIII
VIIIa
X
X K
Xa
AT
T
PT
II K
IIa
Va
V
PCa
PS K
PC K
+ TM
FG
I
F
Ia
F
STABILISÉE
XIIIa
XIII
TRANSGLUTAMINASE
FT
VOIE EXOGÈNE
FT
TFPi
VII a
VII K

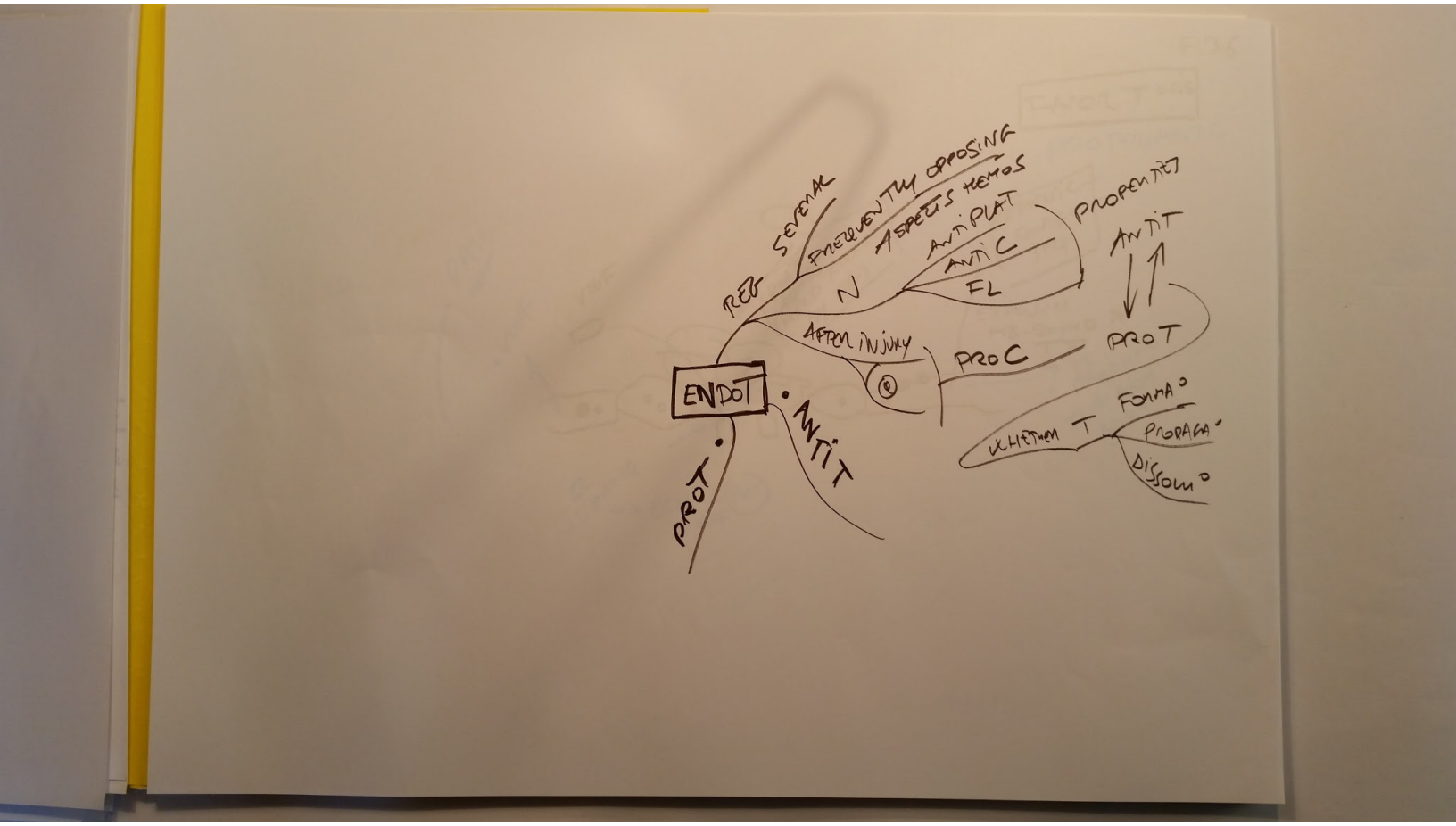

F 4.7
X → Xa
EXTRINSIC
PT → T
II IIa
SPE PL
PROTEOLYTIC
ACTIVE CF
(ENZYME)
INACTIVE CF
(SUBSTRATE)
PLAT
PL SPE)
TF
COF Va

F.G.4

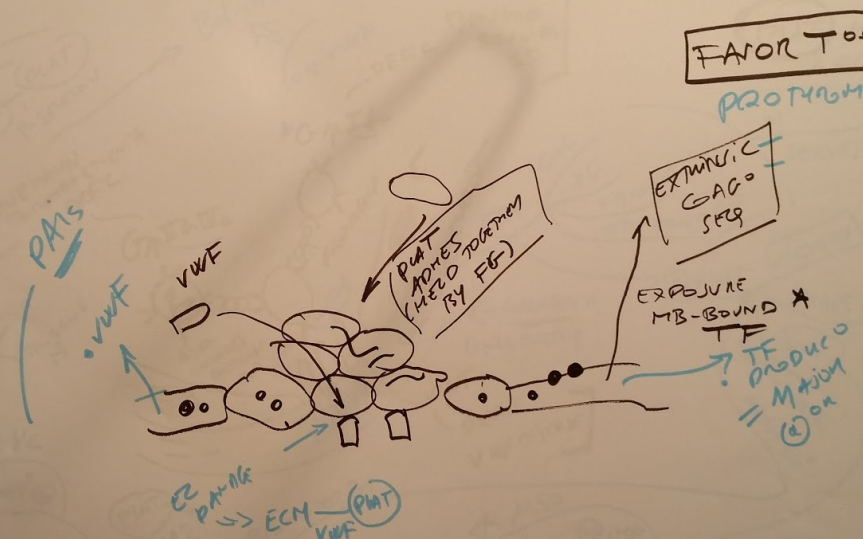
N HEMOSTASIS

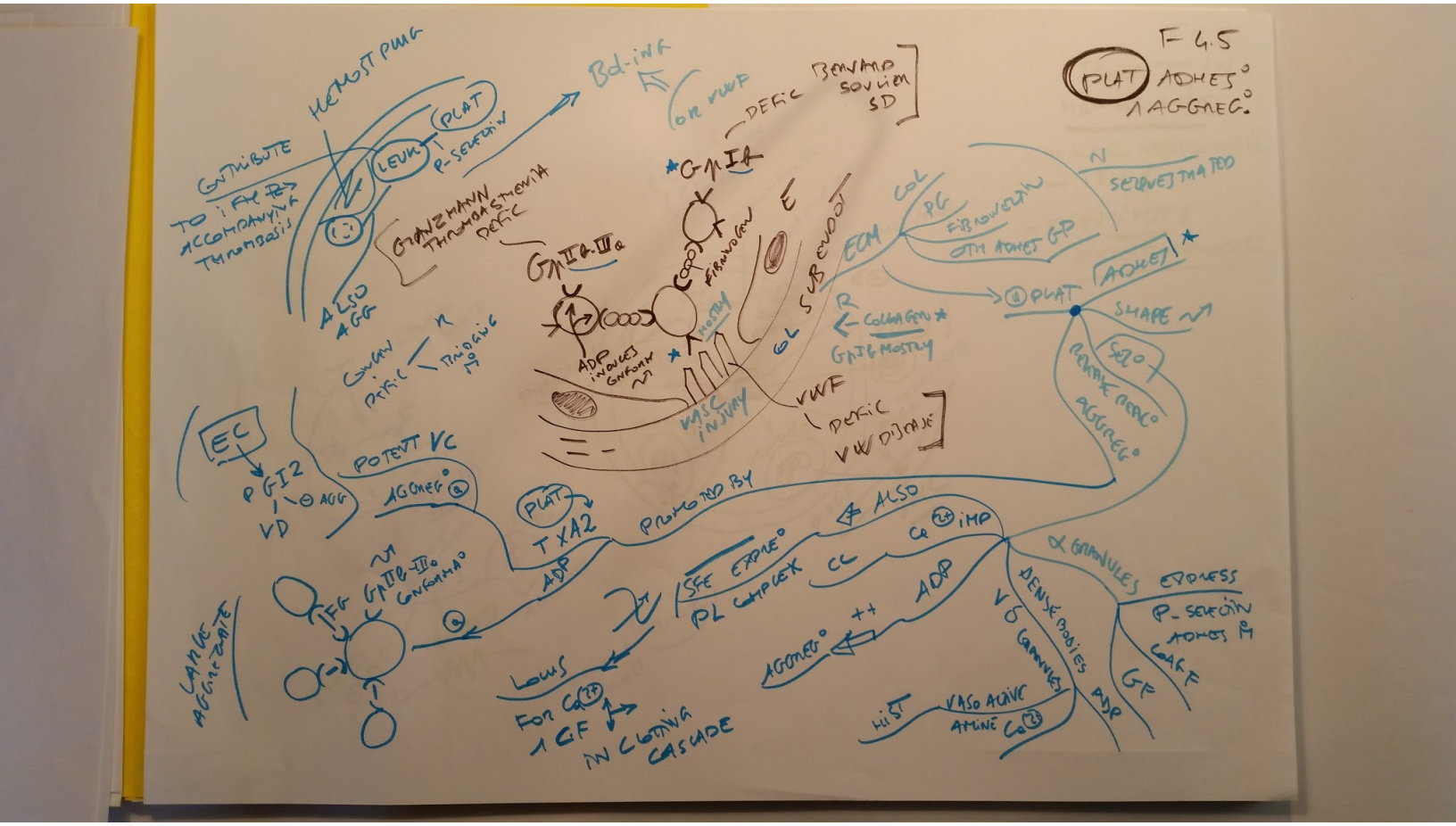



HEMOSTASIS
AFTER INJURY
HEMOSTATIC RESPONSE
REFLEX NEUROGENIC
ANTERIOLAR VC
BY ENDOTHELIN
ENDOT DERIVED
ADH
PLAT
EXPOSED SUBENDOT ECM
CHANGE
SECRETORY GRANULE RELEASE
REC
t-PA
GUNDOM REC MREC
COAG BY CASC
EC
RELEASE
TF
THROMBOPLASTIN
III
TEMPORARY HEMOST PLUG
HEMOST
FG
FIBRINOGEN
MP BOUND LIPID PROCOAGULANT
ADDITIONAL PLAT REC
ALSO INVOLVED
PLAT AGGRE
GRANULE RELEASE
POWERFUL


ENDOT
REG
SEVERAL
FREQUENTLY OPPOSING
ASPECTS THEMOS
N
ANTI PLAT
ANTI C
EL
PROPENSITY
ANTIT
PROT
AFFOR INJURY
PROC
ADHESION T
FORMA°
PROPAGA°
DISSOLU°
PROT°
ANTIT

F.3.6

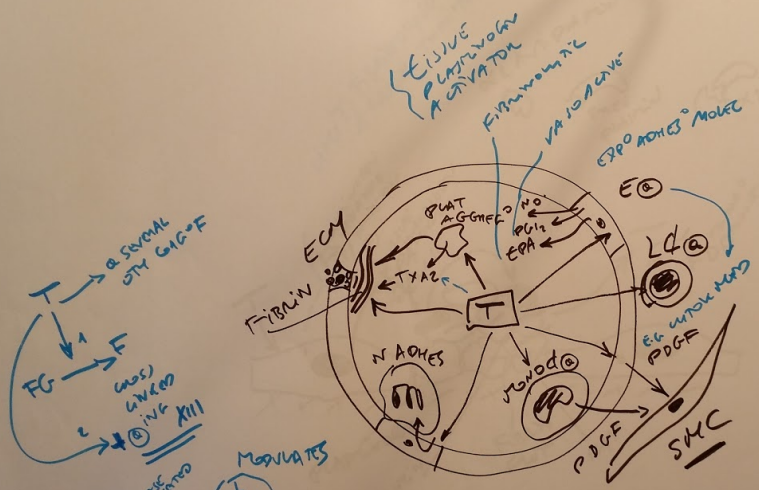

FAVORTORS
PROTHROMBOTIC
EXTRINSIC CASCADE
PAIs
VWF
VWF
PHAT (MEDIATED BY FBS)
EXPOSURE MB-BOUND TF
TF PRODUCTION
ECM
VWF
PHAT


F 4.5
PLAT
ADHES°
AGGREG°
HEMOST PLUG
CONTRIBUTE
TO INFL
ACCOMPANYING
THROMBOSIS
PLAT
LEUK
P-SELECTIN
ALSO AGG
Bd-INF
OR PULP
DEFIC
BERNARD SOULIER SD
GLANZMANN THROMBASTHENIA DEFIC
GPIIa-IIIa
GPIA
FIBRINOGEN
ADP INDUCED CONFORM
MOSTLY
GL
SUBENDOT
ECM
COL
PG
FIBRONECTIN
OTH ADHES GP
N
SEQUEST THROMBO
ADHES
PLAT
SHAPE
SEQ
DENSE GRANULE AGGREG°
R
COLLAGEN
GPIA MOSTLY
VASC INJURY
VWF
DEFIC
VWD DISEASE
EC
PGI2
VD
AGG
POTENT VC
AGGREG°
PLAT
TXA2
ADP
PROMOTED BY
ALSO
Ca
IMP
GPIIb-IIIa
CONFORMAT°
FG
LARGE AGGREGATE
SFE EXPRE°
PL COMPLEX
CL
ADP
AGGREG°
++
LOWS
FOR Ca
1 CF
IN CLOTTING CASCADE
α GRANULES
EXPRESS
P-SELECTIN
ADHES°
VAGF
GF
DENSE GRANULES
V6 GRANULES
ADP
HIST
VASO ACTIVE AMINE
Ca

F4.6

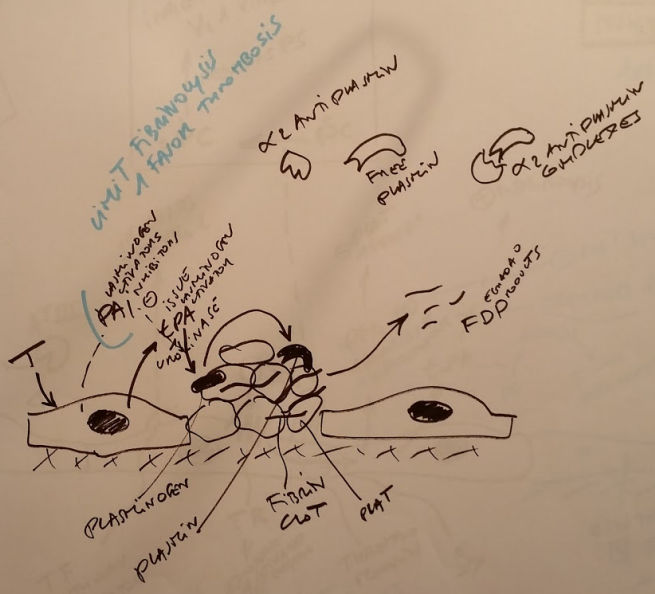
ROLE T IN HEMOSTASIS

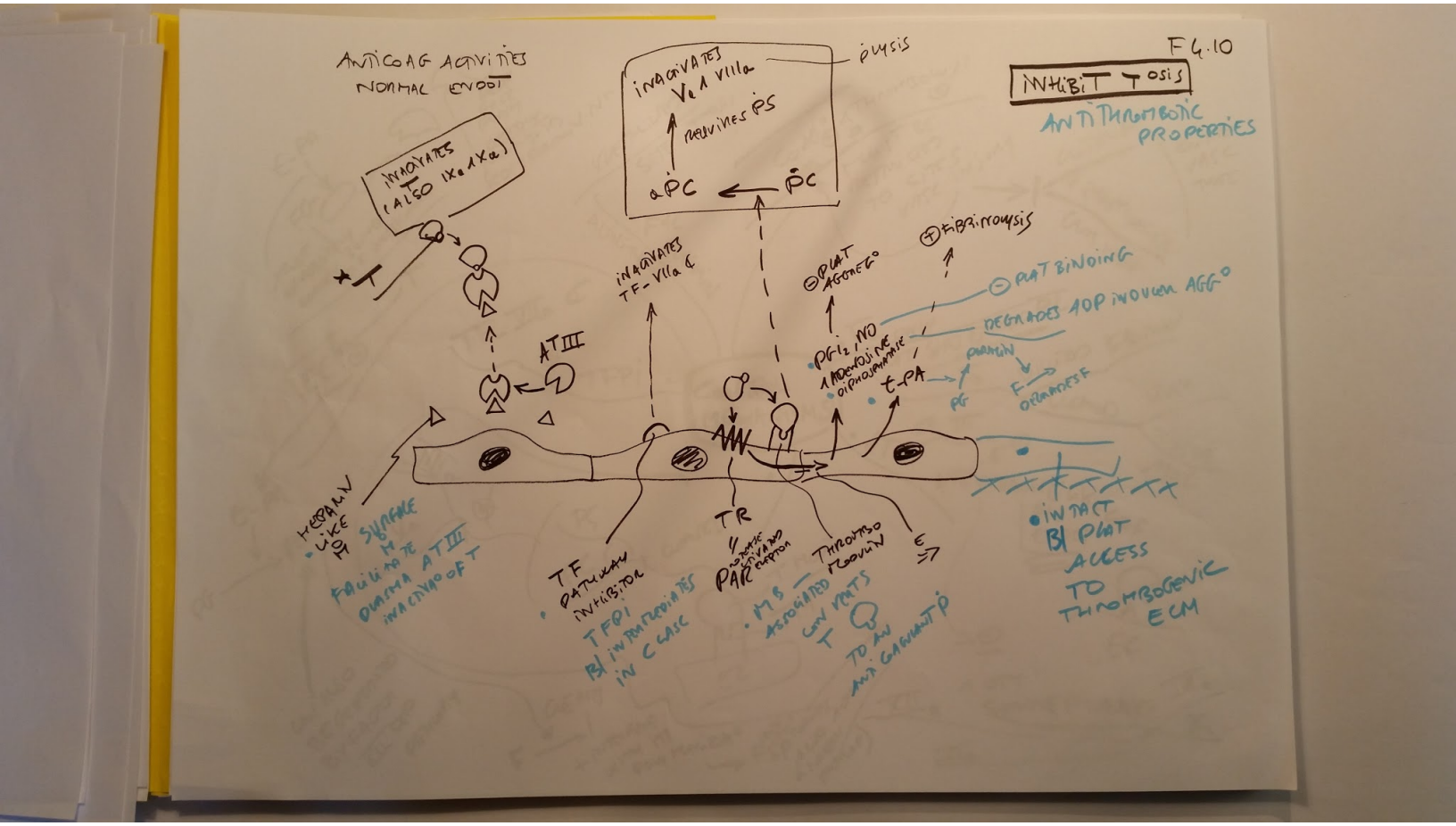
- LOC VESSEL WALL
- iFM ¢
- EVEN LIMITS EXTENT HEMOSTASIS

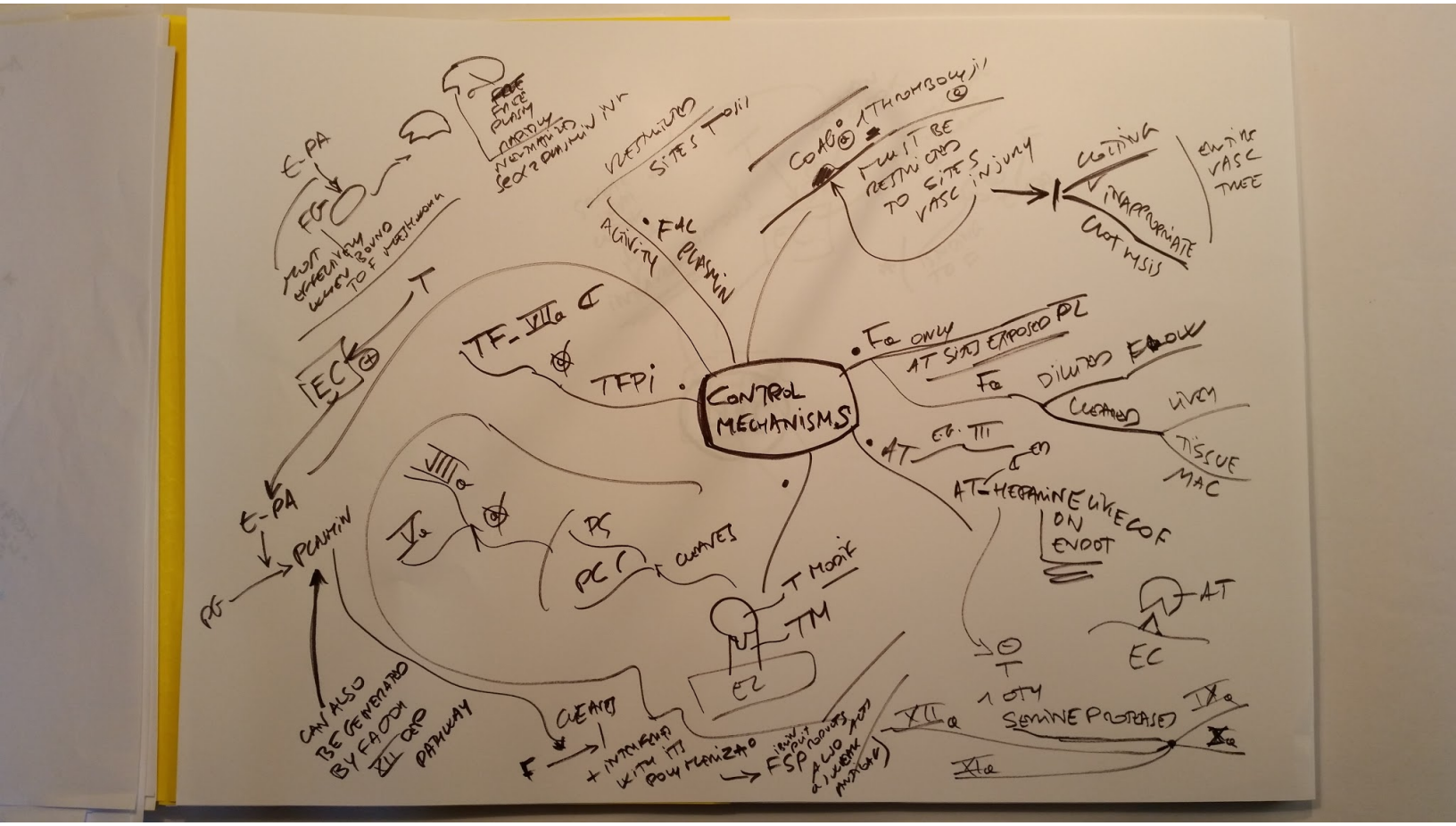


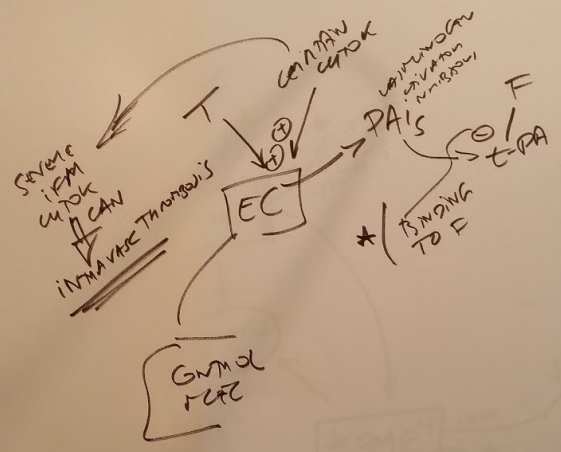
F 4.9

FIBRINOLYTICS



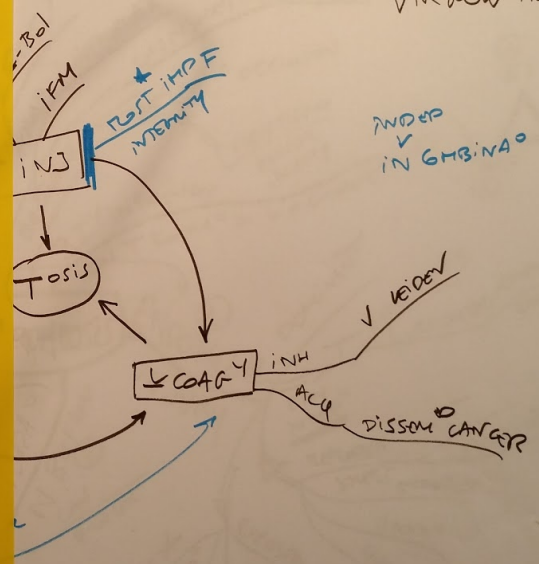

ANTICOAG ACTIVITIES
NORMAL ENDOT
INACTIVATES
Va & VIIIa
PHYSIS
REQUIRES PS
aPC
PC
INACTIVATES
(ALSO IXa & Xa)
T
ATIII
INACTIVATES
TF-VIIa &
⊖PLAT AGGREG°
⊕FIBRINOLYSIS
⊖PLAT BINDING
DEGRADES ADP INDUCED AGG°
PGI2, NO
ADENOSINE
DIPYRIDAMOLE
t-PA
PG
PLASMIN
F. ACTIVATORS F
HEPARIN
LIKE
SULFATE
IN
ENDOTH
ACTIVATES ATIII
INACTIVATES T
TF
PATHWAY
INHIBITOR
TFPI
INTERFERES
IN C CASE
TR
PAR
THROMBIN
ACTIVATED
PLATELETS
CONTACTS
T
TO AN
ANTICOAGULANT
INTACT
BS PLAT
ACCESS
TO
THROMBOGENIC
ECM
INHIBITOSIS
ANTITHROMBOTIC
PROPERTIES
F4.10

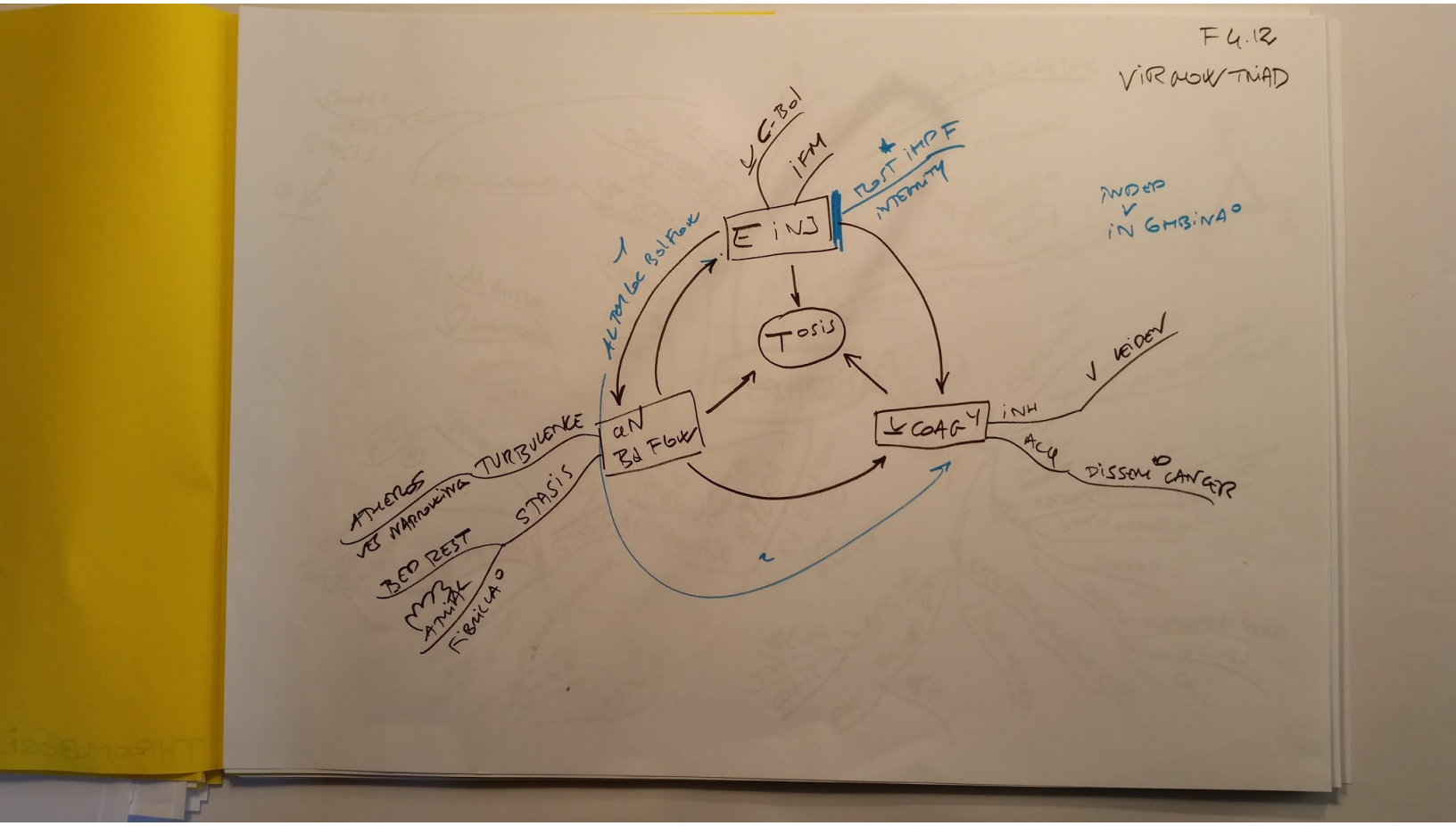

CONTROL MECHANISMS
TF-VIIa
TFPi
Fa ONLY AT SITES EXPOSED PL
Fa DILUTED
CLEARED
LIVER
TISSUE MAC
AT
AT-HEPARIN LIKE COF ON ENDOT
PC
PC
CLEAVES
T MODIF
TM
EC
PLASMIN
CAN ALSO BE GENERATED BY FACTOR XII DEP PATHWAY
CLEAVES
+ INTERACTION WITH ITI POLYANIONS
FSP
SERINE PROTEASES
EC
T
AT
COAG
THROMBOSIS
IT MUST BE RESTRICTED TO SITES VASC INJURY
CLOTTING
INAPPROPRIATE
CLOT LYSIS
ENTIRE VASC TREE
ACTIVITY
FAC PLASMIN
SITES TOO!!
E-PA
FG
T
EC
E-PA
PLASMIN
PG

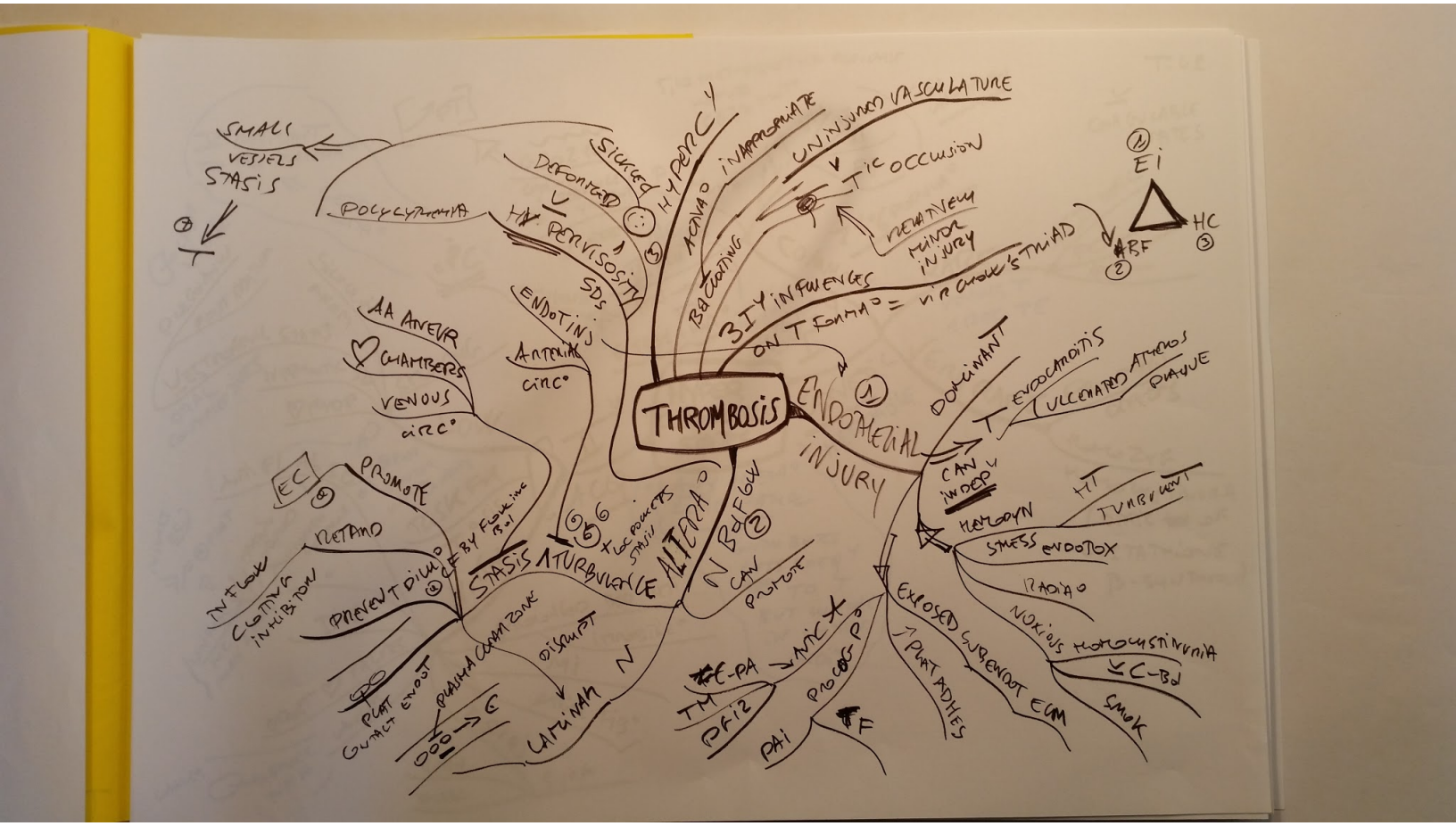

T
EC
PA's
F
t-PA
BINDING TO F

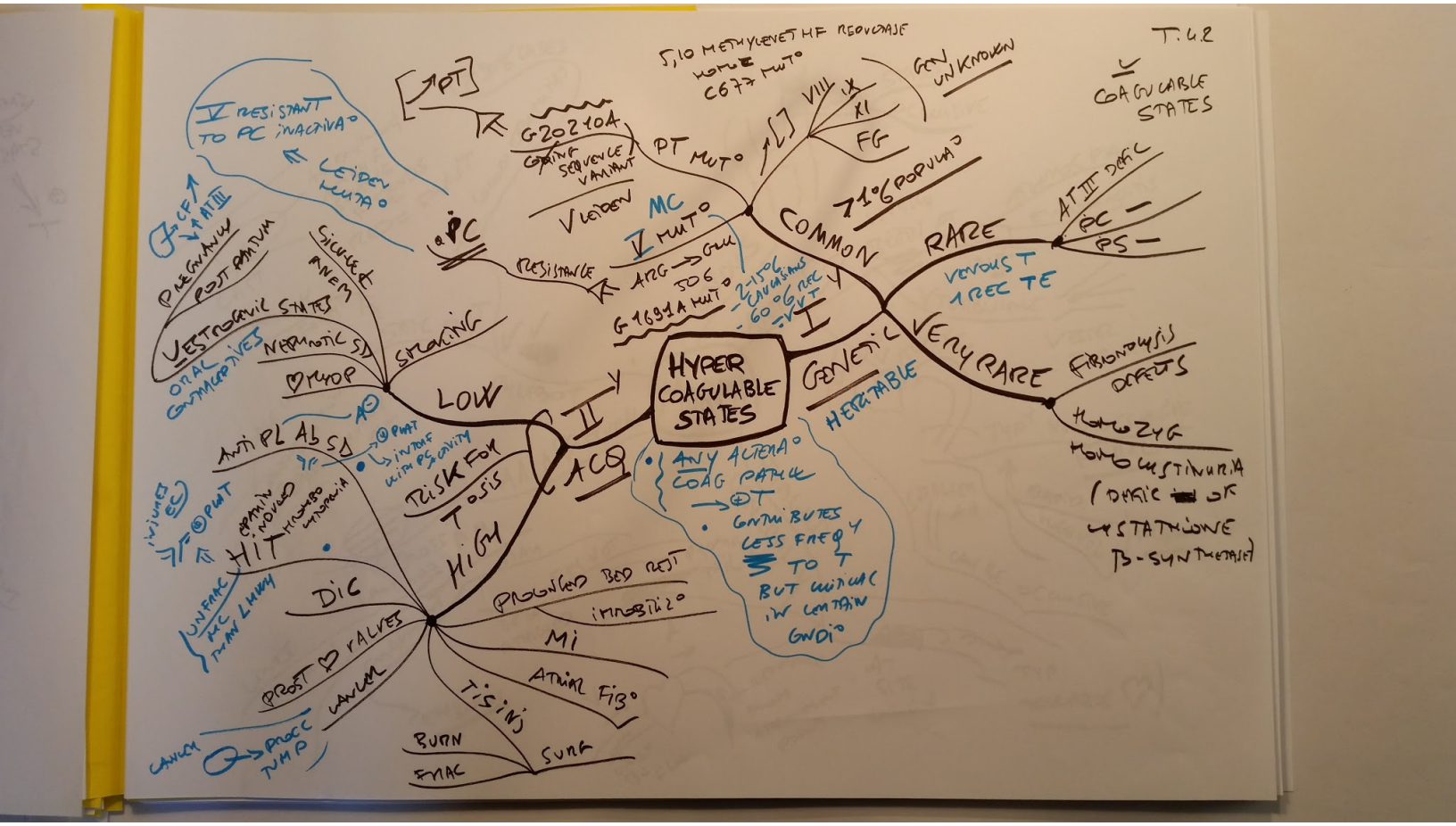
THROMBOSIS

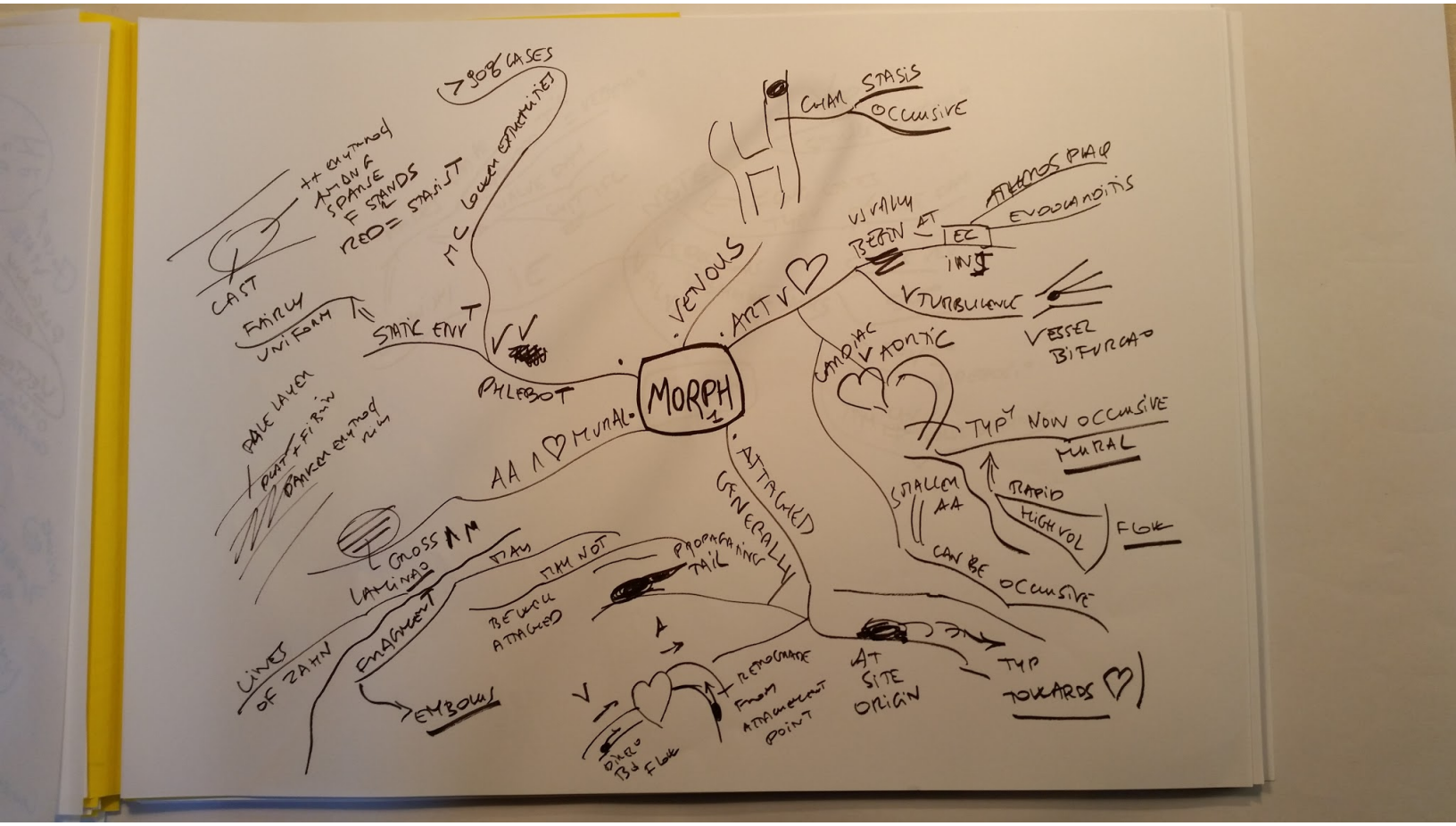
F 4.12
VIRCHOW TRIAD

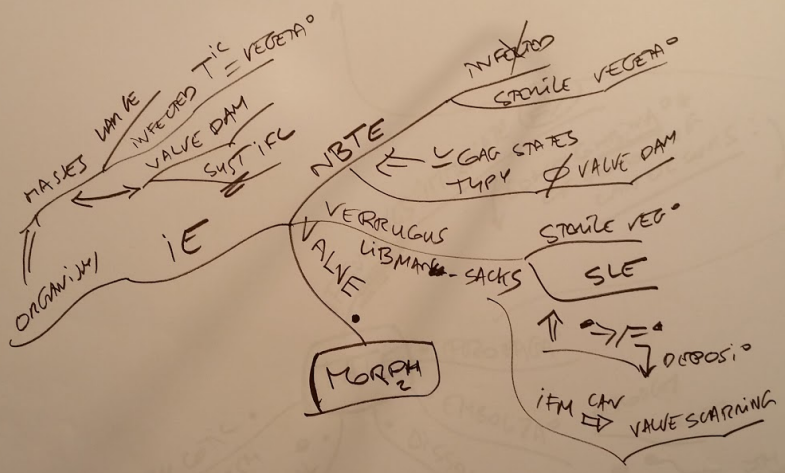


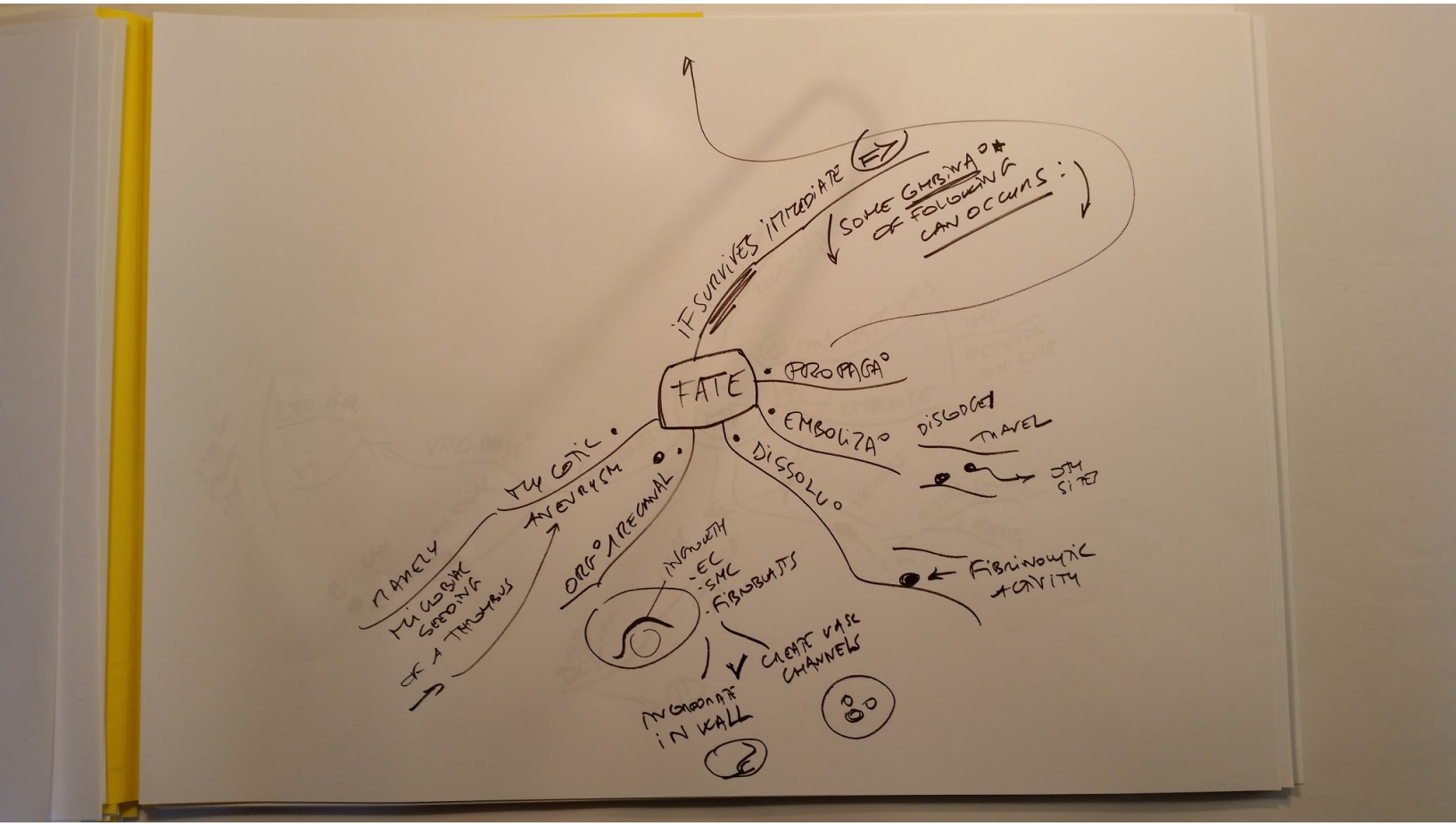

F4.12
VIRCHOW TRIAD
↓ C-Bol
iFM
↑ FIBRIN
↓ PLT iMPF
INTEGRITY
↑ ALTERED BLOOD FLOW
ENDO
↓
iN GMB:NA°
Tosis
↓ COAG-Y
iNH
↓ HEDGY
ACH
DISSOM° CANCER
ATHEROS
VEIN WALL INJURY
TURBULENCE
BED REST
STASIS
AMPUTATION
FIBRILLA°
2

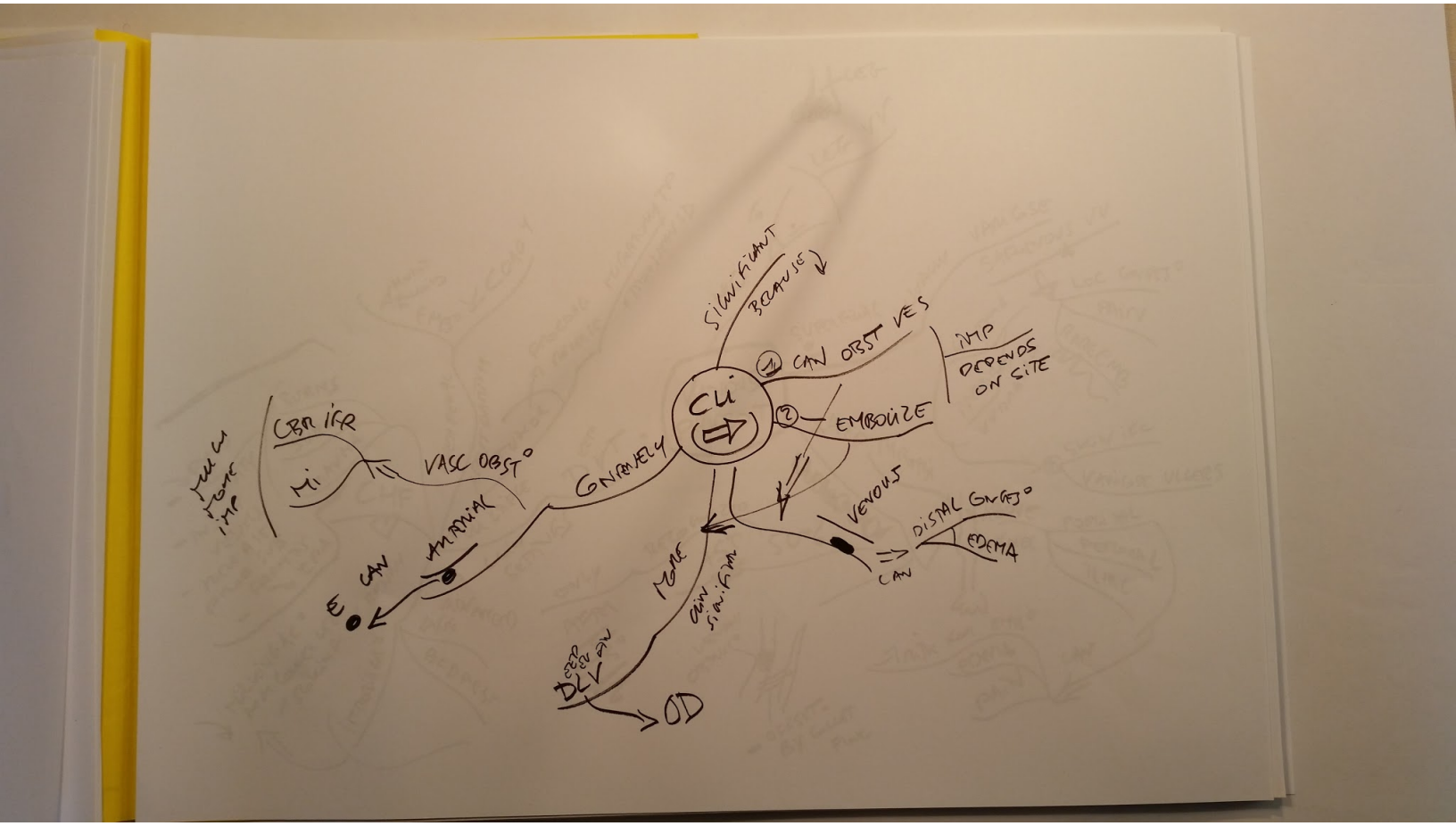

THROMBOSIS
ENDOTHELIAL INJURY
STASIS
ALTERA
SMALL VESSELS STASIS
POLYCYTHAEMIA
Sickled
Dehydrated
HYPERVISCOSITY
SDS
ENDOTOXIN
ANTIPHOS circ°
AA ATRIUM
CHAMBERS
VENOUS circ°
ACTIVATED
BALLOONING
UNINJURED VASCULATURE
TIC OCCLUSION
RELATIVELY MINOR INJURY
3 I'Y INFLUENCES ON THROMBOSIS = VIRCHOW'S TRIAD
Ei
HC
ARF
DOMINANT
ENDOCARDITIS
ULCERATED ATHEROS PLAQUE
CAN INDEP
HAEMOLYN
HT
TURBULENT
STRESS ENDOTOX
RADIATION
NOXIOUS HAEMOSTASIS/INFLAMMA
C-Rd
SMOK
EXPOSED SUBENDOT ECM
PLATADHES
TF
PAI
TM
PF-12
ANTIC
EC
PROMOTE
PLETAMO
INFLUX
CONTRA INHIBITION
PREVENT DILUTE
CF BY FLOW
STASIS TURBULENCE
DISRUPT
CATIONS
PLASMA COAGULATION
PLAT CONTACT ENDOT
CAN PROTOTE
IN BLOOD FLOW

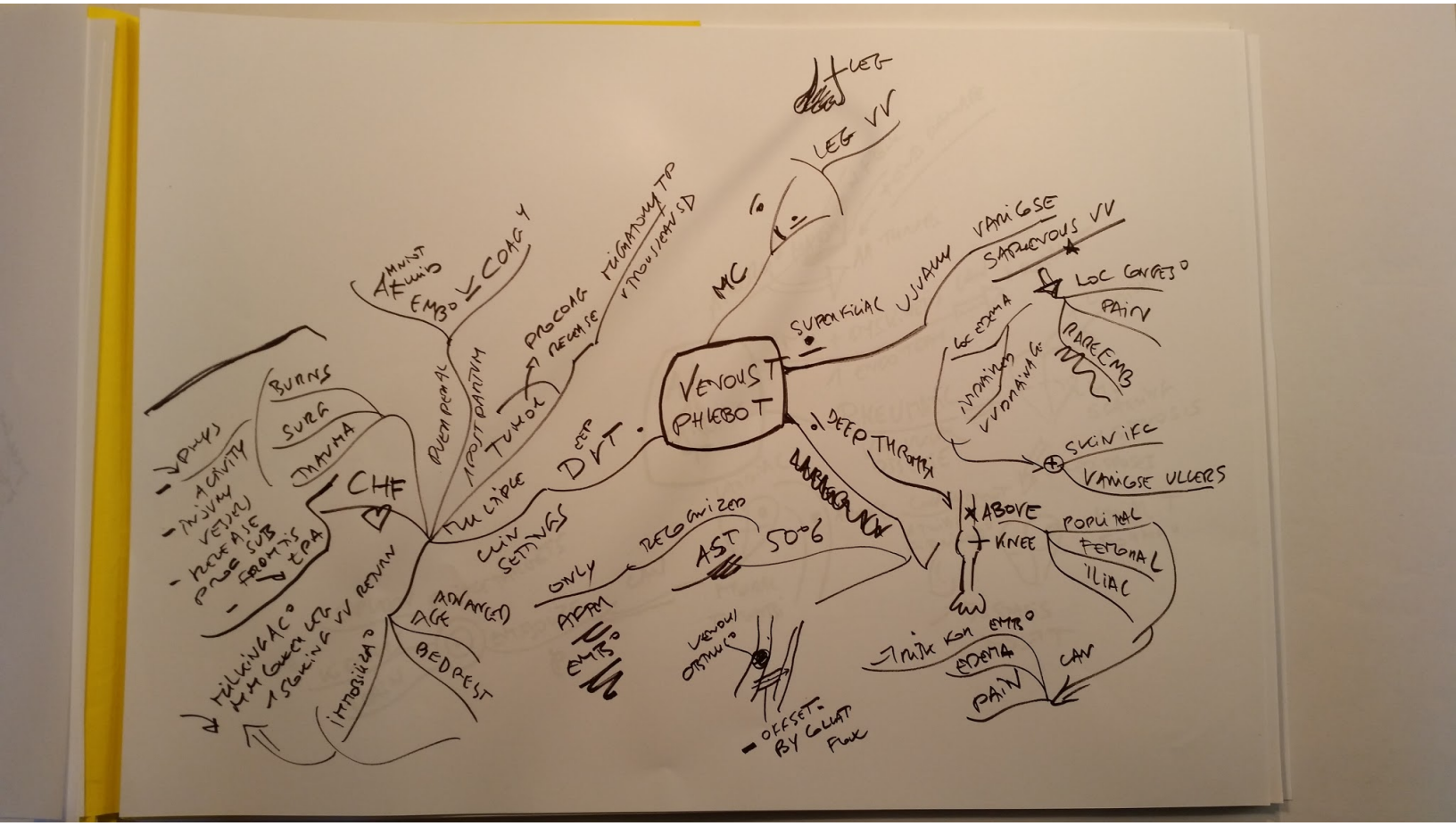

HYPER COAGULABLE STATES
GENETIC
HERITABLE
COMMON
71% POPULA°
PT MUT°
G20210A
GOING SEQUENCE VARIANT
5,10 METHYLENETHF REDUCTASE HOMOZ C677 MUT°
VIII
IX
XI
FG
GEN UNKNOWN
V LEIDEN
MC MUT°
ARG → GLN 506 °
G-1691A MUT°
2-15% UNIVERSAL
60% REC VT
RARE
VENOUS T REC TE
ATIII DEFIC
PC —
PS —
VERY RARE
FIBRINOLYSIS DEFECTS
HOMOZYG HOMOCYSTINURIA (DEFIC OF CYSTATHIONINE B-SYNTHETASE)
T. 6.2
COAGULABLE STATES
ACQ
ANY ALTERA° COAG PATHW
→ DT
CONTRIBUTES LESS FREQ Y TO T BUT CLINICAL IM CONTAIN GNDi°
LOW
RISK FOR T°SIS
HIGH
SMOKING
SEDENT
MYOP
NEPHROTIC SY
VESTIGIAL STATES
ORAL CONTRACEPTIVES
PREGNANCY
POST PARTUM
CP
ATIII
V RESISTANT TO PC INACTIVA°
LEIDEN MUTA°
PC
RESISTANCE
ANTI PL AB SD
PHAT WITH PC CAVITY
PHAT
HIT
HEPARIN INDUCED THROMBOCYTOPENIA
UNFRAC
LMWH
DIC
PROST VALVES
CANCER
PROC TMP
PROLONGED BOD REST
IMMOBILIZ°
MI
ATRIAL FIB°
TISSINJ
BURN
FRAC
SURG

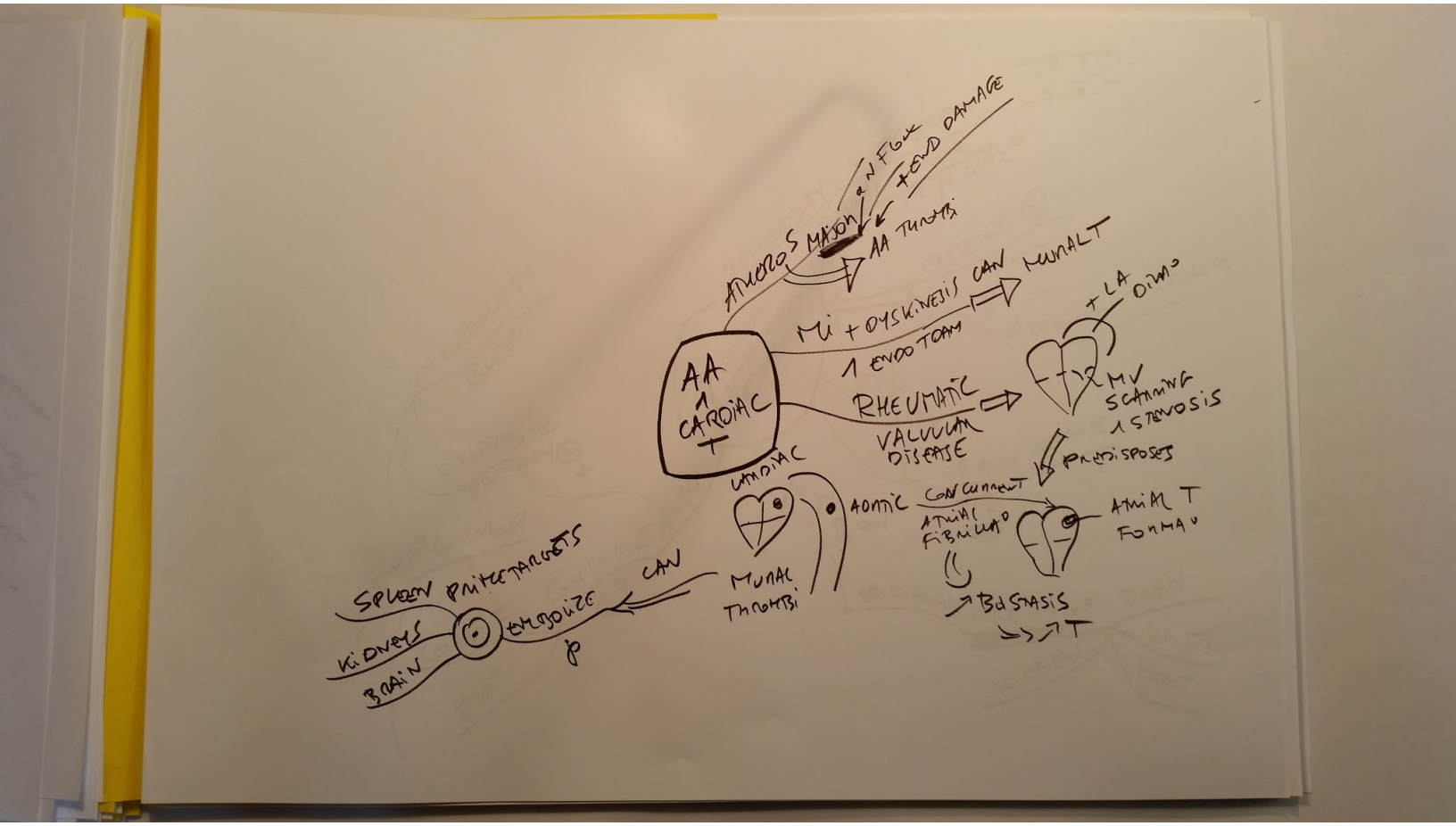

MORPH
1
VENOUS
ART
MURAL
ATTACHED
GENERALLY
PHLEBOT
STATIC ENVT
FAIRLY UNIFORM
CAST
MC
STASIS
OCCLUSIVE
VIRALLY
BEGIN AT
EC
INJ
ENDOCARDITIS
TURBULENCE
VESSEL BIFURCAT
CARDIAC
ATOMIC
TYP NON OCCLUSIVE
MURAL
SMALLER AA
RAPID
HIGH VOL
FLOW
CAN BE OCCLUSIVE
AA
GROSS
LAMINAE
LINES OF ZAHN
ENGAGEMENT
EMBOLI
PROPAGATING TAIL
AT SITE ORIGIN
TYP TOWARDS
RETROGRADE FROM ATTACHMENT POINT
DIRD BY FLOW

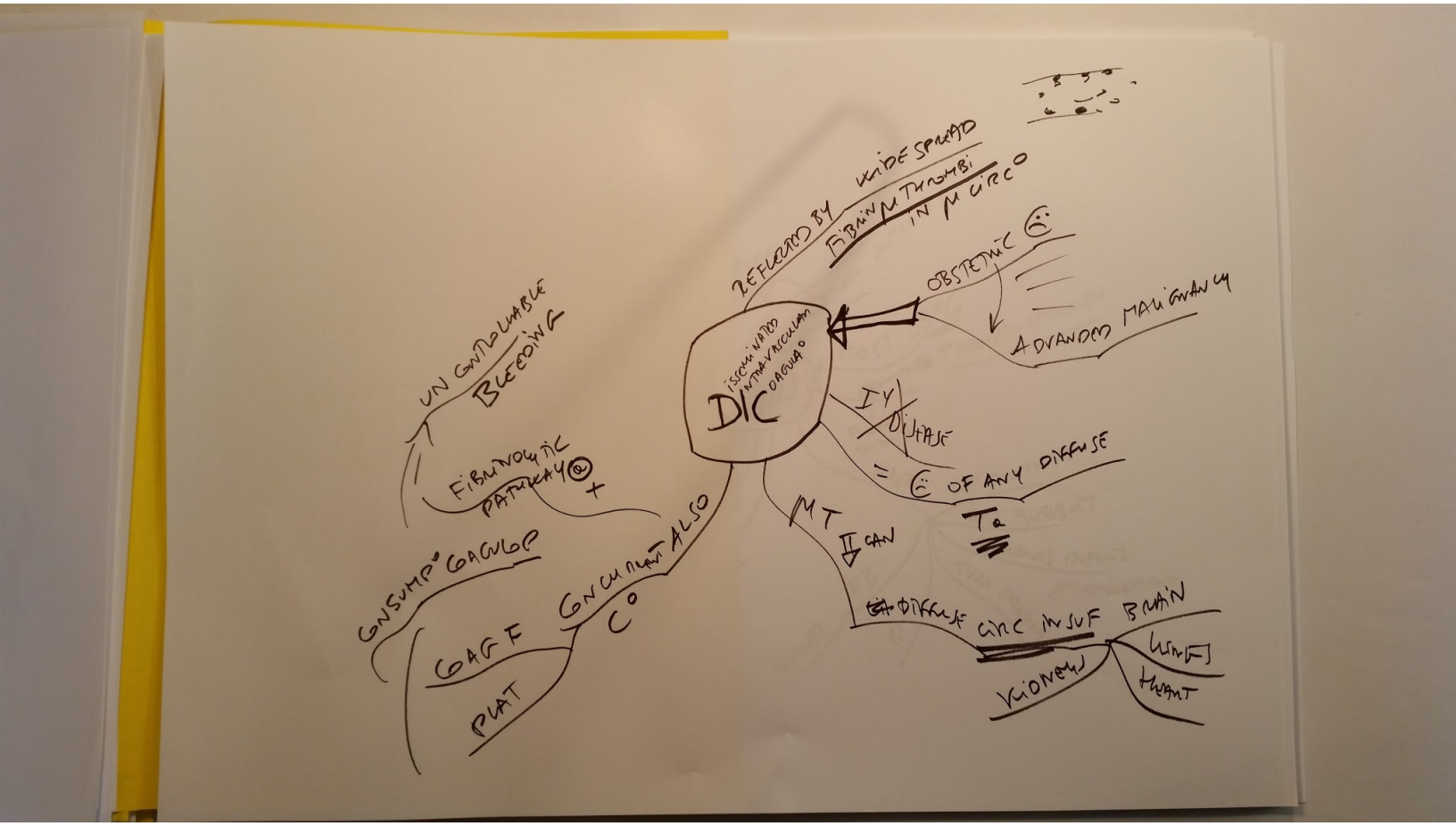

MASSES
INFECTED
VALVE DAM
SYST'IFC
ORGANISM
IE
NBTE
INFECTED
STERILE VEGETA°
GAG STATES
TYPY
VALVE DAM
VERRUCOUS
VALVE
LIBMAN-SACKS
STERILE VEG°
SLE
MORPH
IFM CAN
VALVE SCARRING

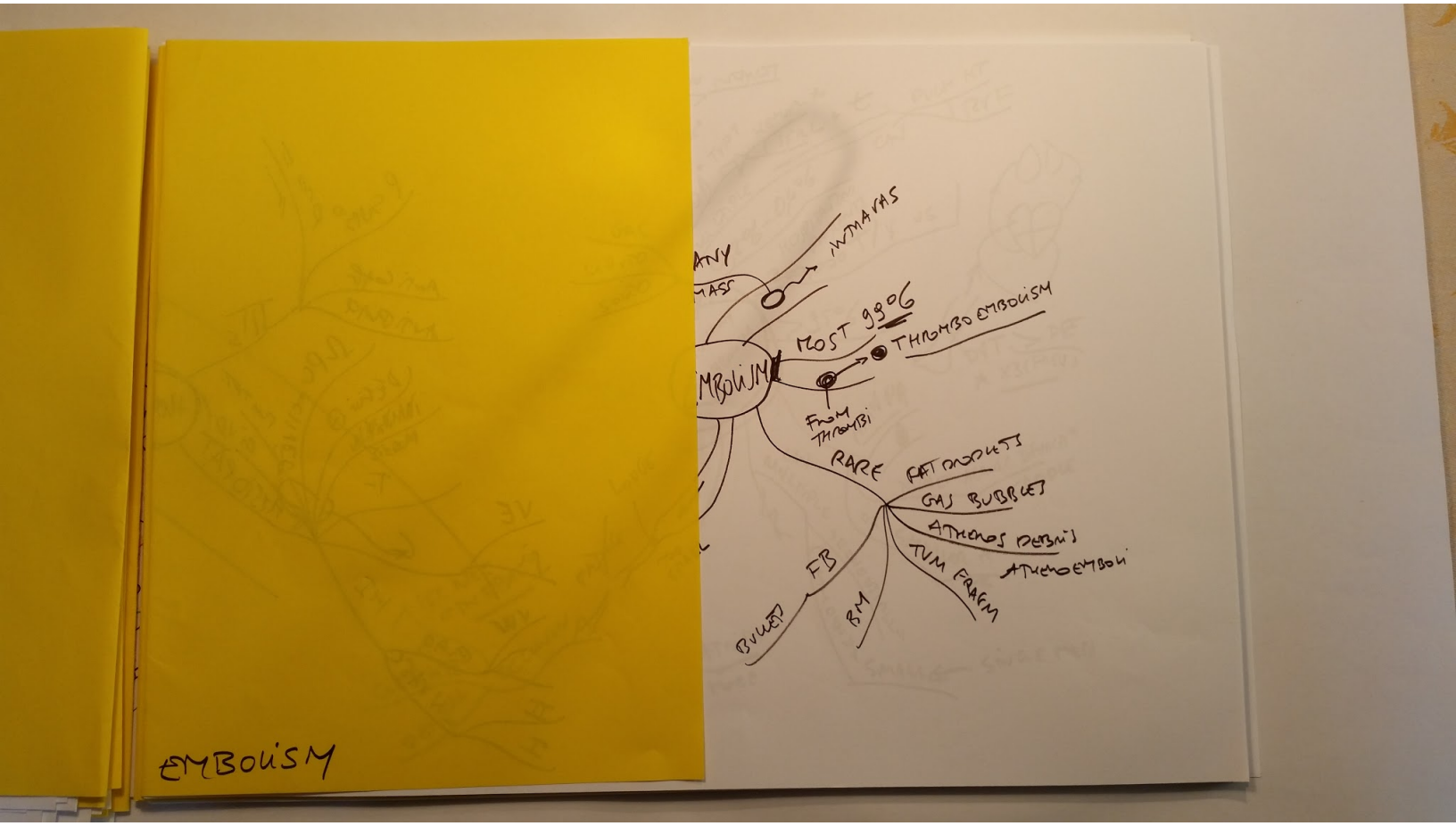

IF SURVIVES IMMEDIATE (E)
SOME COMBINATION OF FOLLOWING CAN OCCURS:
FATE
PROPAGATION
EMBOLIZATION
DISSOLUTION
DISLODGED TRAVEL
ATHEROSCLEROTIC ANEURYSM
ORGANIZED THROMBUS
NAMELY
INTIMA
-EC
-SMC
-FIBROBLASTS
FIBRINOLYTIC ACTIVITY
CLEAR VASC CHANNELS
INCORPORATE IN WALL

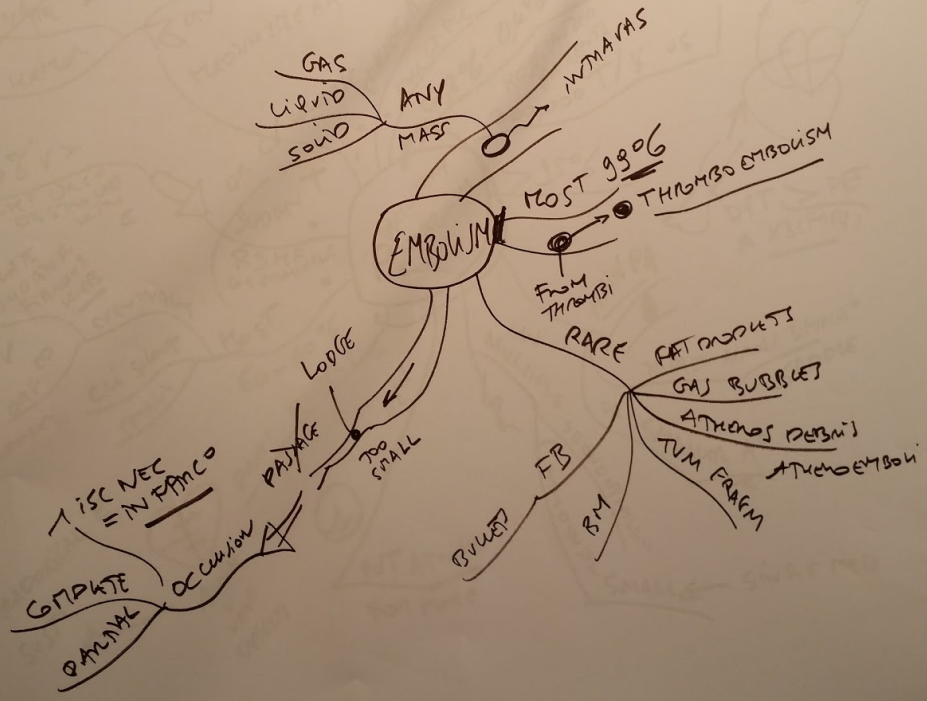

SIGNIFICANT
BECAUSE
CLI
CAN OBST VES
EMBOLIZE
IMP
DEPENDS
ON SITE
GNRMLY
VASC OBST°
VENOUS
DISTAL GNGJ°
EDEMA
CAN
CAN
DLV
OD

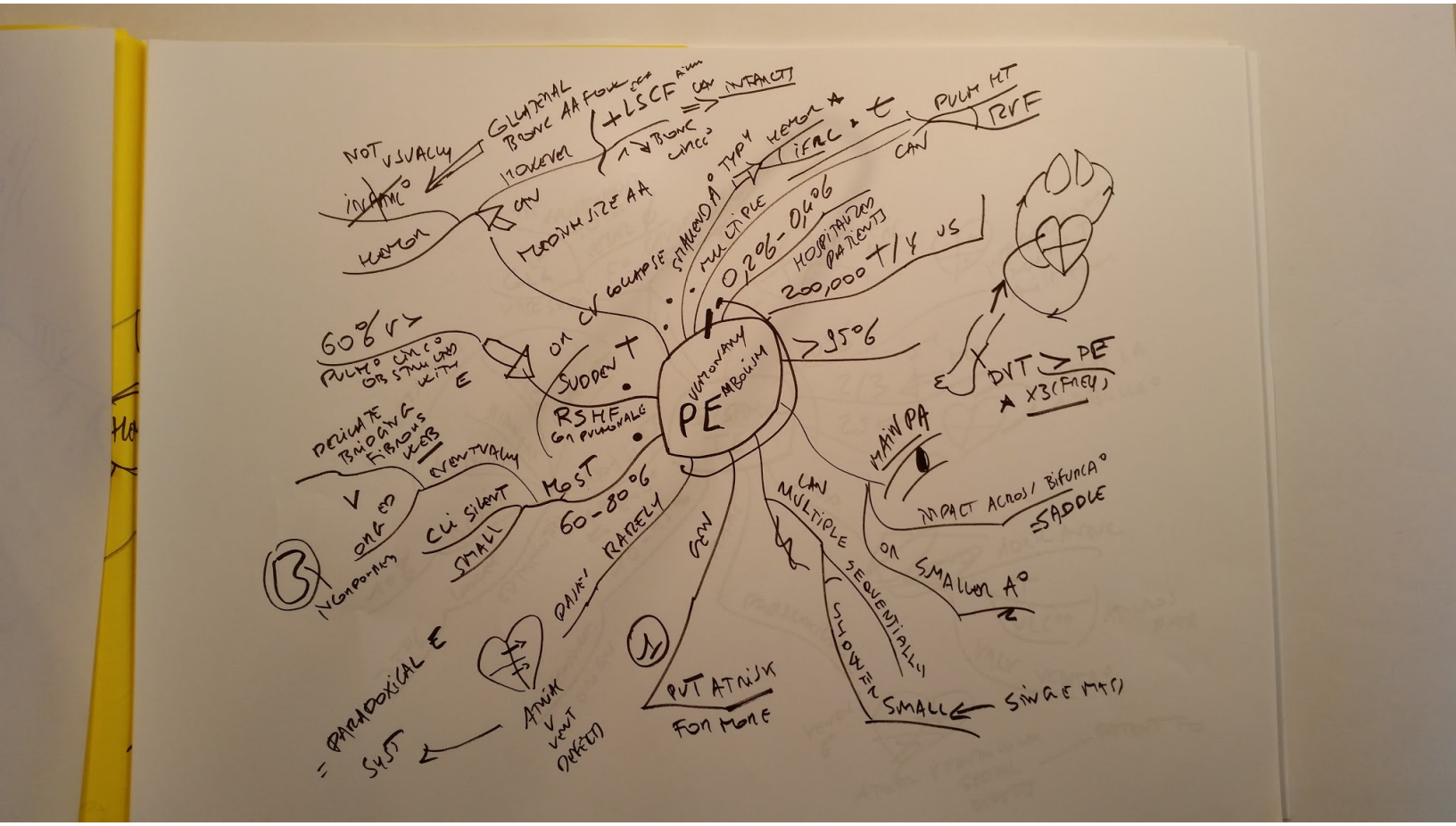

VENOUS PHLEBOT
DVT
DEEP
MC
LEG
LEG VV
SUPERFICIAL
VARICOSE
SAPHENOUS VV
LOC COMPES
PAIN
RARE EMB
SKIN IFC
VARICOSE ULCERS
DEEP THROMB
ABOVE
KNEE
POPLITEAL
FEMORAL
ILIAC
CAN
PAIN
EDEMA
CHF
BURNS
SURG
TRAUMA
ACTIVITY
TUMOUR
POST PARTUM
PULM EMB
ADVANCED AGE
BEDREST
IMMOBILIZED
CLIN SETTINGS
ONLY
AST
50%
ADEM
VENOUS OBSTRUCT
OFFSET BY CLOT FLOW

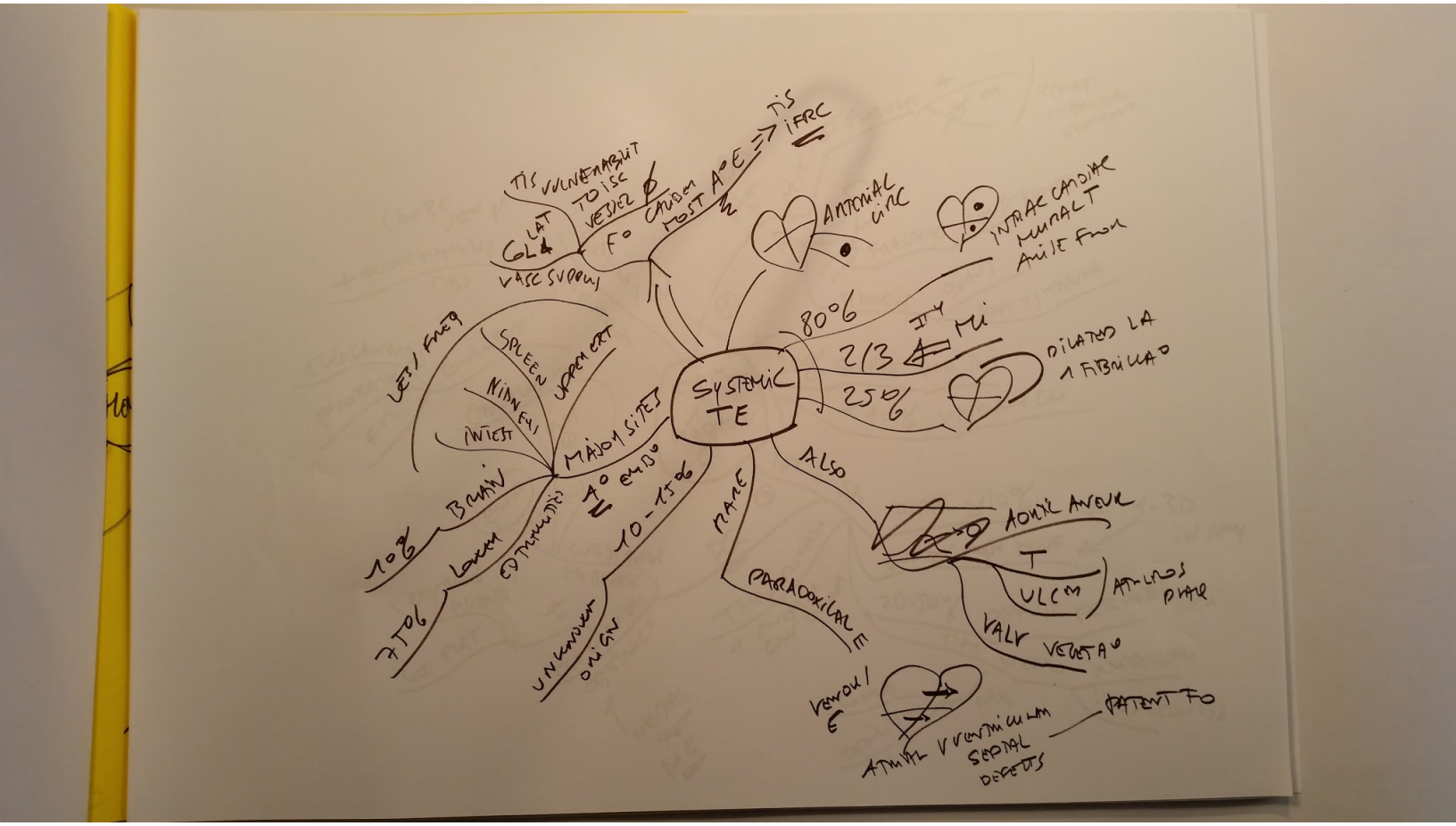

ATHEROSCLEROSIS
+ ENDOTHELIAL DAMAGE
AA THROMBI
AA
↑
CARDIAC
↑
MI + DYSKINESIS CAN MURAL T
↑ ENDOCARDIAL
RHEUMATIC
VALVULAR
DISEASE
+ LA DILAT°
MV
SCARRING
↑ STENOSIS
PREDISPOSES
CARDIAC
AORTIC
CAN CONCURRENT
ATRIAL
FIBRILLAT°
ATRIAL T
FORMAT°
STASIS
↑ T
MURAL
THROMBI
CAN
EMBOLIZE
SPLEEN
KIDNEYS
BRAIN
PERIPHERAL TARGETS
↓

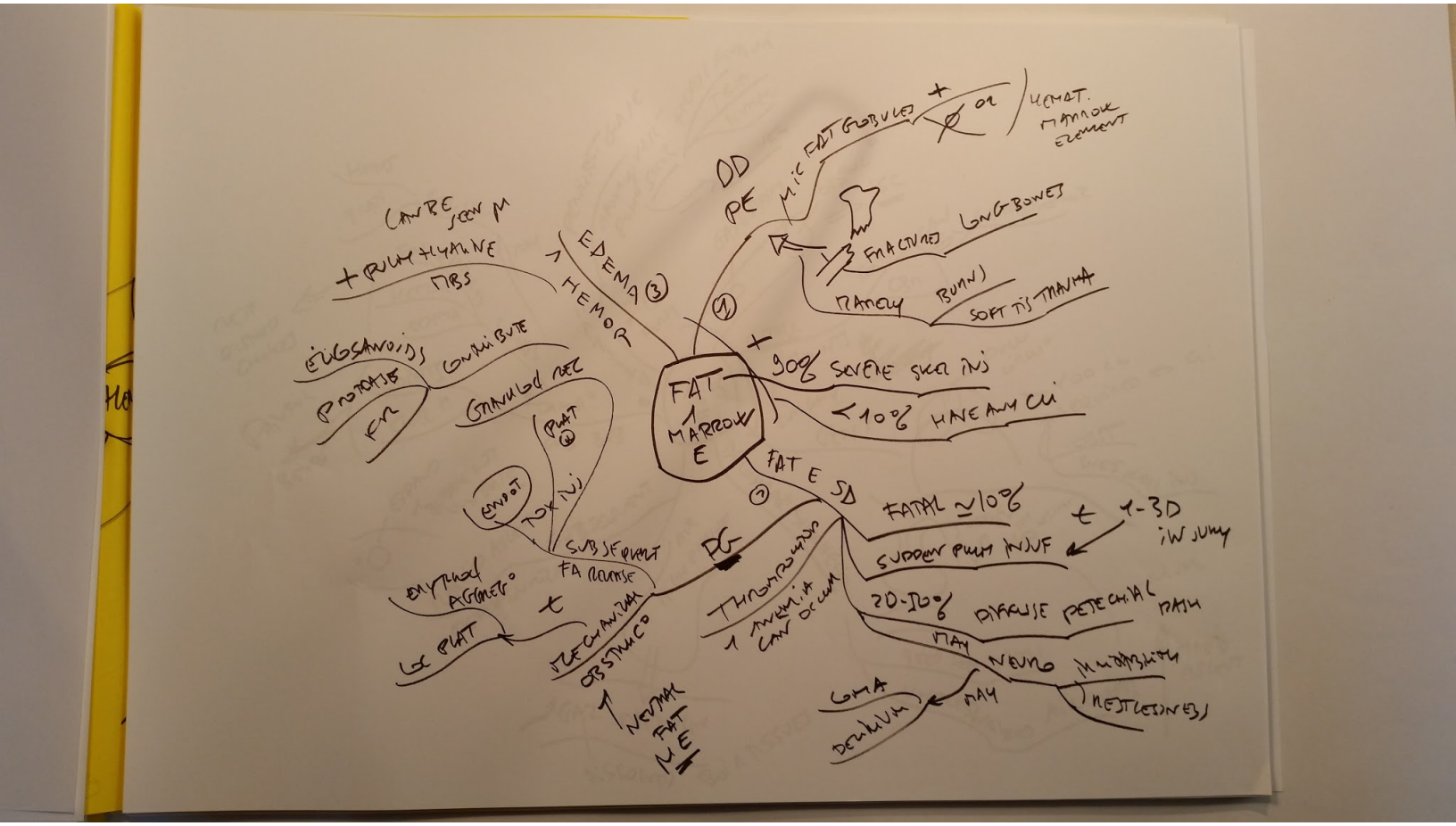

DIC
UNCONTROLLABLE BLEEDING
FIBRINOLYTIC PATTERN
CONSUMPTIVE COAGULOP
COAGF
PLAT
CONSUMPT ALSO
REFLECTED BY WIDESPREAD
BRAIN THROMBI IN MICRO
OBSTETRIC
A RANDOM MALIGNANCY
IV
OF ANY DIFFUSE
MT BRAIN
DIFFUSE CIRC INSUF BRAIN
VISION

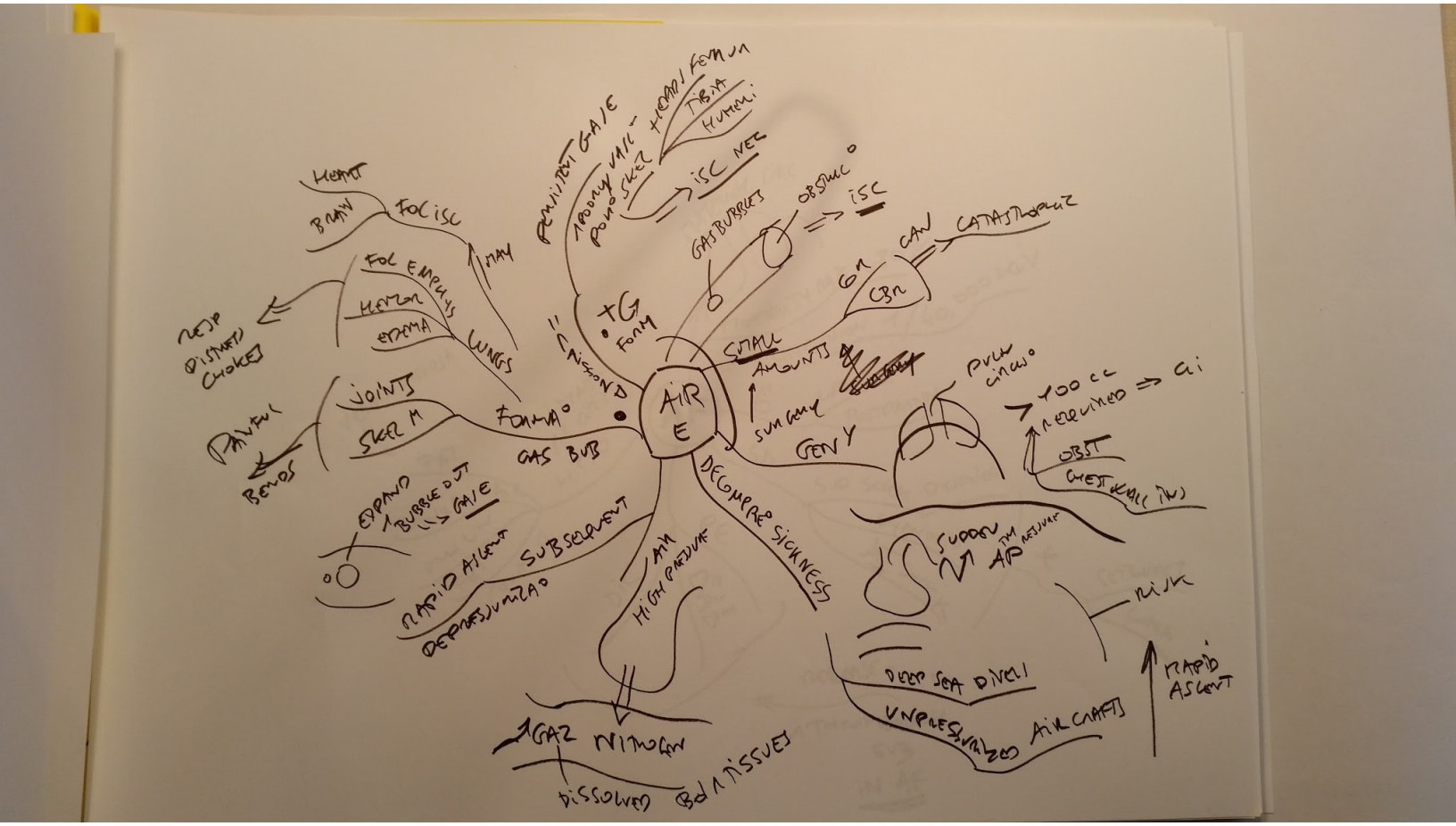

EMBOLISM
MOST 90%
THROMBO EMBOLISM
FROM THROMBI
RARE
FAT DROPLETS
GAS BUBBLES
ATHEROS DEBRIS
TUM FRAGM
ATHEROEMBOLI
FB
BM

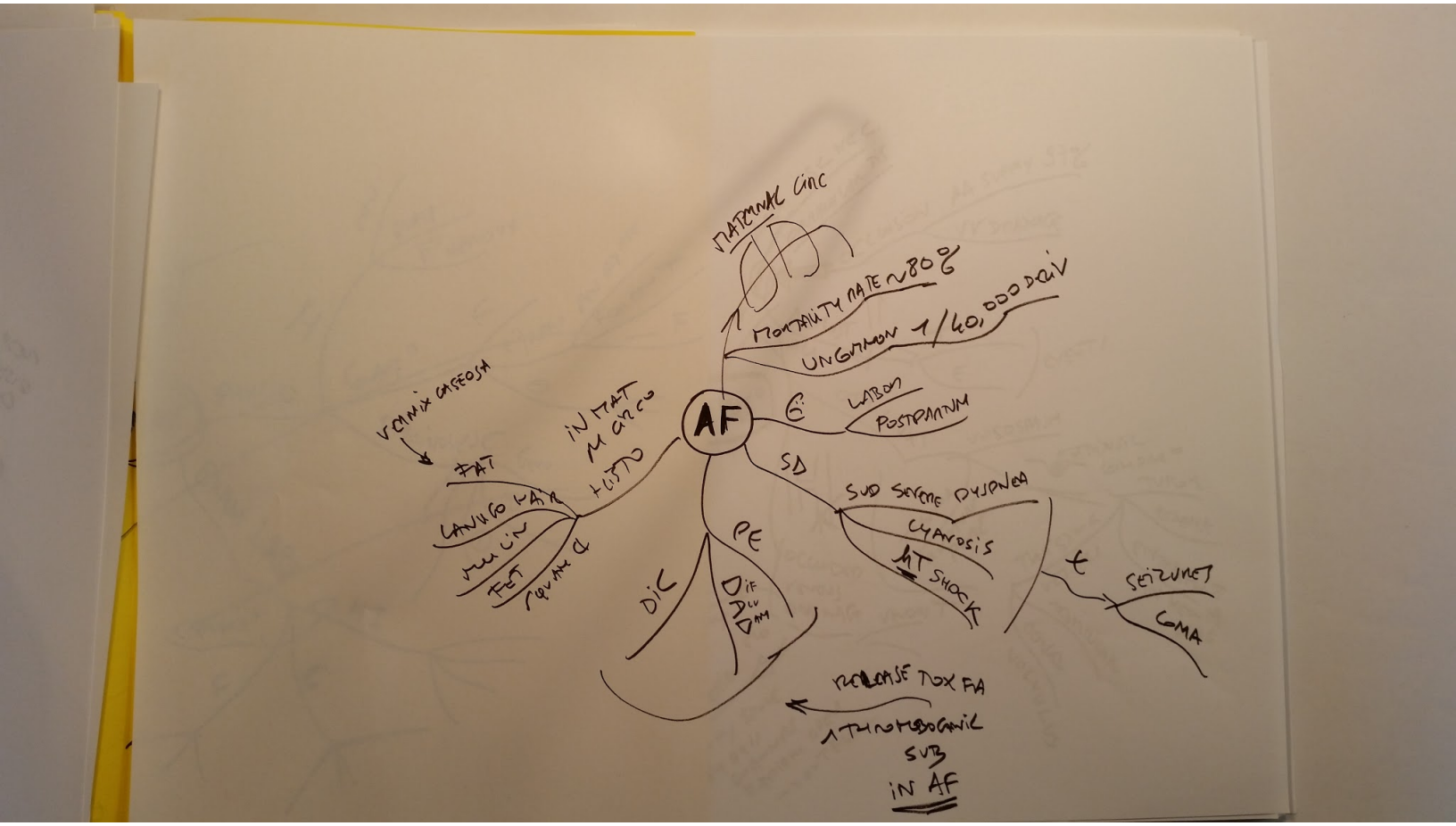

GAS
LIQUID
SOLID
ANY
MASS
INTRAVAS
EMBOLISM
MOST 99%
THROMBOEMBOLISM
FROM THROMBI
RARE
FAT DROPLETS
GAS BUBBLES
ATHEROS DEBRIS
ATHEROEMBOLI
TUM FRAGM
BM
FB
BULLET
LODGE
PASSAGE
TOO SMALL
OCCLUSION
COMPLETE
PARTIAL
ISCHEC
=INFARCT


PE
VENOUS EMBOLISM
NOT VISUALLY
GLUTEAL
BRANCH AA
HOWEVER
+LSCF
CAN INFARCT
PULM HT
RVF
CAN
MEDIUM SIZE AA
TYPY HEMOP
IFRC
CAN
SYMPTOMD
MULTIPLE
0,2%-0,4%
HOSPITALISED PATIENTS
200,000 T/Y US
>95%
60% VS
PULM CANCER OR SMALL WITH E
ON CT
SUDDEN
RS HF
ON PULSOXIMETRY
DVT > PE
X3 (FAMILY)
DELICATE BRIDGING FIBROUS WEBS
EVENTUALLY
V
ONE ED
INCIDENTAL
CLI SILENT
SMALL
MOST
60-80%
RARELY
MAIN PA
IMPACT ACROSS BIFURCA°
=SADDLE
CAN
MULTIPLE
SEQUENTIALLY
SLOWLY
SMALL
SINGLE MAS
ON SMALLER A°
GEN
PVT ATRIUM
FOR MORE
PARADOXICAL E
=
SVT
ATRIAL
VENT
DEFECTS

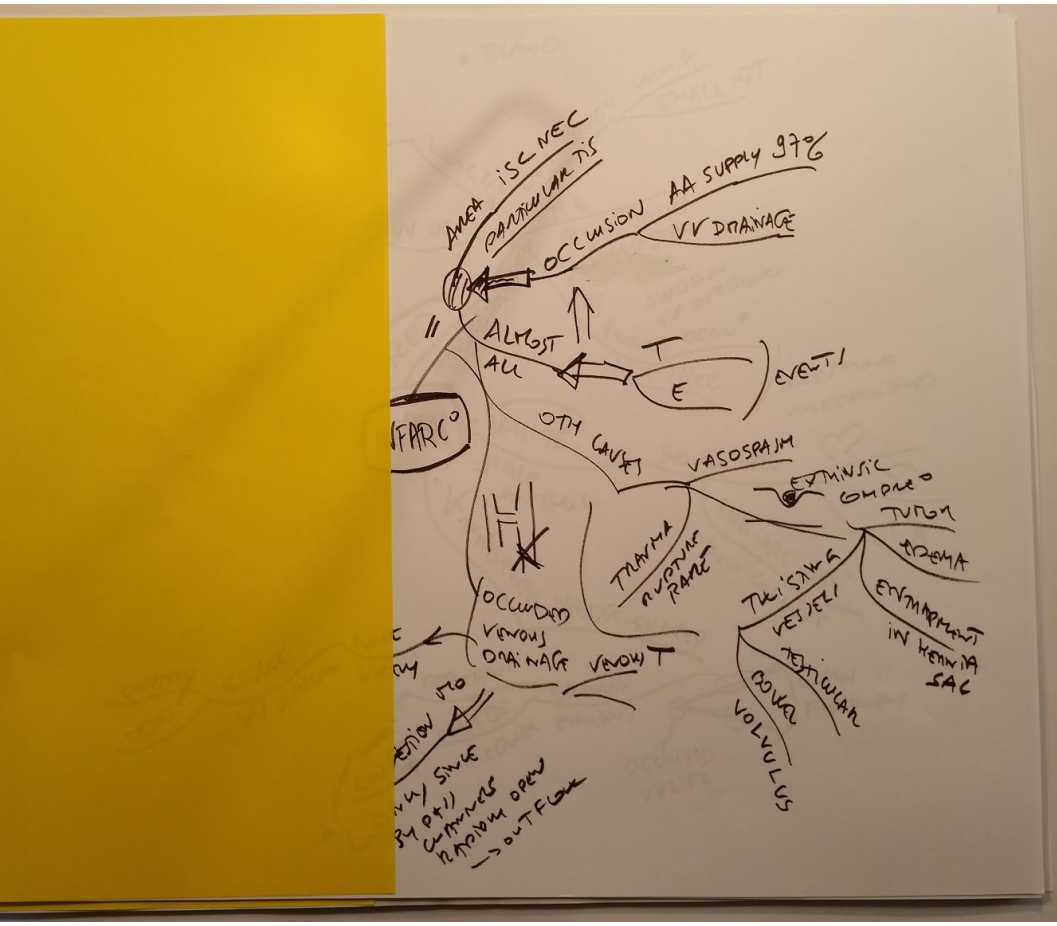

SYSTEMIC TE
TIS
IFRC
TIS
VULNERABILITY
TO ISC
VESICLE
F°
CAUSES
MOST
A°E
GL4
LAT
LASC SYDDY
ANTERIAL
CIRC
INTRAC CARDIAC
NEONATAL
ANGLE FROM
80%
213
25%
DILATED LA
FIBRILLA°
SPLEEN
NIDNEY
INTEST
MAJOR SITES
BRAIN
LUNG
10%
7-10%
A° EMBO
10-15%
MARE
ALSO
PARADOXICAL E
UNKNOWN
ORIGIN
ACUTE ARTERIAL
T
ULCM
ATHEROS
PLAQ
VALV
VEGETA°
VENOUS
ATRIAL
VENTRICULAR
SEPTAL
DEFECTS
PATENT FO

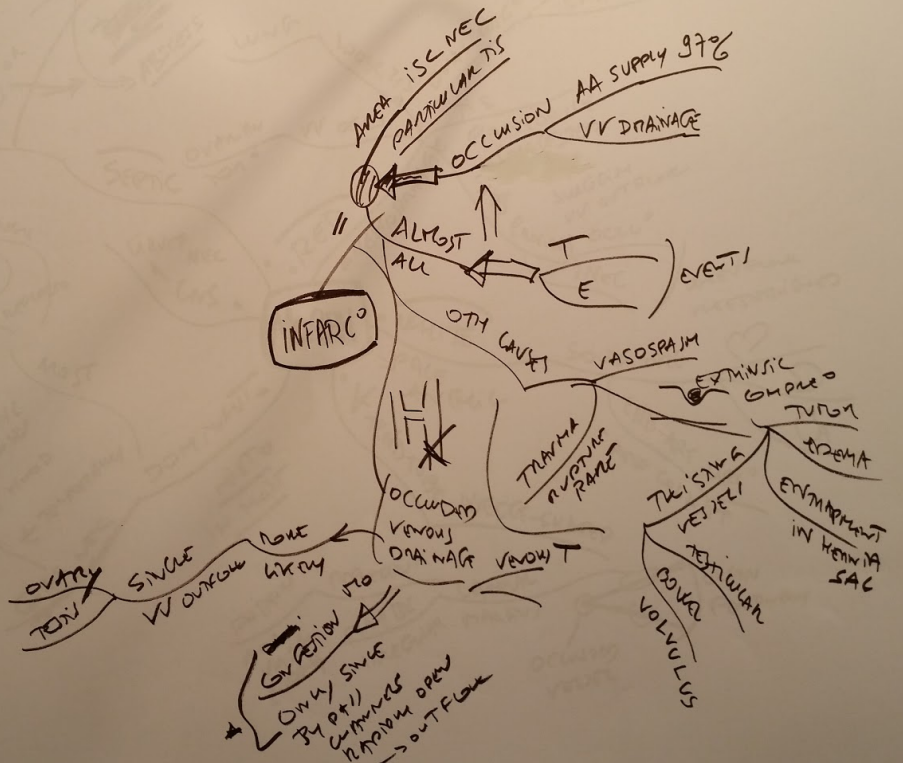

FAT
MARROW
E
DD
PE
MIC FAT GLOBULES
HEMAT.
MANNOR
ELEMENT
FRACTURED LONG BONES
PANCLY
BURN)
SOFT TIS TRAUMA
30% SEVERE SKEL INJ
<10% HAEMM CU
FAT E
FATAL ~10%
SUDDEN PULM INJUF
1-3D
iW JURY
20-10% DIFFUSE PETECHIAL
MAY
NEURO
GOMA
DECIDUM
MAY
CAUSE
PULM HYALINE
MBS
EDEMA
HEMOR
ENDOT
TOX INJ
PLAT
SUBSEQUENT
FA RELEASE
ERYTHROC
AGGREGS
LOC PLAT
TECHANICAL
OBSTRUC
NORMAL
FAT
ME
THROMBOSIS
ANEMIA
CAN OCCUR

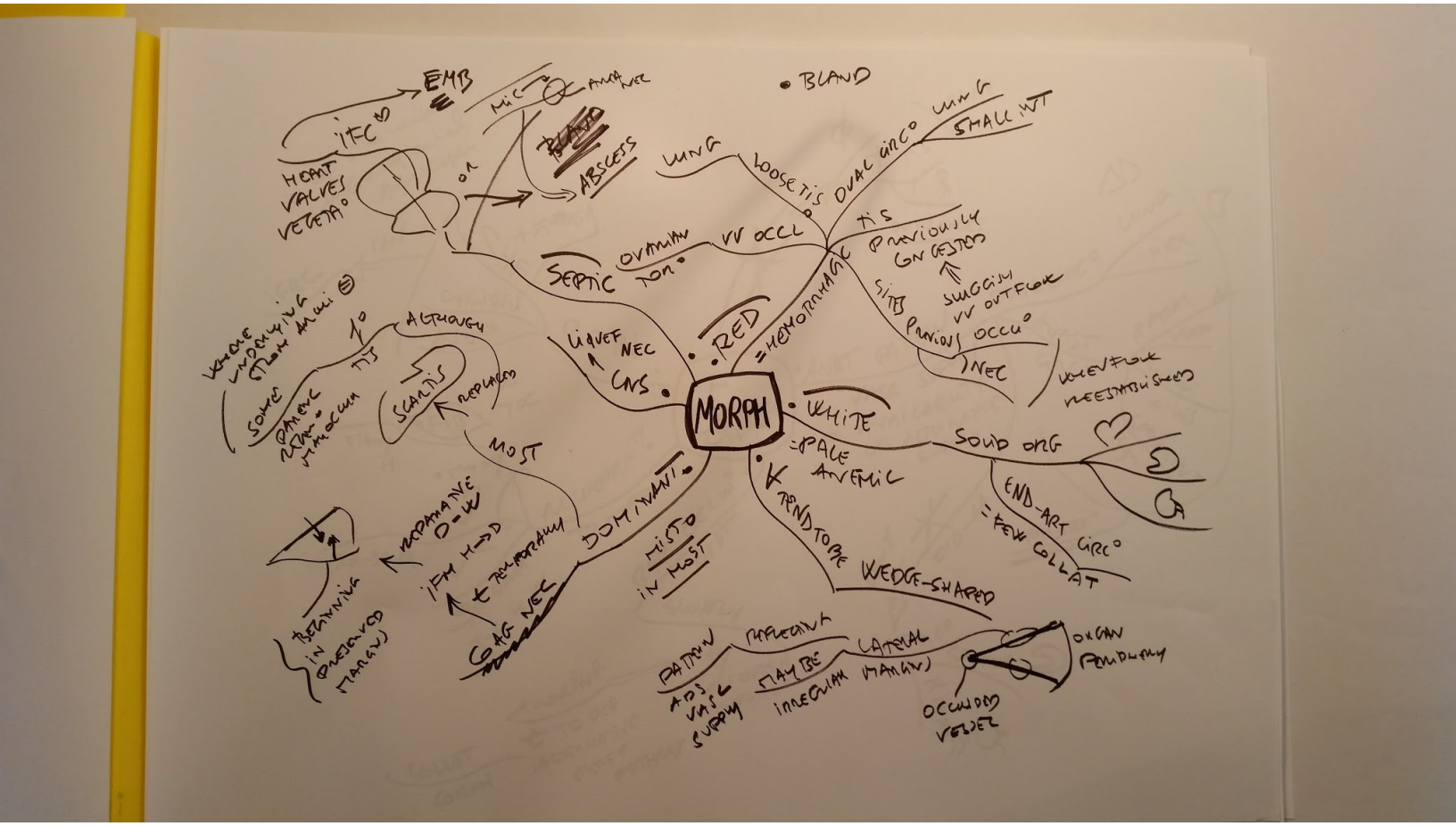

AIR
E
+G
FORM
SMALL
AMOUNTS
SURFACE
GENY
DECOMPRESSION
SICKNESS
GAS BUB
JOINTS
SKELM
PAINFUL
BENDS
LUNGS
HUMAN
EDEMA
CHEST
PAIN
BRAIN
HEART
ISC NEC
TIBIA
HUMER
FEMUR
OBSTRUC
ISC
GAS BUBBLES
CAN
CATASTROPHIC
CBn
GN
RISK
RAPID
ASCENT
DEEP SEA DIVES
UNPRESSURIZED
AIR CRAFTS
SUDDEN
AIR
PRESSURE
CHEST PAIN
OBST
PAIN
RAPID ASCENT
SUBSEQUENT
GAS
DISSOLVED
TISSUES

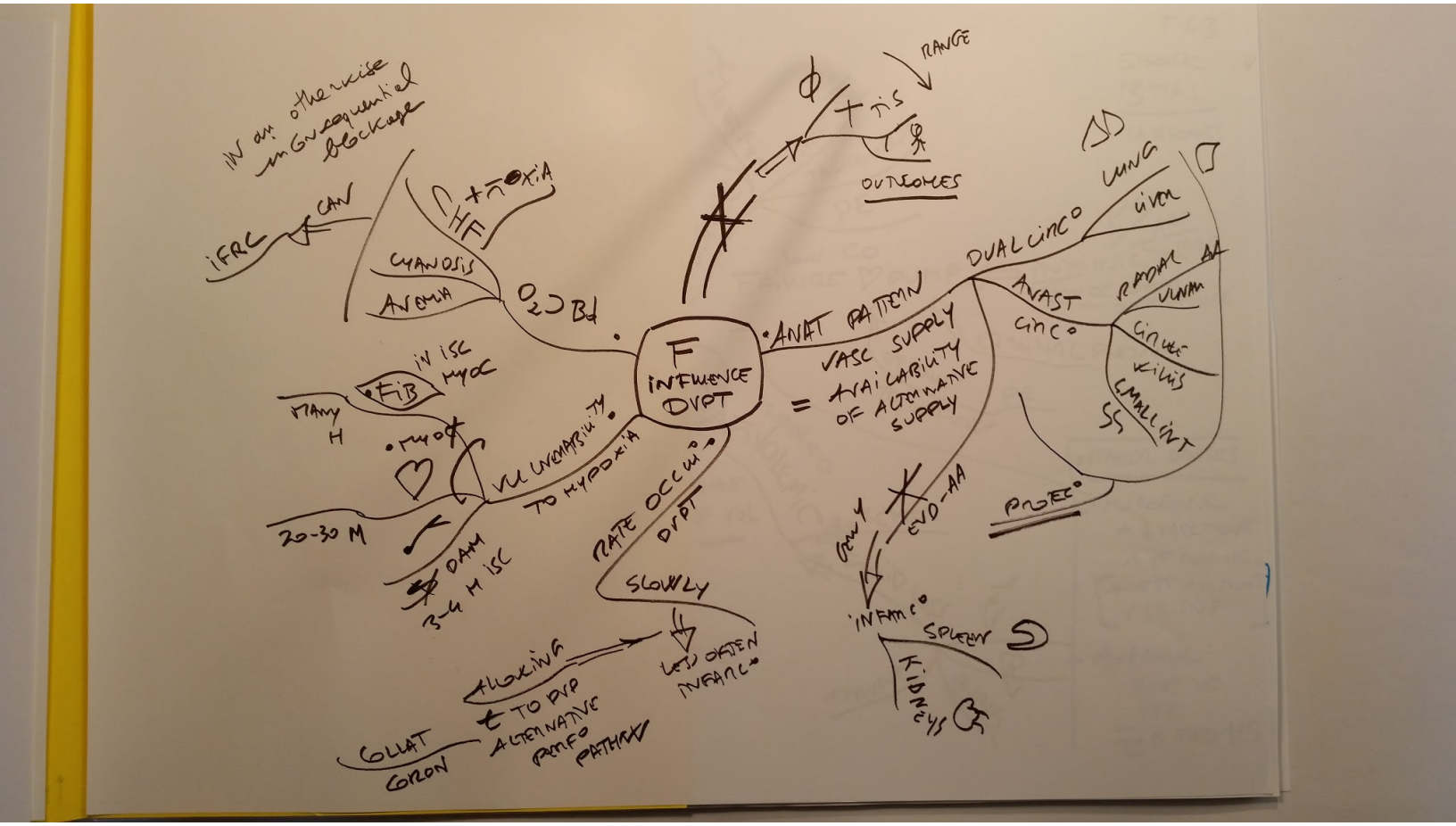

AF
MATERNAL CINC
MORTALITY RATE UP 8%
INCIDENCE 1/40,000 DELIV
LABOR POSTPARTUM
SD
SUD SEVERE DYSPNEA
CYANOSIS
AT SHOCK
SEIZURES
COMA
PE
DIC
DIF DX DVT DMM
INTRAT MANAG TESTS
VENOUS CATHS
FAT
LARVAGE FAT
VEIN CIN
FET
RELEASE TOXINS
THROMBOGENIC SUB IN AF

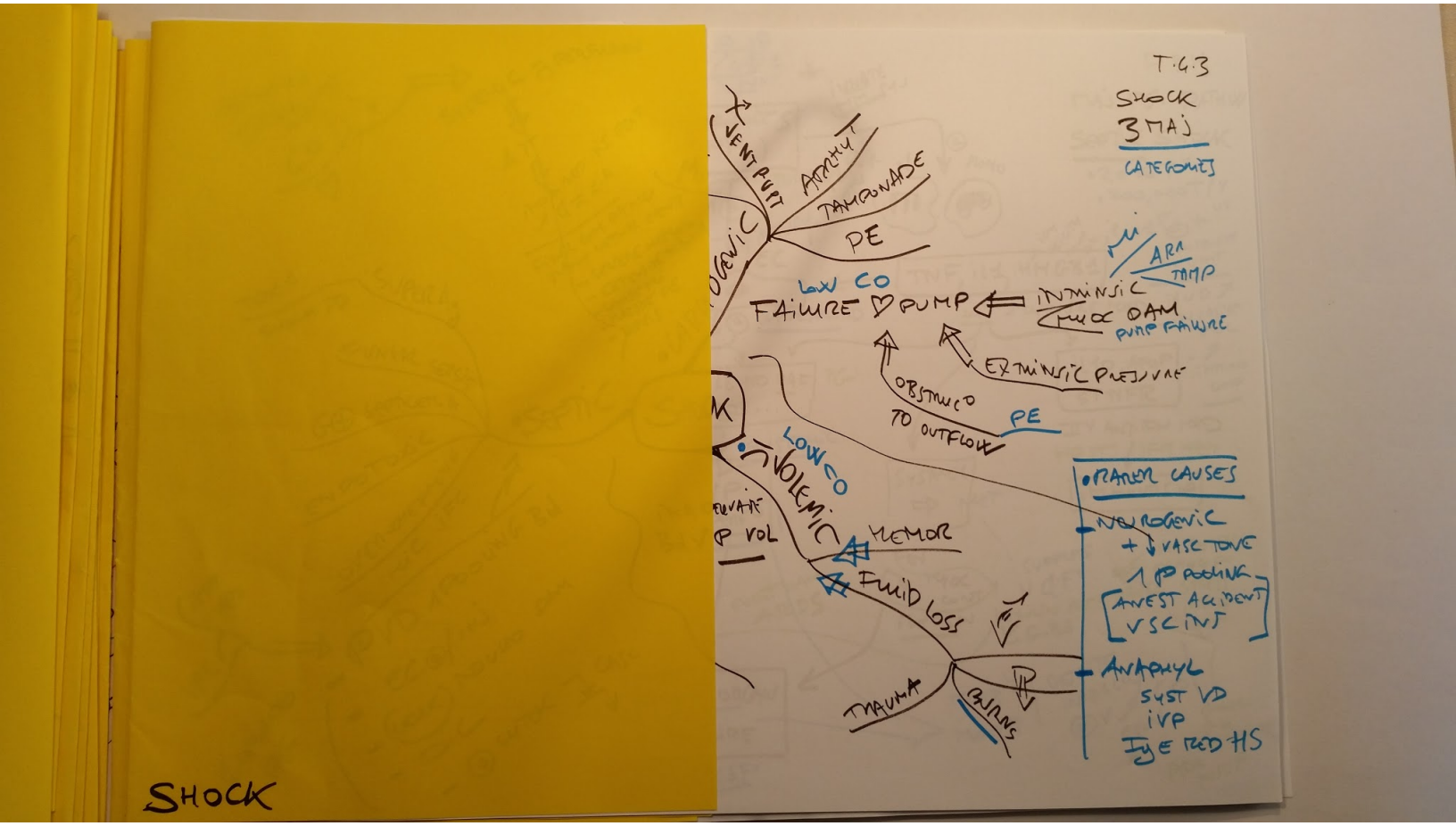
INFARCTION

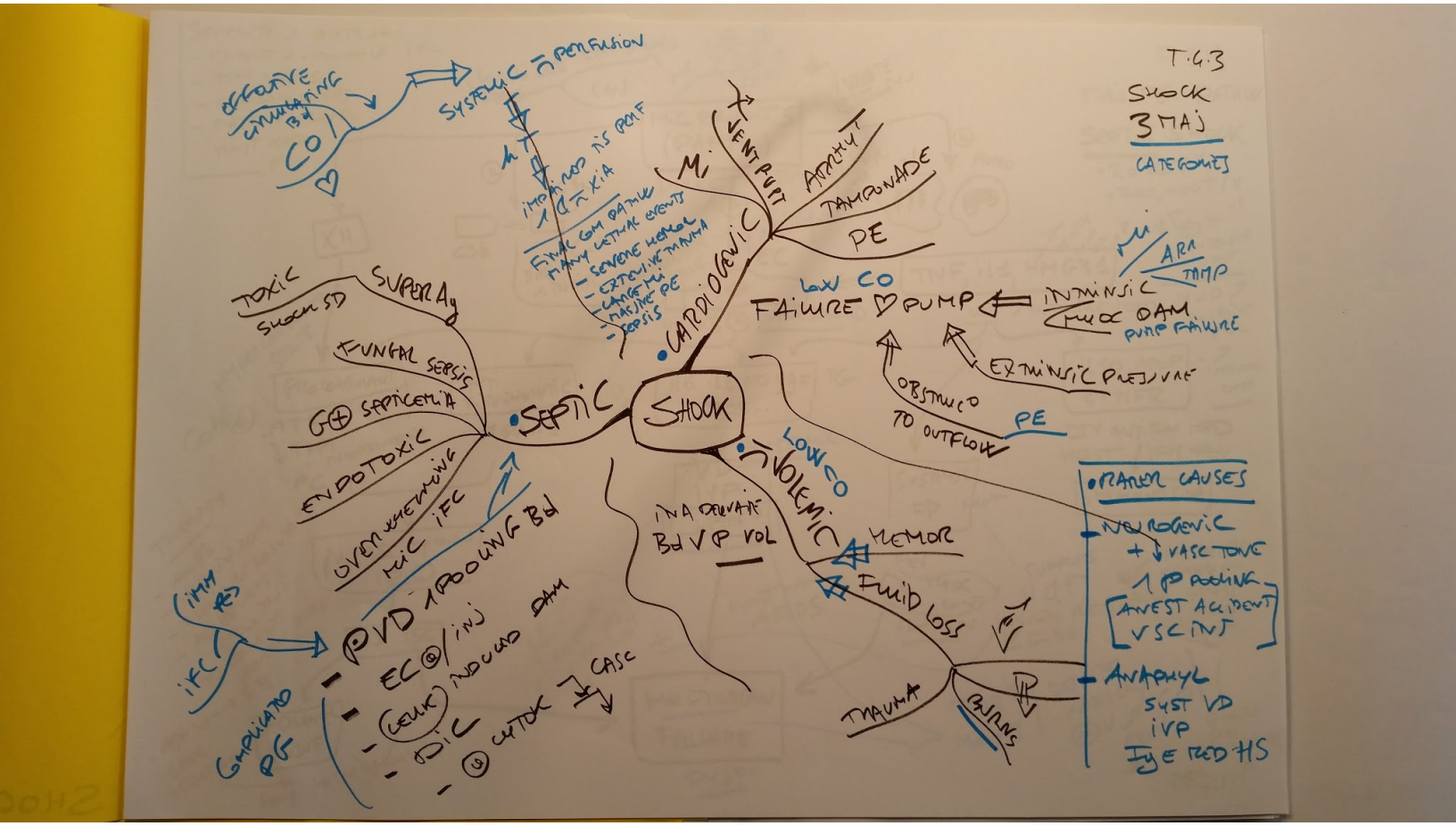

AREA ISCHEC
PARTICULAR TIS
OCCLUSION
AA SUPPLY 97%
VV DRAINAGE
ALMOST
ALL
T
E
EVENTI
OTH CAUSES
VASOSPASM
EXTRINSIC
COMPRES
TUMOR
EDEMA
THROMBUS
RUPTURE
RARE
OCCLUDED
VENOUS
DRAINAGE
VENOUS T
THROMBUS
VEINS
PERIVASCULAR
EDEMA
VOLVULUS
ENTRAPMENT
IN HERNIA
SAC

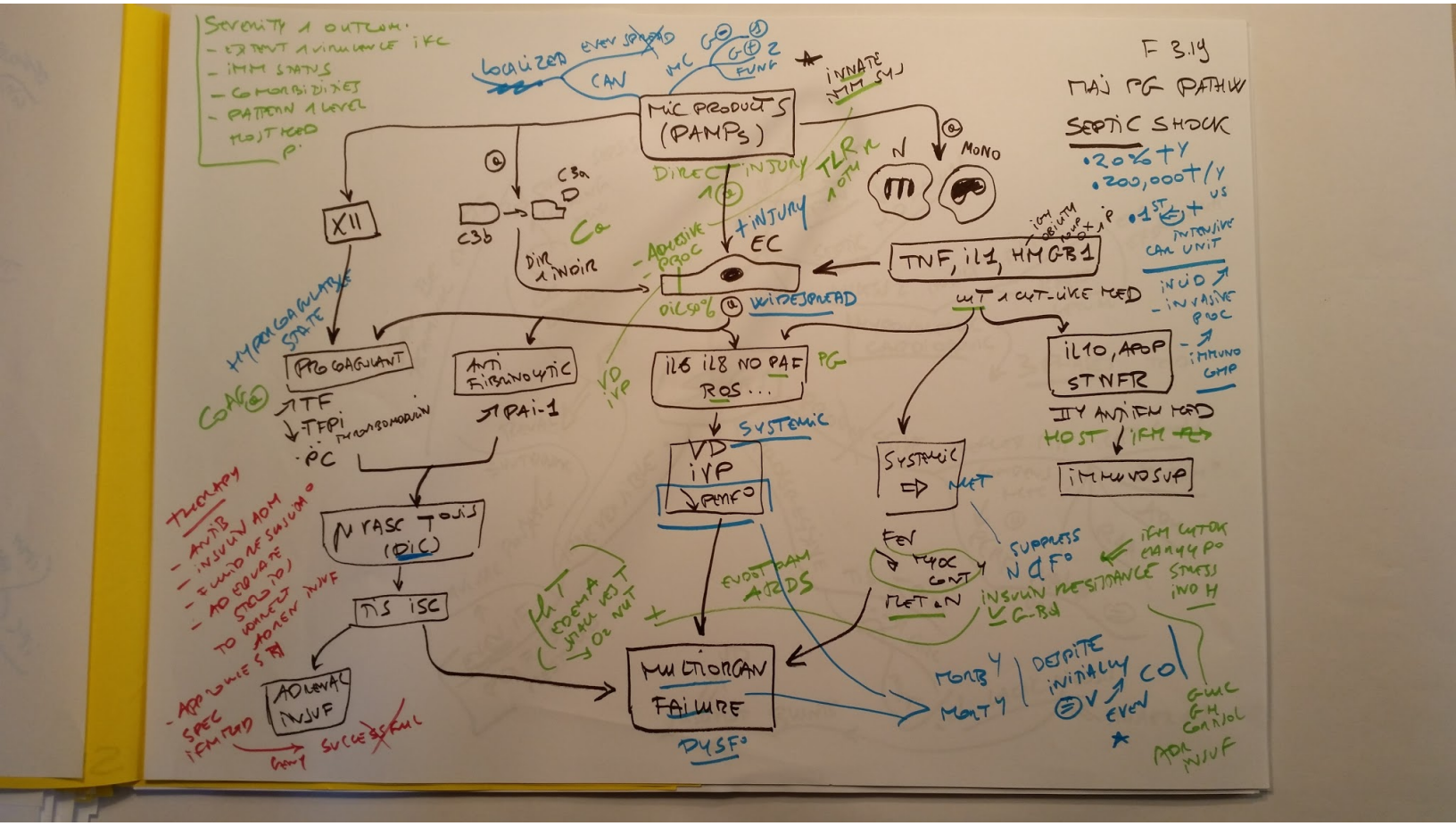

AREA ISCHEC
PARTICULAR TIS
OCCLUSION AA SUPPLY 97%
VV DRAINAGE
ALMOST ALL
T
E
EVENTI
INFARC°
OTH CAUSE
VASOSPASM
TRAUMA
RUPTURE RARE
OCCLUDED VENOUS DRAINAGE
VENOUS
SINGLE VV OUTFLOW
LONE
LITTLE
OVARY
TORSI
GANGRENOUS
ONLY SINGLE BY PATU CHANNELS RANDOM OPEN OUTFLOW
TWISTING VESSELS
PERITUBULAR
COMPLETE VOLVULUS
ENTRAPMENT IN HERNIA SAC
TUMOR
EDEMA

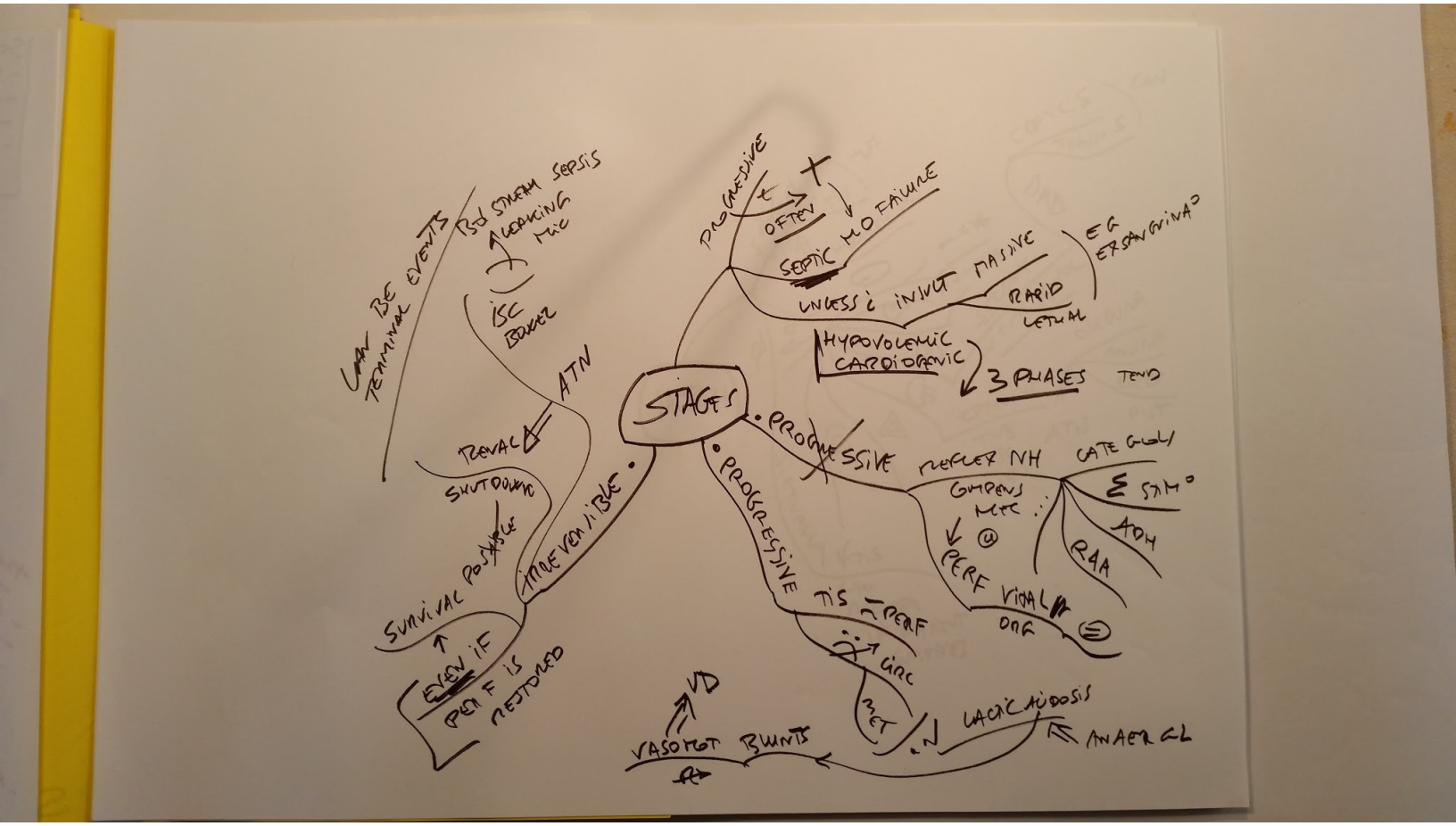

MORPH
RED
=HEMORRHAGIC
BLAND
WHITE
=PALE ANEMIC
DOMINANT
MOST
SEPTIC
ABSCESS
EMB
INFC
HEART VALVES VEGETA°
LUNG
LOOSE TIS
OVAL CIRC°
SMALL INT
TIS
PREVIOUSLY GANGRENOUS
SUGGEST W/ OUTFLOW
SITE PREVIOUS OCCL°
INFC
LIQUEF NEC
CNS
SOLID ORG
END-ART CIRC°
=NEW COLLAT
WEDGE-SHAPED
PATHOM
ADJ VASC SUPPLY
INFECTION
CAPILLARY
MAYBE
CAPILLARY
OCCLUDED VESSEL
OXGN PENDULUM
SCANTIS
REPLACED
ALTHOUGH
1°
TTT
SOME
AMONG
NECROSIS
HYPOXIC
UNDERLYING
STEM ANGIO
HYPOXANTE
D-W
ITH H→D
← REMARKABLY
GAS NEC
BEGINNING IN PREEXISTING
MISTO
IN MOST

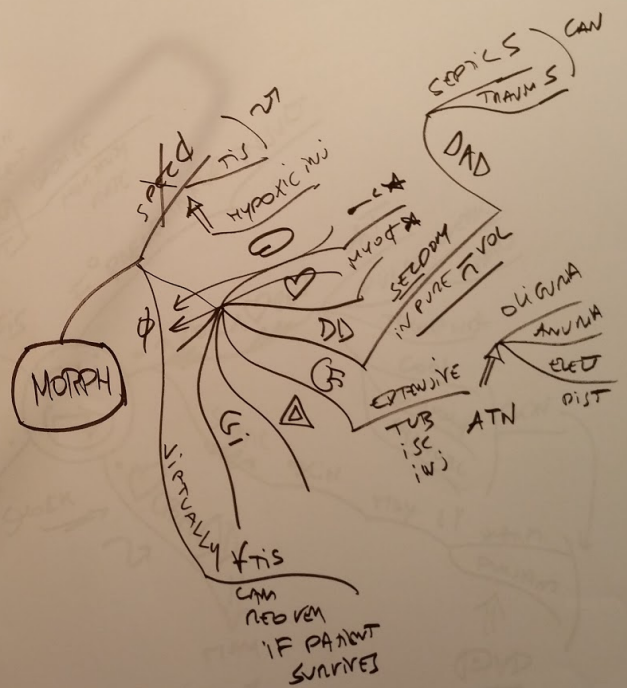

F
INFLUENCE DVPT
ANAT PATTERN
VASC SUPPLY
= AVAILABILITY OF ALTERNATIVE SUPPLY
DUAL CIRC°
RADIAL AA
ULNAR
CIRCUM-FLEX
SMALL INT
PROFF°
AVASC CIRC°
CVD-AA
INFARC°
SPLEEN
KIDNEYS
RATE OCCUR° DVPT
SLOWLY
LESS OFTEN INFARC°
BLOCKING
TO DVP ALTERNATIVE PERFO PATHWAY
COLLAT CORON
VULNERABILITY TO HYPOXIA
20-30 M
DAM 3-4 H ISC
MANY H
FIB
IN ISC MYOC
CHF
TOXIA
CYANOSIS
ANEMIA
IFRC
CAN
IN an otherwise uncomplicated blockage
RANGE
OUTCOMES

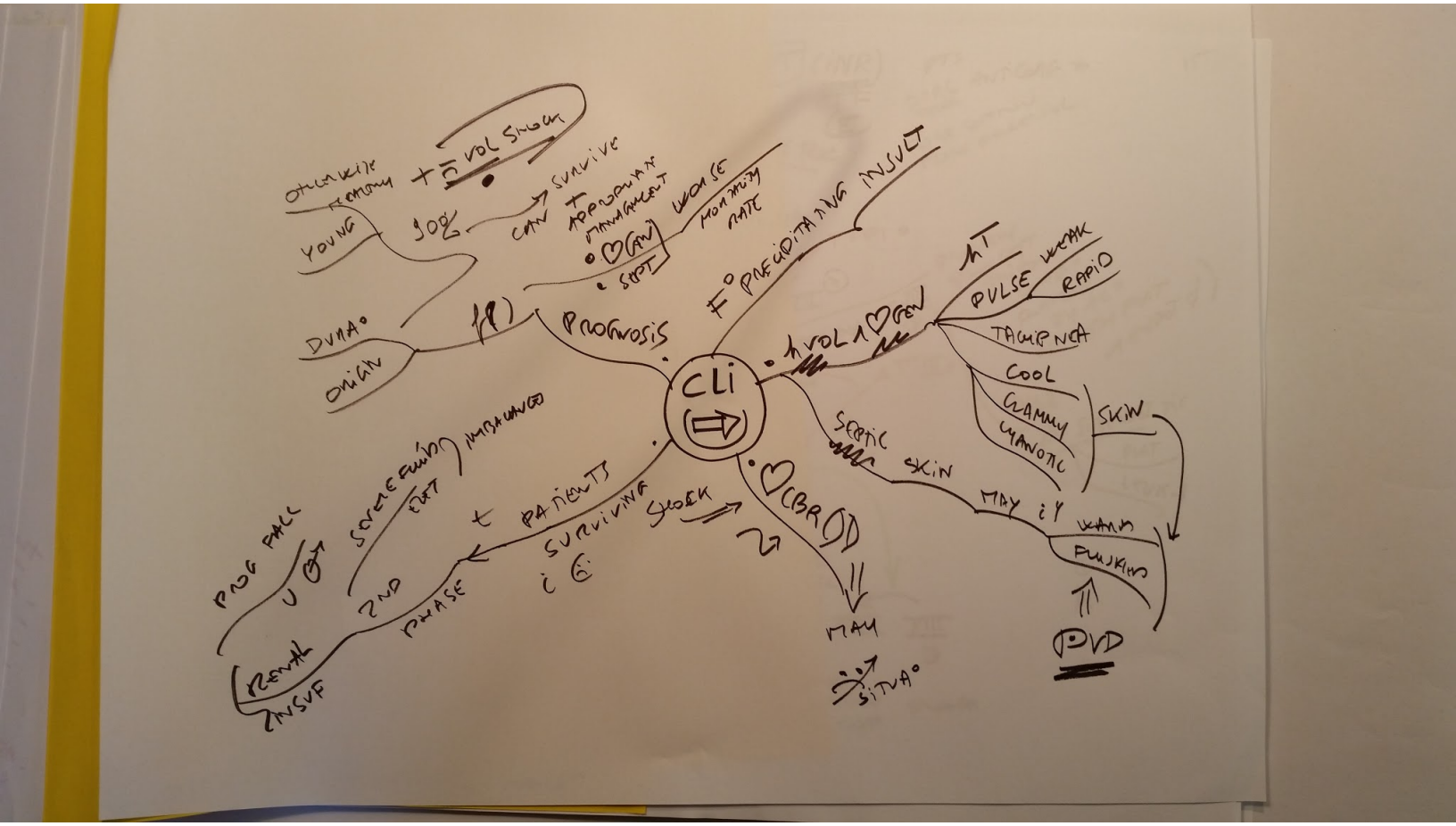

T·G·3
SHOCK
3 MAJ
CATEGORIES
HYPOVOLEMIC
ANAPHY
TAMPONADE
PE
LOW CO
FAILURE
PUMP
INTRINSIC
MI
CARDIOMYOPATHY
PUMP FAILURE
EXTRINSIC PRESSURE
OBSTRUCTION TO OUTFLOW
PE
LOW CO
HYPOVOLEMIC
HEMORR
FLUID LOSS
TRAUMA
BURNS
MAJOR CAUSES
NEUROGENIC
+ ↓ VASC TONE
↓ PRELOAD
[ANEST ACCIDENT
VSC INJ
ANAPHYL
SYST VD
↓ VP
↑ E RED HS
SHOCK

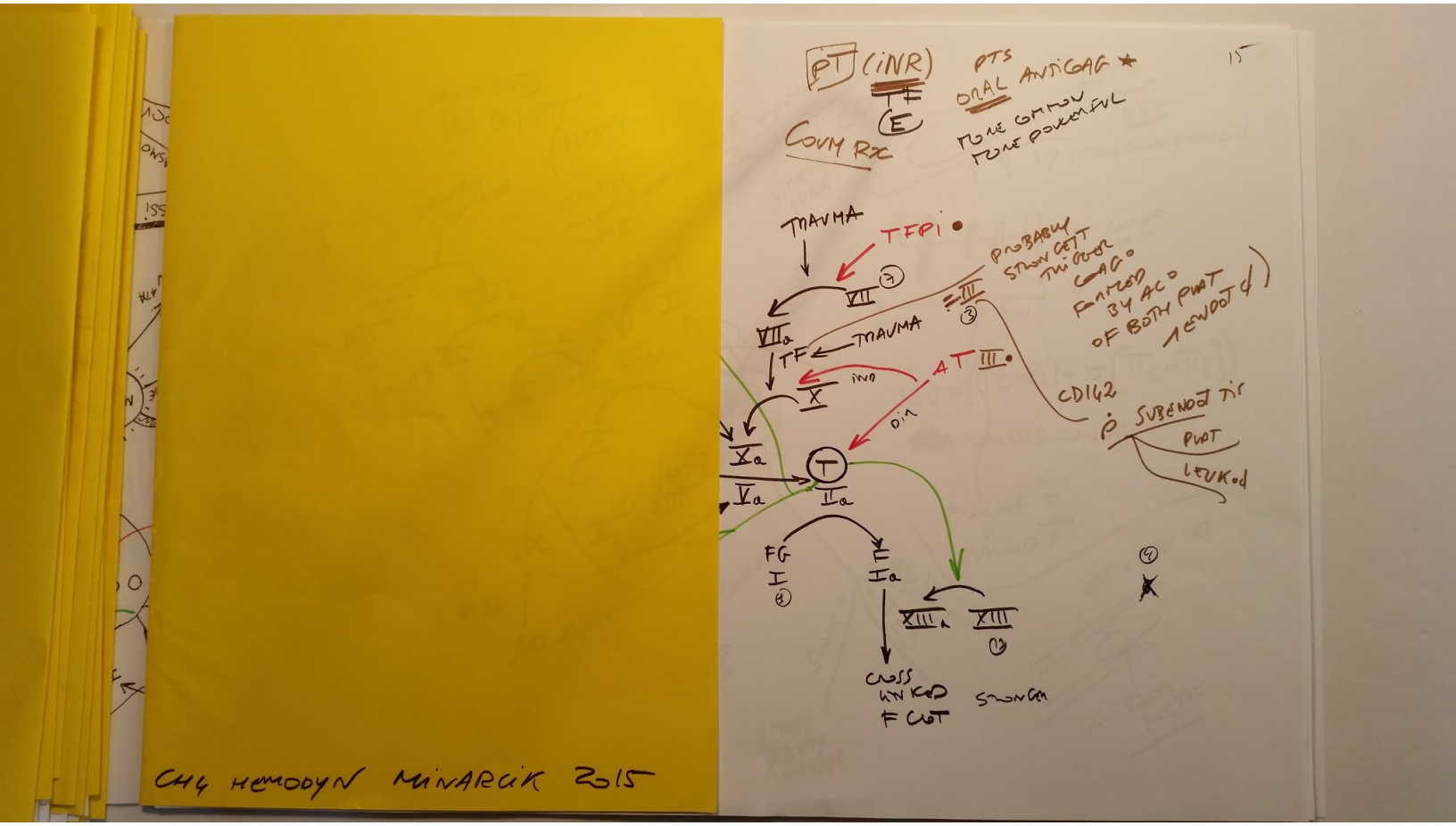

T.G.3
SHOCK
3 MAJ
CATEGORIES
SHOCK
CARDIOGENIC
Mi
VENTRICULAR
ARRHYT
TAMPONADE
PE
FINAL COMMON PATHWAY
MANY LETHAL EVENTS
- SEVERE MEMBR
- EXTENSIVE TRAUMA
- LARGE PE
- MASSIVE PE
- SEPSIS
SEPTIC
TOXIC SHOCK SD
SUPERAg
FUNGAL SEPSIS
SEPTICEMIA
ENDOTOXIC
OVERWHELMING
HIC IFC
PVD
ECG/INJ
INDUCED DAM
DIC
CYTOK
CASC
IMM RX
IFC
COMPLICATED PG
HYPOVOLEMIC
INADEQUATE BV VP VOL
HEMOR
FLUID LOSS
TRAUMA
BURNS
LOW CO
FAILURE PUMP
INTRINSIC
DAM
PUMP FAILURE
EXTRINSIC PRESSURE
OBSTRUCTO TO OUTFLOW
PE
ARR
TAMP
PRIMARY CAUSES
NEUROGENIC
+ VASCTONE
1 PP POOLING
[ANEST ACCIDENT VSC INJ]
ANAPHYL
SYST VD
IVP
IgE MED HS

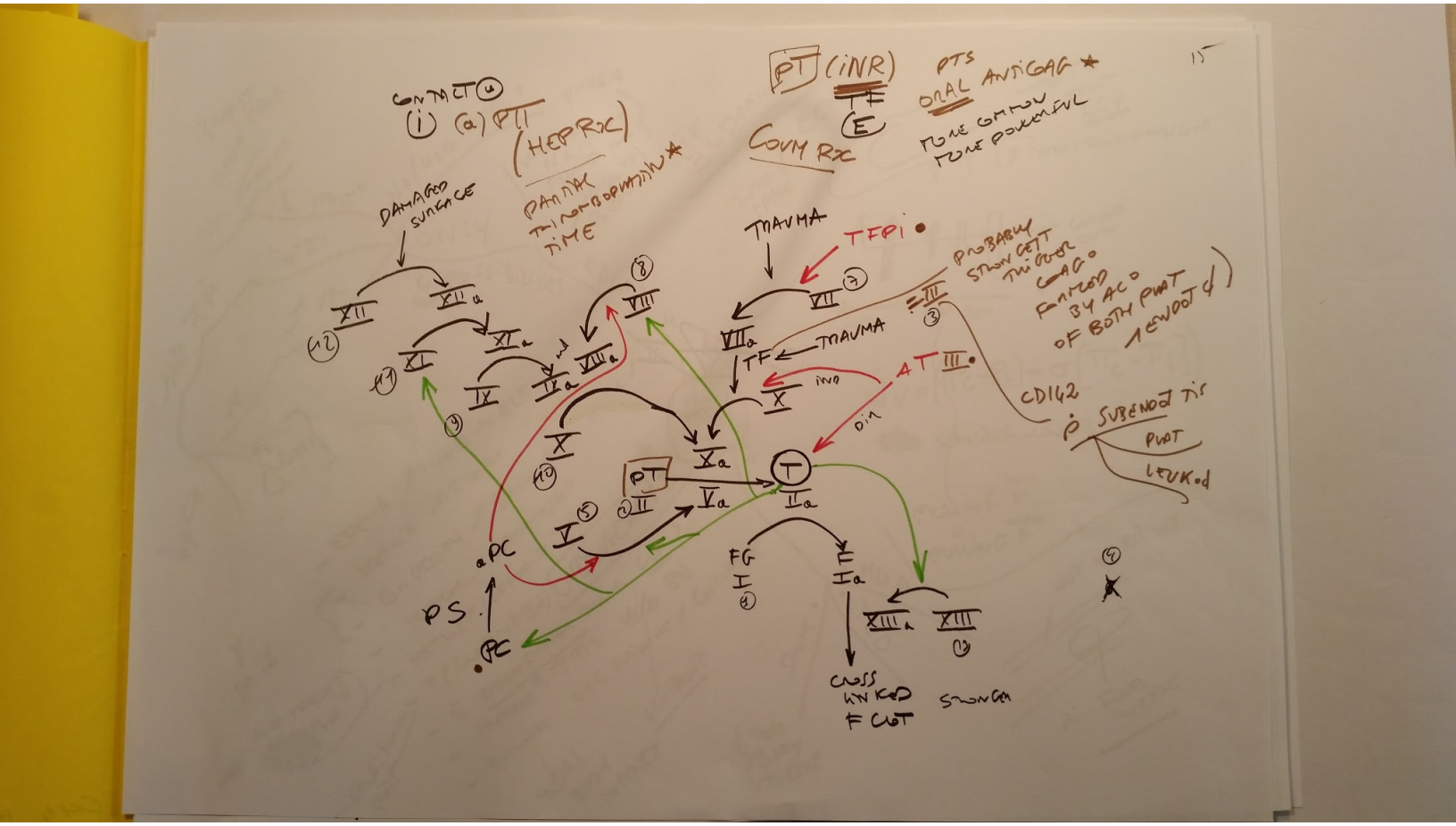

Severity / outcome:
- extent / virulence / IFC
- imm status
- comorbidities
- pattern / level
MAJ PG pathway
Septic shock
•20% +Y
•200,000 +/Y
US
•1st
Localized
CAN
MC
Innate imm sys
MICROBIAL PRODUCTS (PAMPs)
DIRECT INJURY
TLR
NOTH
N
MONO
XII
C3a
C3b
Ca
DIR / INDIR
+INJURY
EC
- ADHESIVE PROC
- PROC
TNF, il1, HMGB1
IL1 / CXCL-like med
iNOS
- invasive proc
Hypercoagulable state
PRO COAGULANT
ANTI FIBRINOLYTIC
il6 il8 NO PAF ROS ...
PG
il10, ADAP sTNFR
- immuno comp
TF
↓TFPi
Thrombomodulin
↓PC
↑PAI-1
VD
iVP
SYSTEMIC
VD
iVP
PERFO
SYSTEMIC
LEFT
Immunosup
Therapy
- Antib
- insulin adm
- Fluid resuscitation
- AD equivalent
stabilize
to control
Adren inhf
MYASCTOMIS (PIC)
TTS ISC
Appropriate spec if limited
Adrenal insuf
Fail
Success
Multiorgan failure
Dysf°
Edema
Tissue vert
↓O2 nut
Endotoxaemia
ARDS
FEV
↑Proc cmt
↑Left shift
Suppress NO f°
Insulin resistance
↓C-Bd
IFM cytok changes
Stress inoh
Mortality
Despite initially
Even
Glyc
GH
Control
ADR insuf

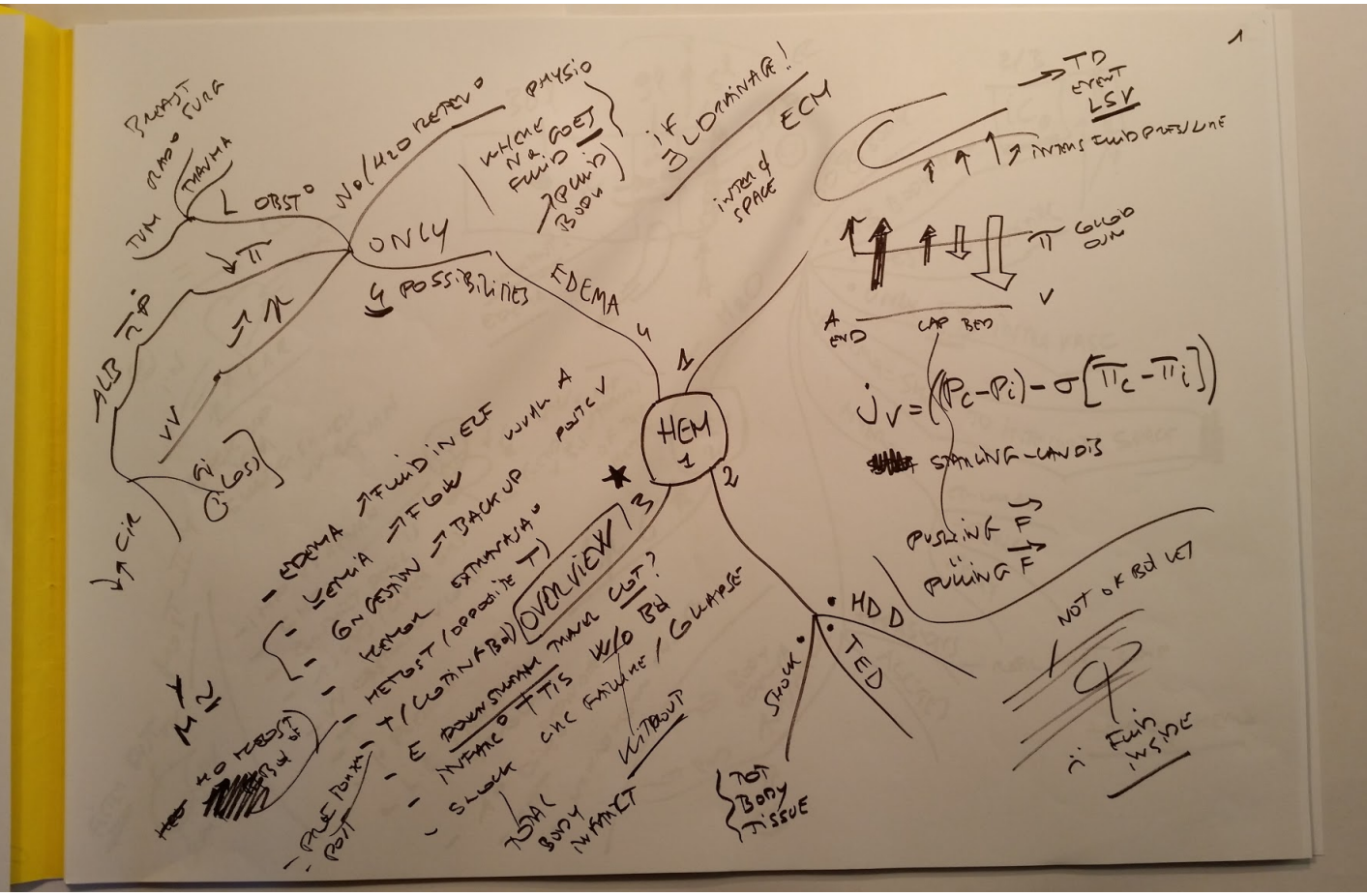

STAGES
PROGRESSIVE
SEPTIC MO FAILURE
OFTEN
UNLESS i INVLT
MASSIVE
RAPID
LETHAL
EG EXSANGUINA°
HYPOVOLEMIC
CARDIOGENIC
3 PHASES
TEND
PROGRESSIVE
REFLEX NH
COMPENS MECH
@
PERF VISAL
ORG
RAA
AOH
SIM°
PROGRESSIVE TIS ↓PERF
↑ CIRC
MET
↓N
LACTICAIDOSIS
ANAER GL
VD
VASOPGT
BLUNTS
ATN
ISC
BOWEL
BSI STREAM SEPSIS
LEAKING
MIC
CAN BE TERMINAL EVENTS
TRIVAL
SHUTDOWN
SURVIVAL POSSIBLE
IRREVERSIBLE
EVEN IF
PERF IS
RESTORED

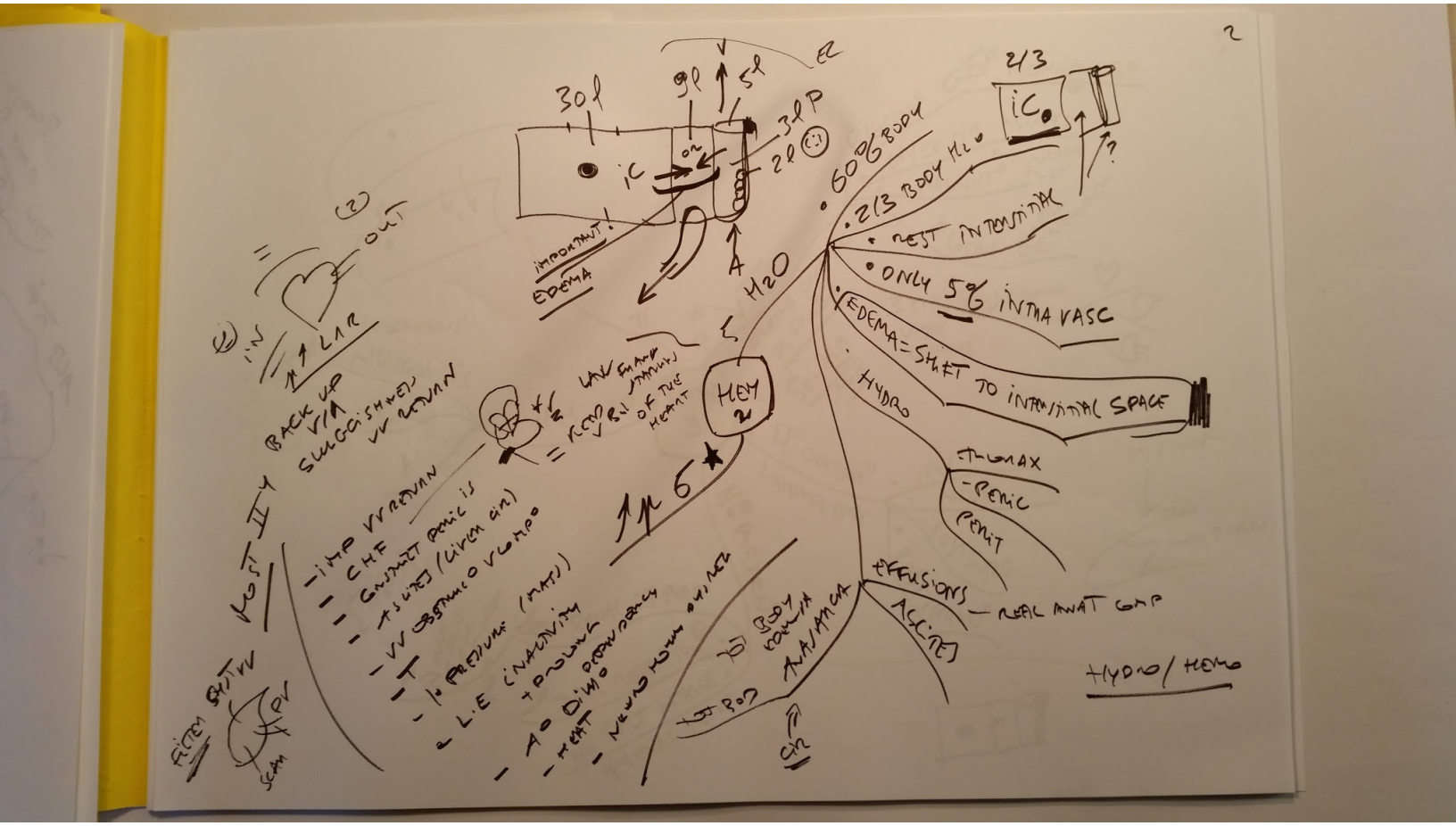

MORPH
DAD
DD
GS
Gi
ATN
TUB ISC INJ
EXTENSIVE
SEPTICS
TRAUMAS
HYPOXIC INJ
MYOGL
SEVERITY
OLIGURIA
ANURIA
ELEC
DIST
VIRTUALLY
CAN RECOVER IF PATIENT SURVIVES

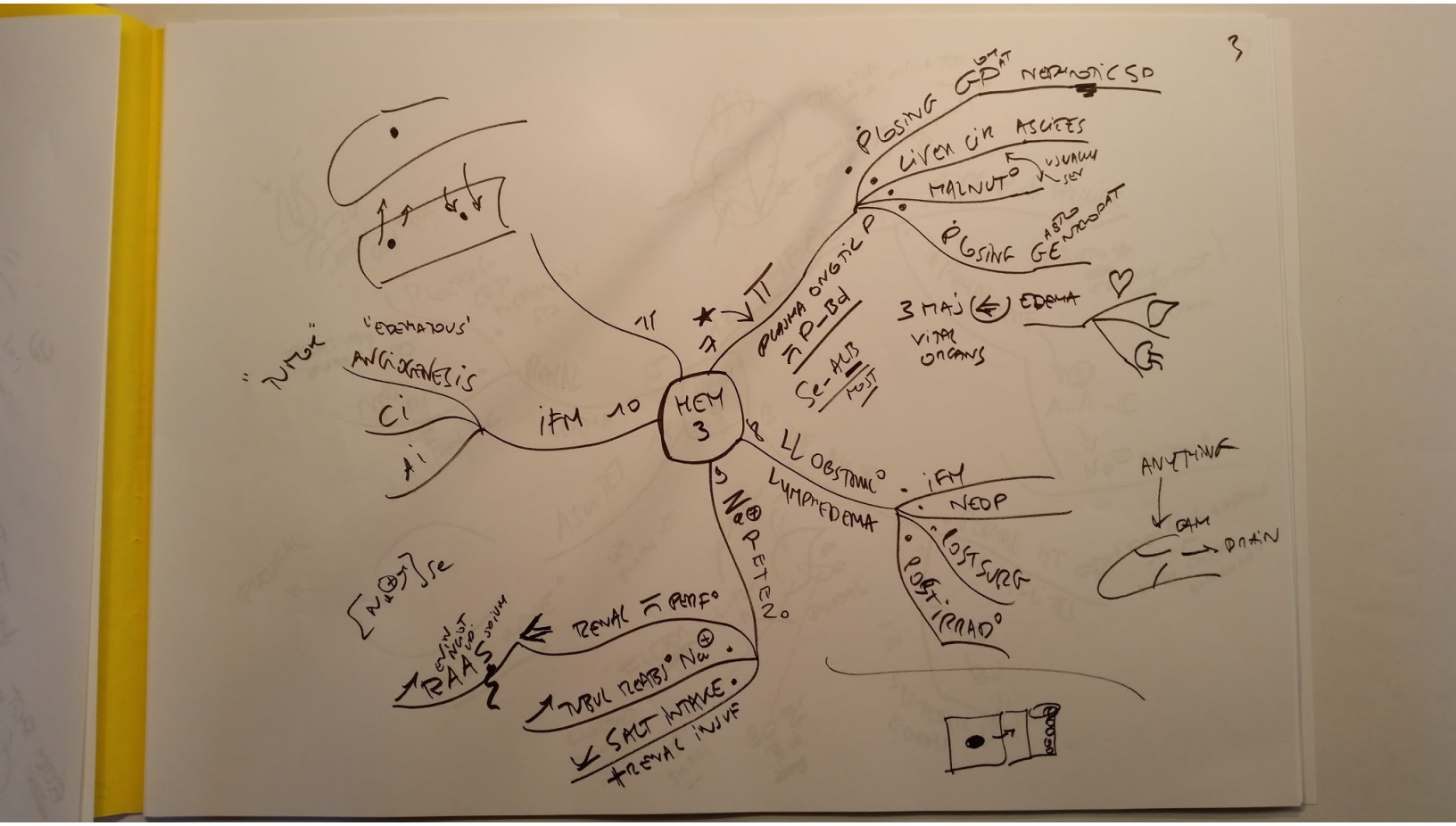

CLi
PROGNOSIS
DX
ORIGIN
YOUNG
SURVIVE
APPROPRIATE
MANAGEMENT
SEPT
HOSPITAL
MORTALITY
RATE
PRECIPITATING
INSULT
PULSE
RAPID
TACHYPNEA
COOL
CLAMMY
DIAPHORETIC
SKIN
SEPTIC
MAY
PURPURIC
PVD
CBRN
MAY
SIRS
PATIENTS
SURVIVING
SHOCK
SECONDARY
INSULT
2ND
PHASE
PROG FAIL
RENAL
INSUF

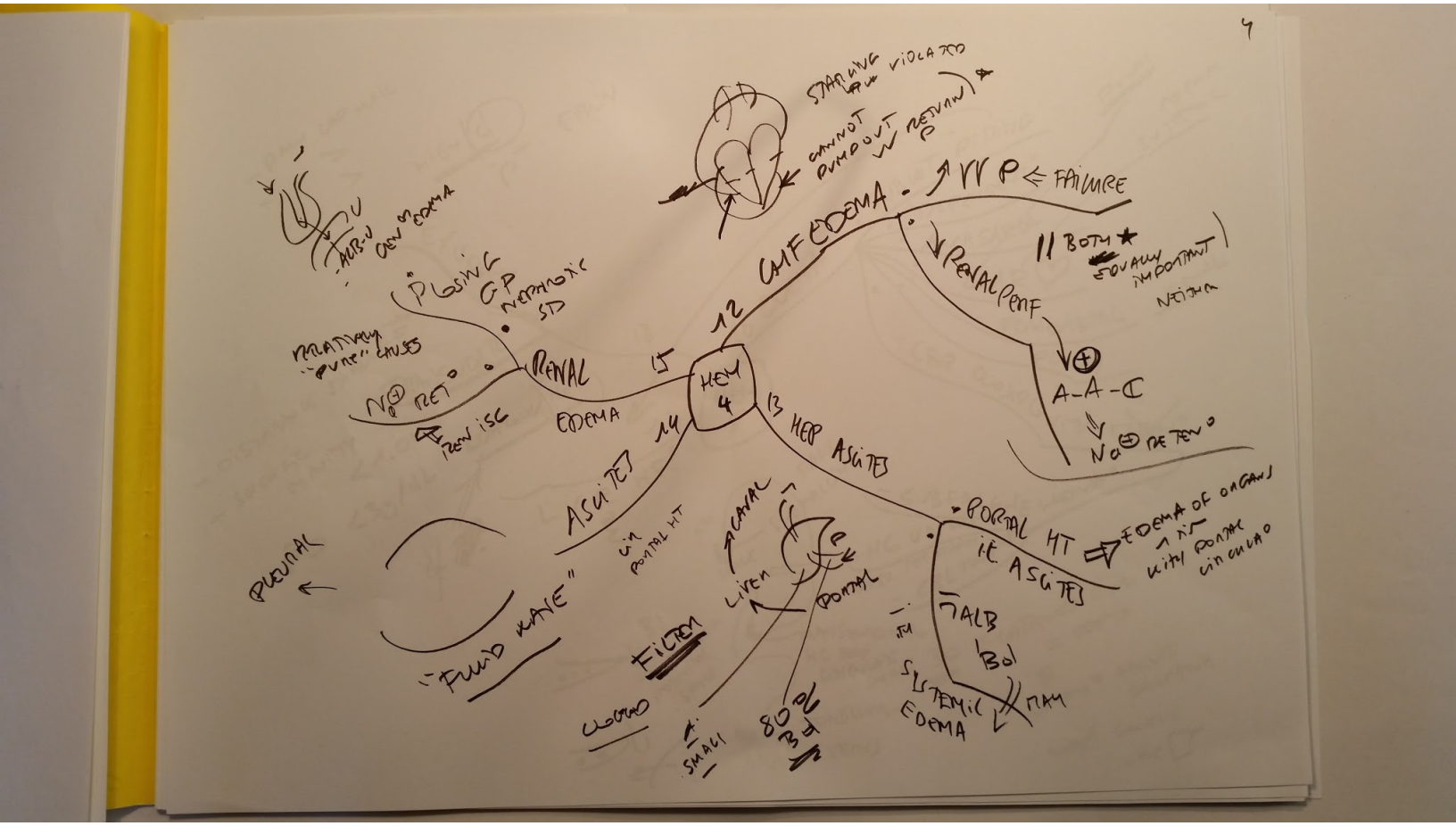

PT (iNR)
PTS
ORAL ANTICOAG ★
COUM RX
TRAUMA
TFPI
VII
TF
TRAUMA
AT III
X
Xa
Va
II
IIa
FG I
IIa
XIII
XIII
CROSS LINKED F CLOT
STRONG
PROBABLY SLOW GETT THROMB CAC FACILITATED BY AC OF BOTH PLAT + ENDOT ↓
CD142
SUBENDOT TIS
PLAT
LEUK.d
CHG HEMODYN MINARCIK 2015

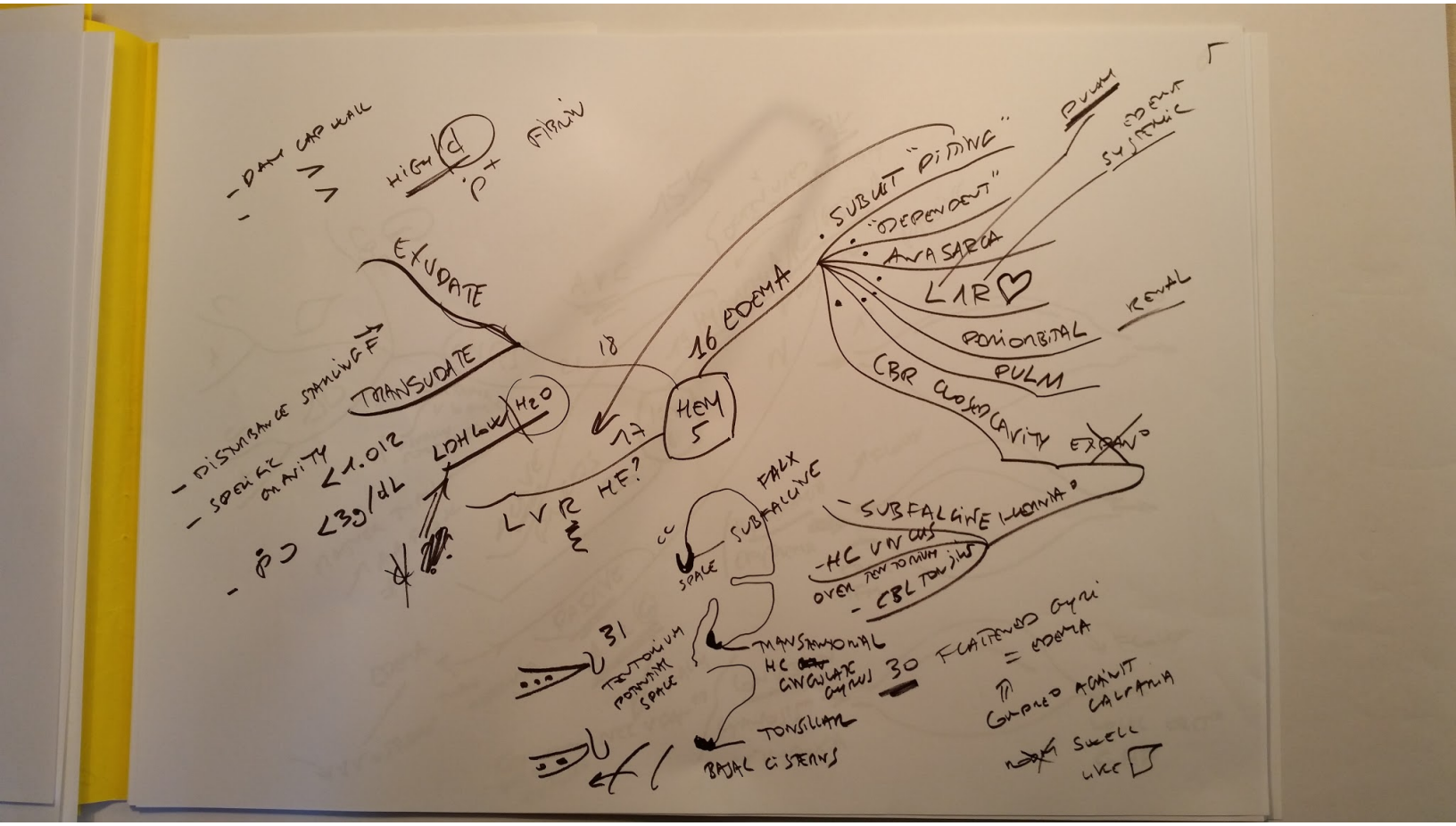

DAMAGED SURFACE
TRAUMA
TFPi
TF
AT III
CD142
PS
PC
FG
CROSS LINKED FIBRIN

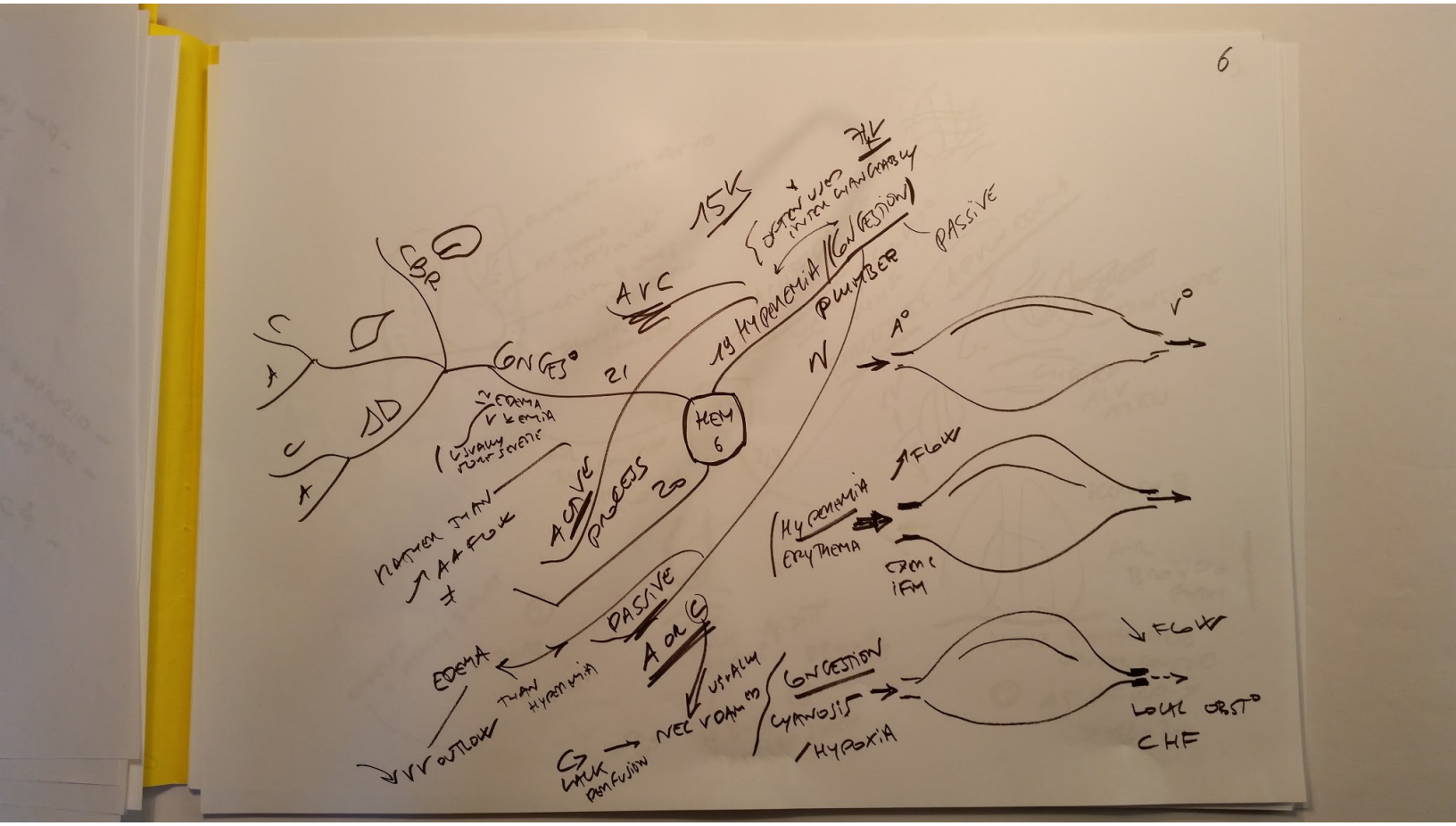

HEM
ONCY
EDEMA
OVERVIEW
HDD
TED
SHOCK
ALB
VV
V cir
L OBST°
PHYSIO
IF DOMINANT!
interst SPACE
ECM
TD
LSV
CAP BED
PUSHING F
PULLING F
NOT OF BLD VET
FLUID INSIDE
TOTAL BODY INFANCT
FOR BODY TISSUE

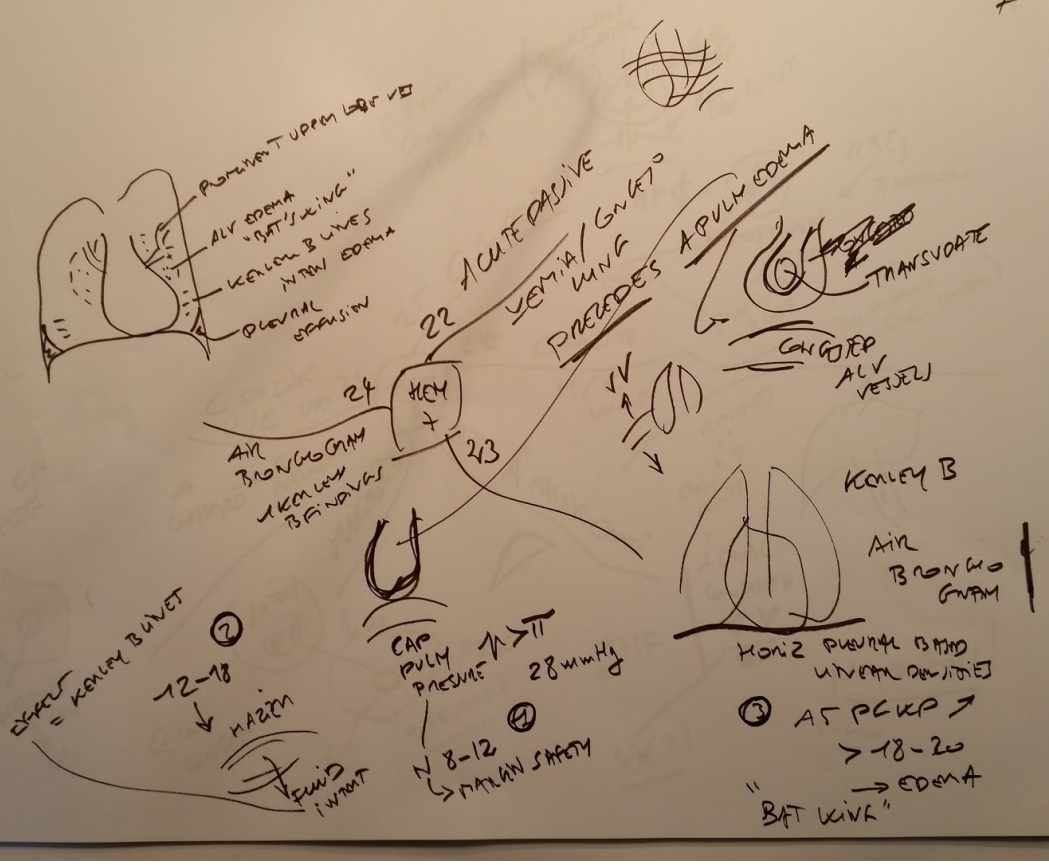

2
30 l
gl
5 l
ec
3 l P
2 l
iC
on
IMPORTANT!
EDEMA
A
H2O
2/3
iC
?
60% BODY
2/3 BODY H2O
MOST INTERSTITIAL
ONLY 5% INTRA VASC
EDEMA = SHIFT TO INTERSTITIAL SPACE
HYDRO
THORAX
PERIC
PERIT
EFFUSIONS
ASCITES
REAL ANAT GAP
HYDRO / HEMO
HEART
2
1/1 6
BACK UP VIA SLUGGISHNESS OF RETURN
IN
OUT
1/1 LAR
MOST IT
-IMP VARIATION
- CHF
- GALLOP PERIC IS
- TAMP (CLICK SIM)
- VV OBSTRUCTION
-T
- ↓ PRELOAD (MAY)
- LIE (NARROWING
+ DYSPNEA
- AO DISS PERIPHERALLY
- HEART
- NEUROHORM ANOMALY
↓ BODY COMPOSITION ANALYSIS
↓ BP
CVP
FLUID STATUS
SCAN

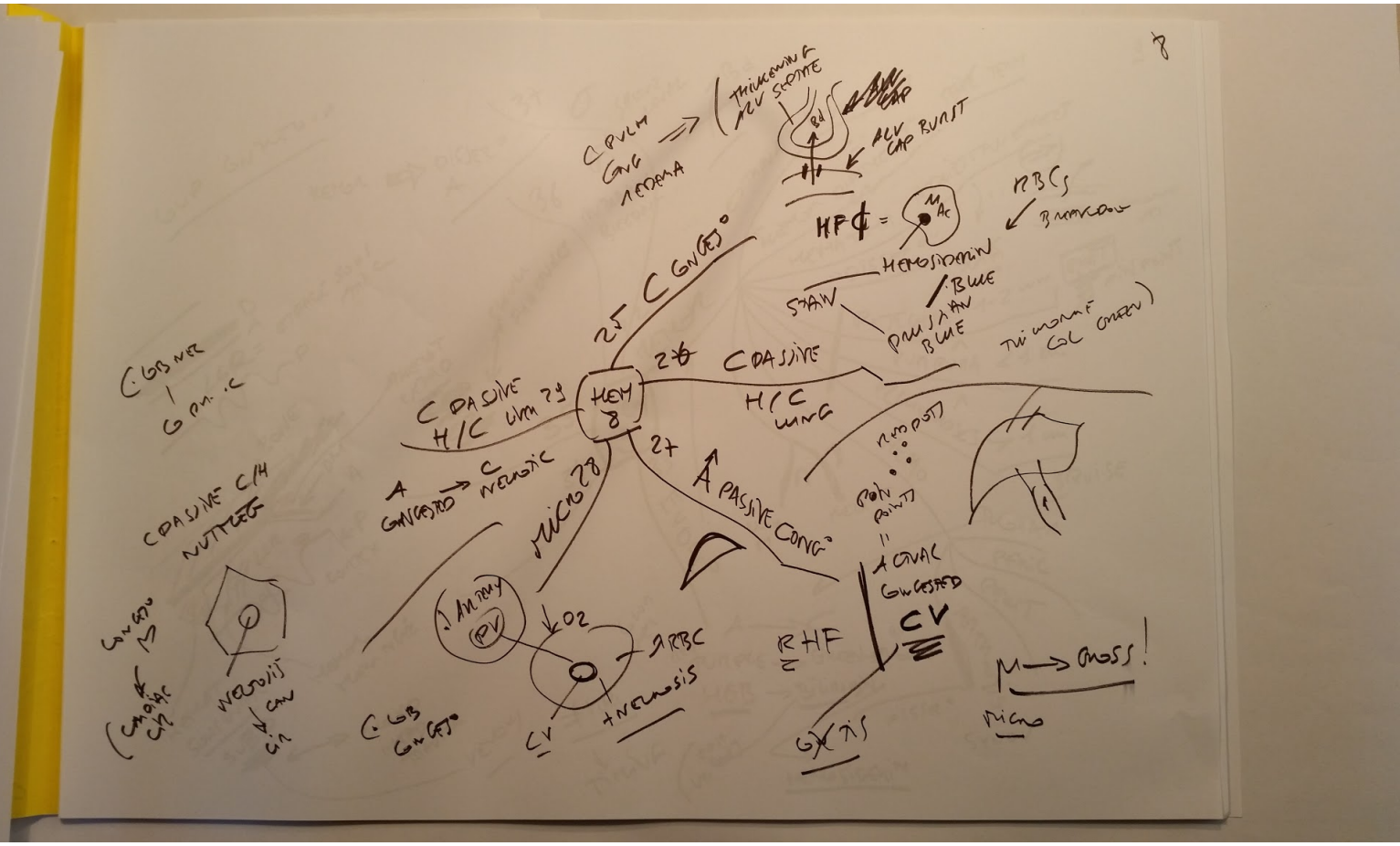

HEM 3
iFM
"EDEMATOUS"
ANGIOGENESIS
Ci
Ai
VT
PLASMA ONCOTIC P
P_Bd
Se-ALB
3 MAJ
EDEMA
VITAL ORGANS
LL OBSTRUC°
LYMPHEDEMA
iFM
NEOP
LOCOREG
POST IRRAD°
ANYTHING
PAIN
3 Na PETEN°
RENAL ↓ PERF°
RAAS
TUBUL REABS° Na
SALT INTAKE
RENAL INJ

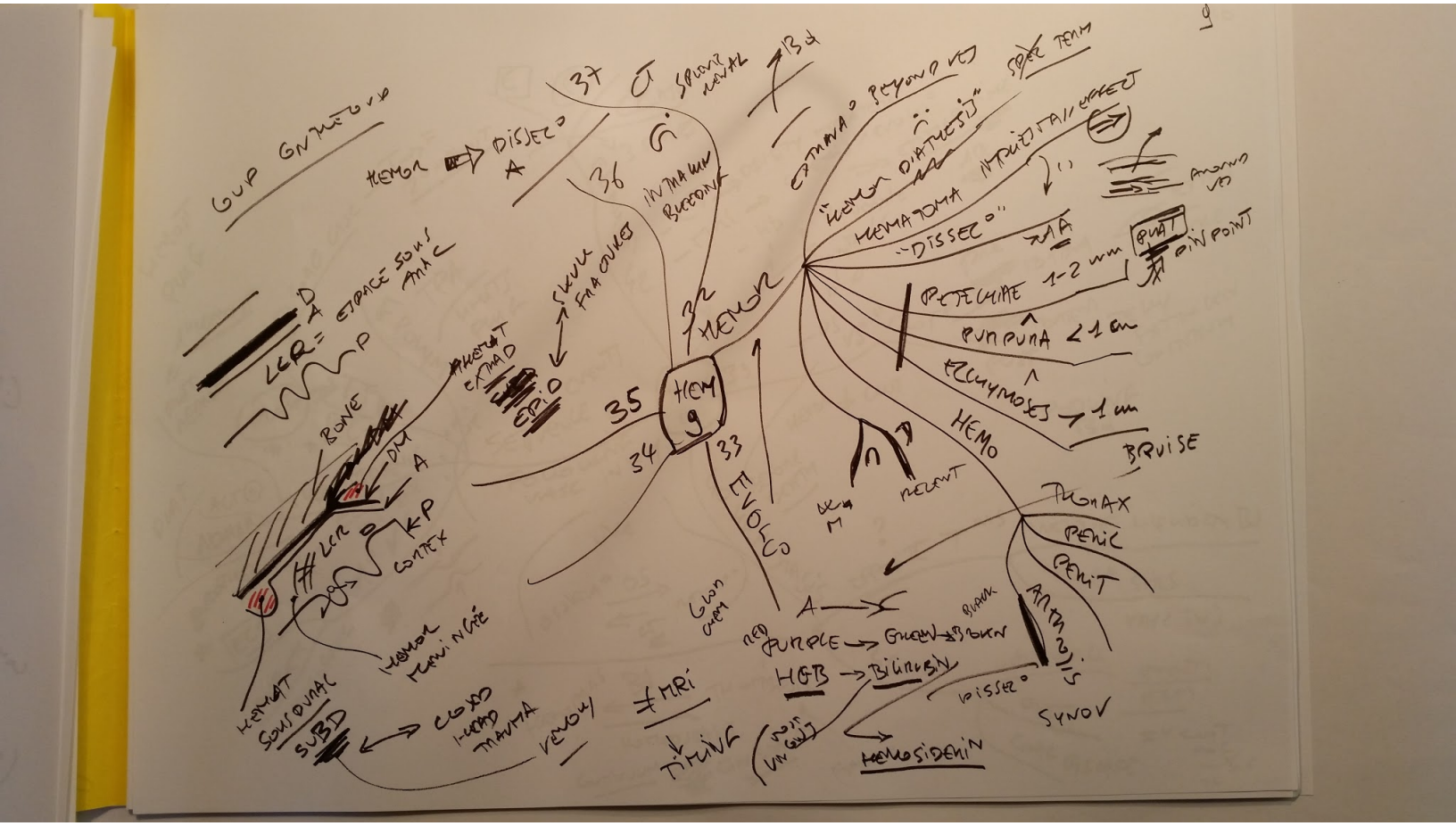

HCM
4
12 CHF EDEMA
↑ RVP ⇐ FAILURE
↓ RENAL PERF
11 BOTH
A-A-C
13 HEP ASCITES
RENAL
EDEMA
15
ASCITES
14
PORTAL HT
PLEURAL
"FLUID KANE"
LIVER
PORTAL
SYSTEMIC EDEMA
ALB
BOI
SMALL

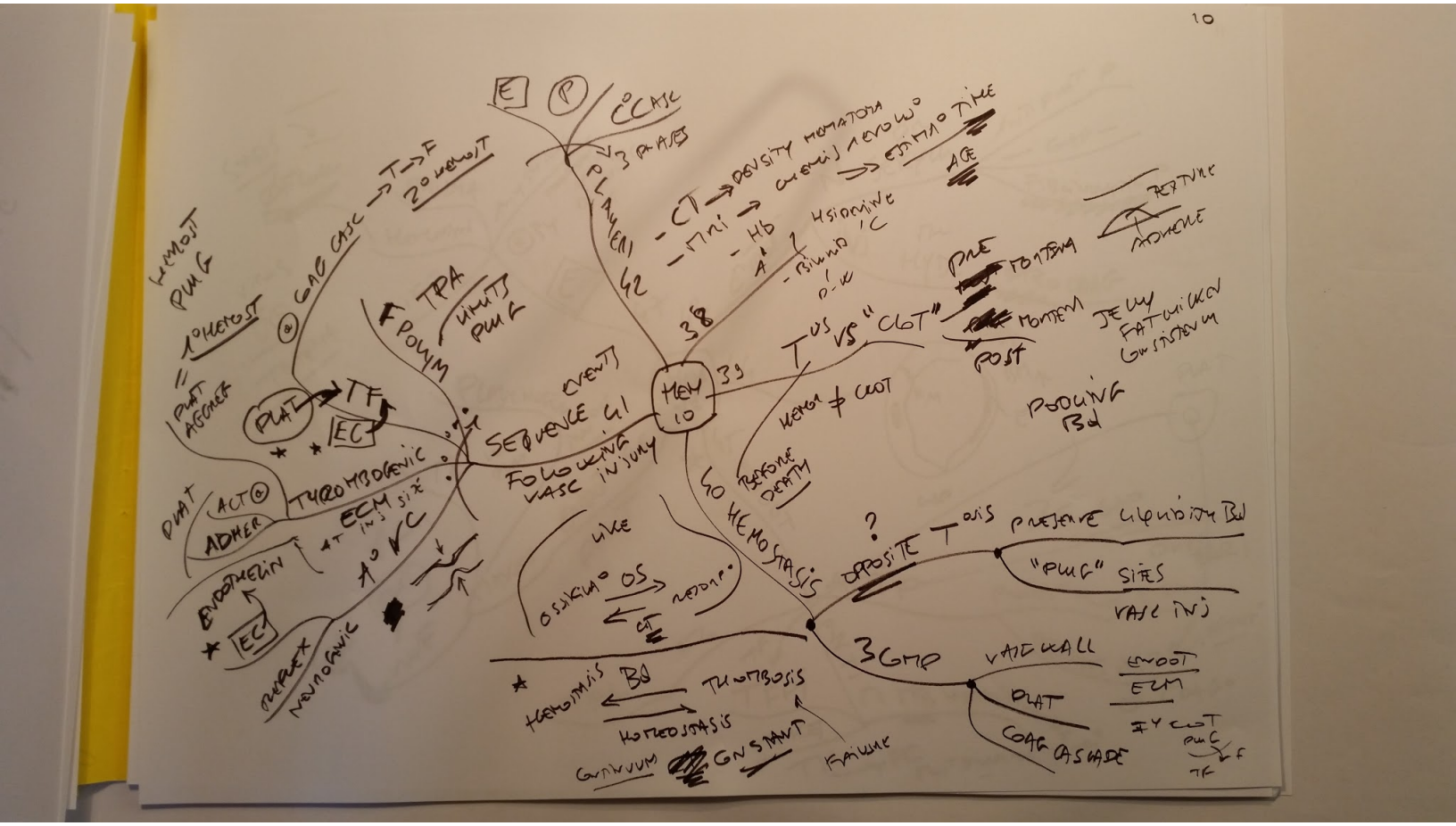

EXUDATE
TRANSUDATE
18
16 EDEMA
17
HEM 5
LVR HF?
SUBCUT "PITTING"
"DEPENDENT"
ANASARCA
CBR
RENAL
PULM
SUBFALCINE HERNIA
TRANSTENTORIAL
TONSILLAR
BASAL CISTERNS
30
SWELL LIKE

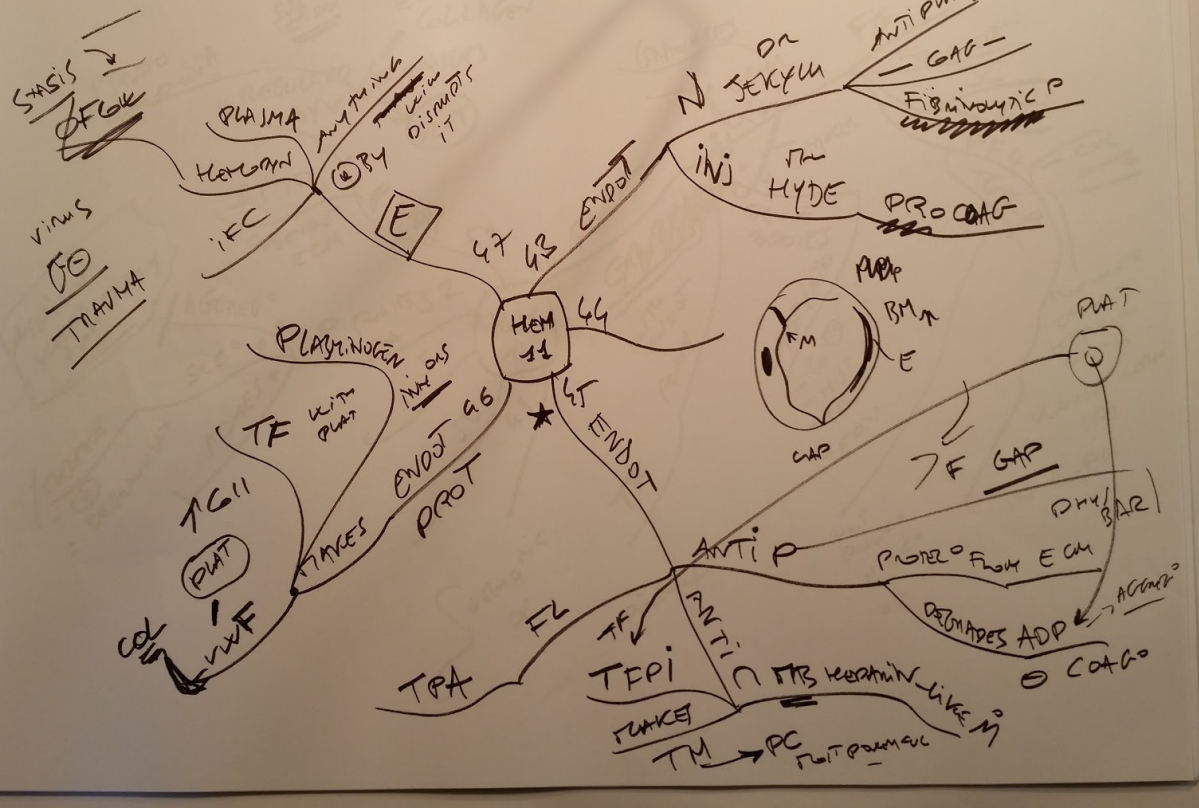

15K
7K
ARC
19 HYPEREMIA / CONGESTION
PASSIVE
PLUMBER
CONGEST°
21
EDEMA
HEM
6
ACTIVE
PROCESS
20
PASSIVE
EDEMA
CHF
LOCAL OBST°

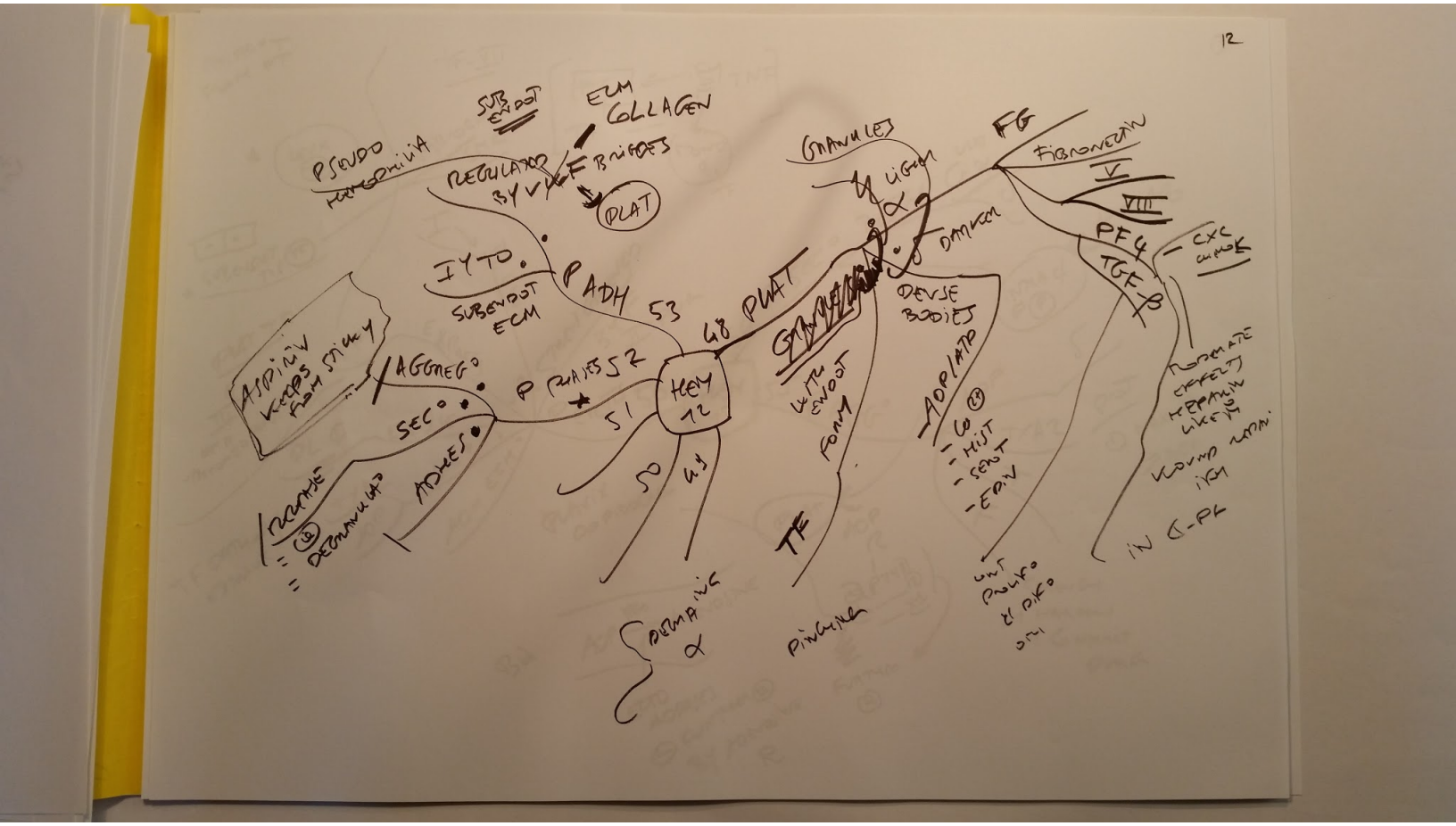

ALV EDEMA
"BAT'S WING"
KERLEY B LINES
INTERSTITIAL EDEMA
PLEURAL EFFUSION
22
ACUTE PASSIVE VENOUS/CAPILLARY LUNG
PRECEDES ALVEOLAR EDEMA
TRANSUDATE
CONGESTED ALV VESSELS
24
HCM +
23
AIR BRONCHOGRAM
KERLEY B LINES
KERLEY B
AIR BRONCHOGRAM
HORIZ PLEURAL BAND
ULTRASOUND DEFINITION
CAP PULM PRESSURE
28 mmHg
N 8-12
↳ MAXIMUM SAFETY
AT PCWP
> 18-20
→ EDEMA
"BAT WING"
12-18
MAXIMUM
FLUID
INTAKE

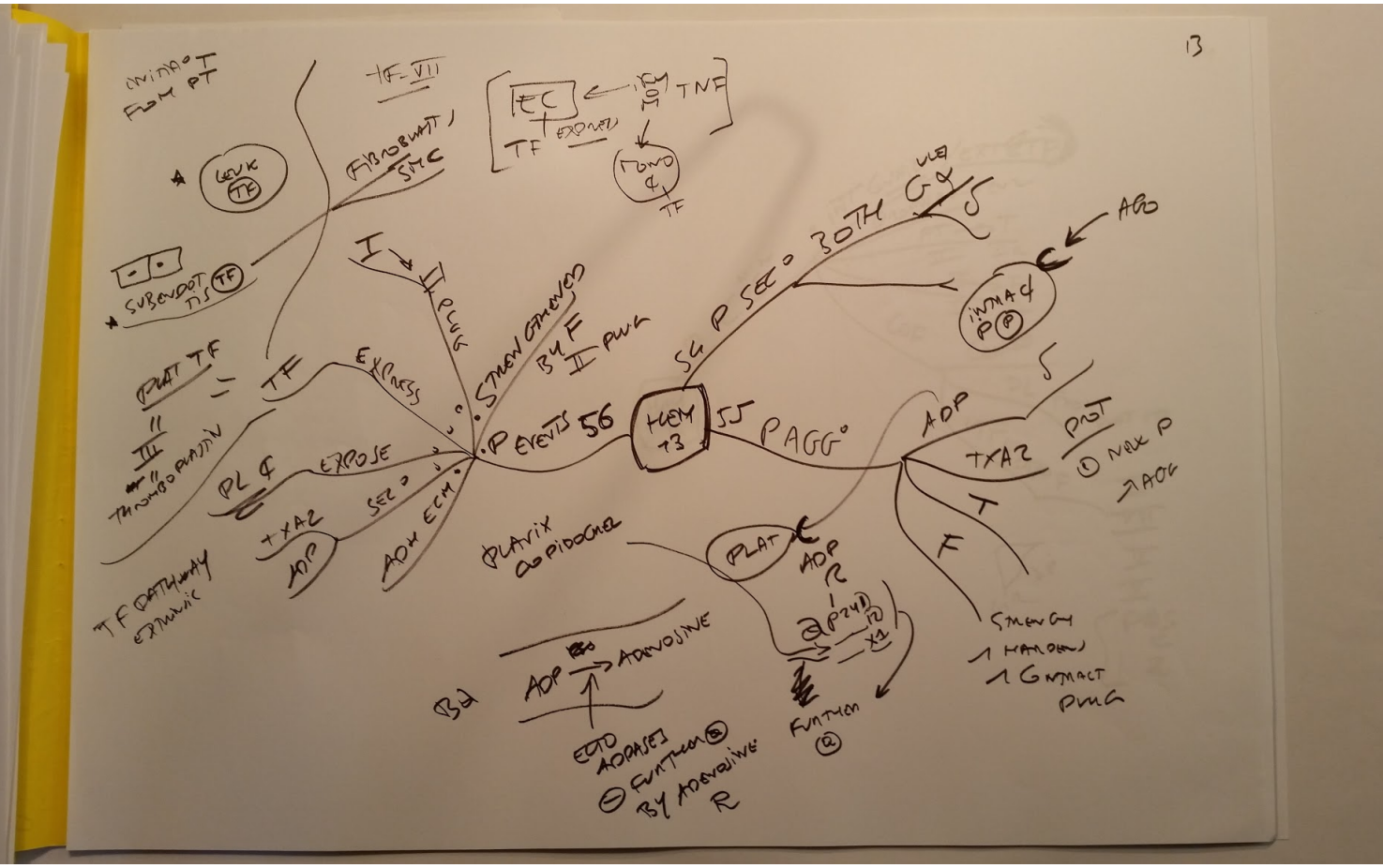

HEM
8
HPC
RBCs
STAIN
BLUE
PASSIVE
HIC
MICRO
ARTERY
PV
RBC
RHF
CV
GRAM
CAPILLARY
VENOUS
ARTERIAL
CV
MICRO

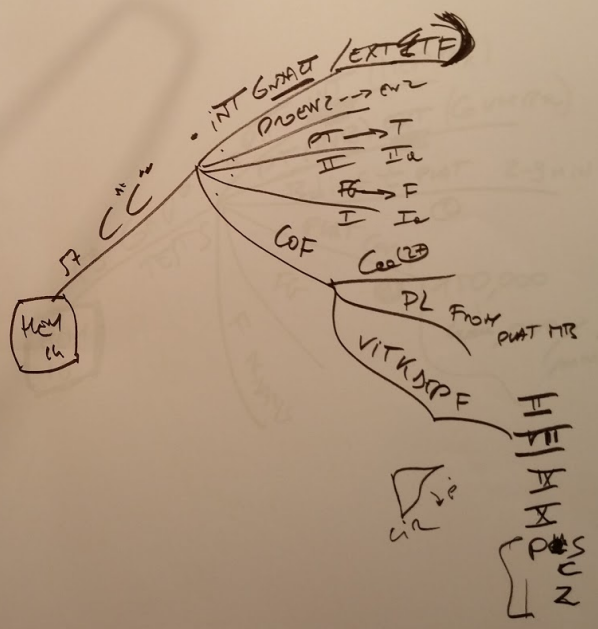

HEMO
DISSEC°
37
36
35
34
33
32
HEM
9
EVOLC
HEMATOMA
"DISSEC°"
PETECHIAE 1-2 mm
PURPURA < 1 cm
ECCHYMOSES > 1 cm
HEMO
BRUISE
THORAX
PERIC
PERIT
ASCITIS
SYNOV
HGB
Bilirubin
HEMOSIDERIN
ECCHYMOSES
PETECHIAE
PURPURA
HEMATOMA
SUBD
EPID
LCR
BONE
DM
KP
CONTEX
VENOU
≠ MRI
TITRAGE

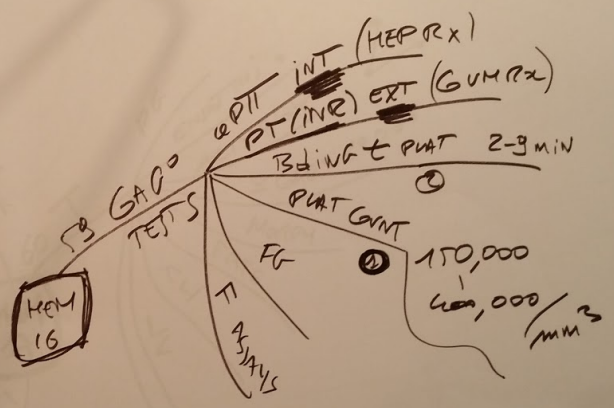

E
P
CCASE
3 PHASES
PLANNING
42
38
39
HEM
10
SEQUENCE 41
EVENT
FOLLOWING
BASIC INJURY
PLAT
TF
EC
ACT
ADHER
THROMBOGENIC
ECM
INJ site
AVC
ENDOTHELIN
PERIPH
NEUROGENIC
TPA
FIBRIN
LIMITS
PLUG
CAG CASC
T→F
2° HEMOST
HEMOST
PLUG
1° HEMOST
=
PLAT AGGREG
CT
PLATELET
Hb
A
Hsiderine
Bilirub
D-dimer
DENSITY MONITORING
CHANGE NEEDLE
ESTIMATE TIME
AGE
TOTHEM
PLAT
POST
RETRACT
MOMENT
JELLY FATTY ACID SYSTEM
POOLING
BD
T US
VS "CLOT"
HEMOST ≠ CLOT
BEFORE DEATH
LIVE
OSMOL°
OS
CT
TO HEMOSTASIS
OPPOSITE
?
T axis
PREVENT
"PLUG" SITES
BASIC INJ
3 COMP
WALL
PLAT
COAG CASCADE
SMOOT
ECM
IF CLOT
PLUG
TF
HEMOSTASIS
BD
THROMBOSIS
HOMEOSTASIS
CONTINUUM
CONTINUUM
FAILURE

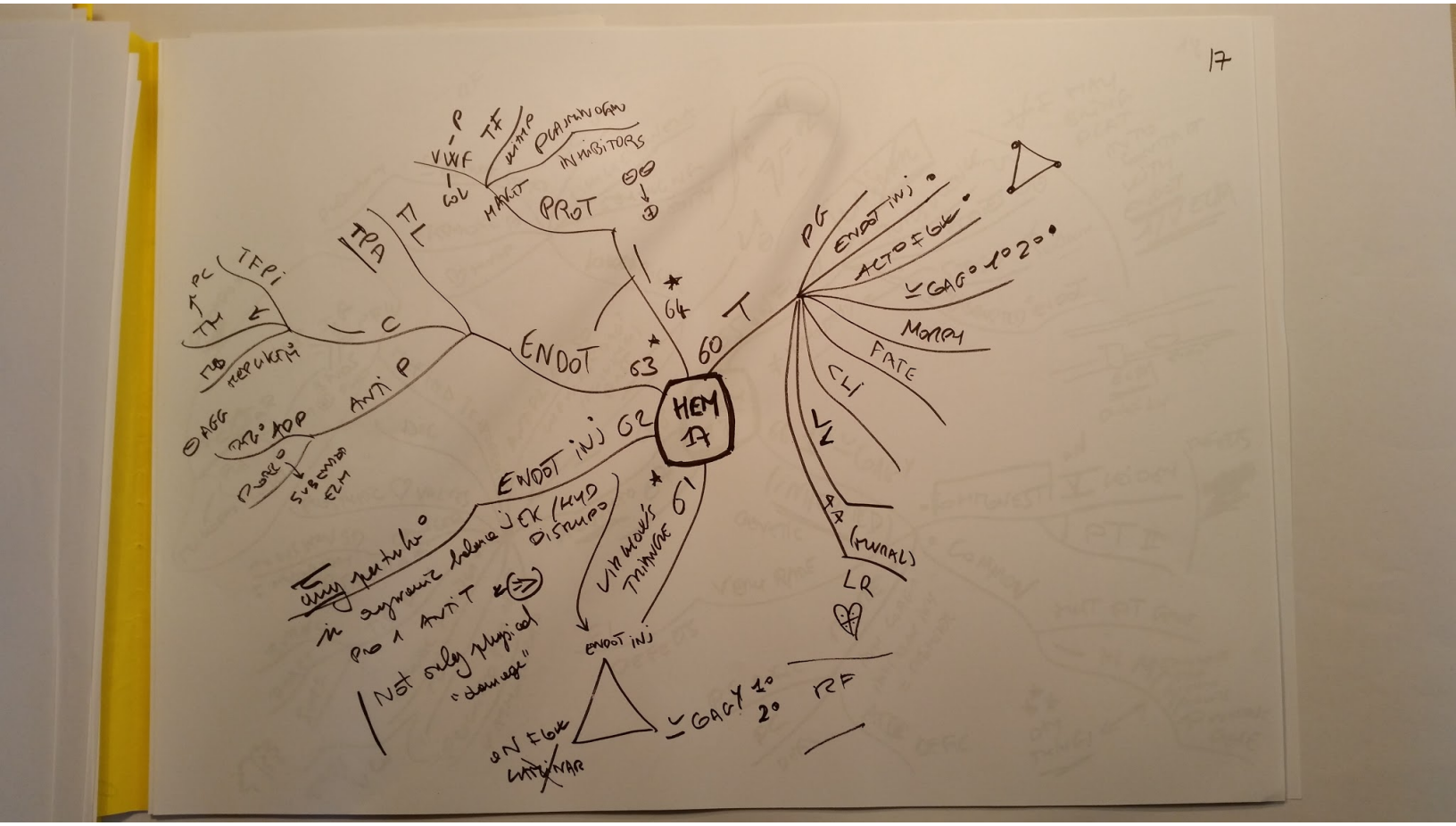

STASIS
VIRUS
TRAUMA
PLASMA
HEPARIN
iFC
E
47
43
ENDOT
HOM 11
44
DR JEKYLL
ANTI PLAT P
GAG
INJ
HYDE
PRO COAG
PLASMINOGEN
TF
TAKES
ENDOT PROT
TG11
PLAT
VWF
46
ANTI P
PLATELET FLOW ECM
DEGRADES ADP
COAG
TPA
TFPi
TM
PC
PLAT
GAP
BM
FM
E

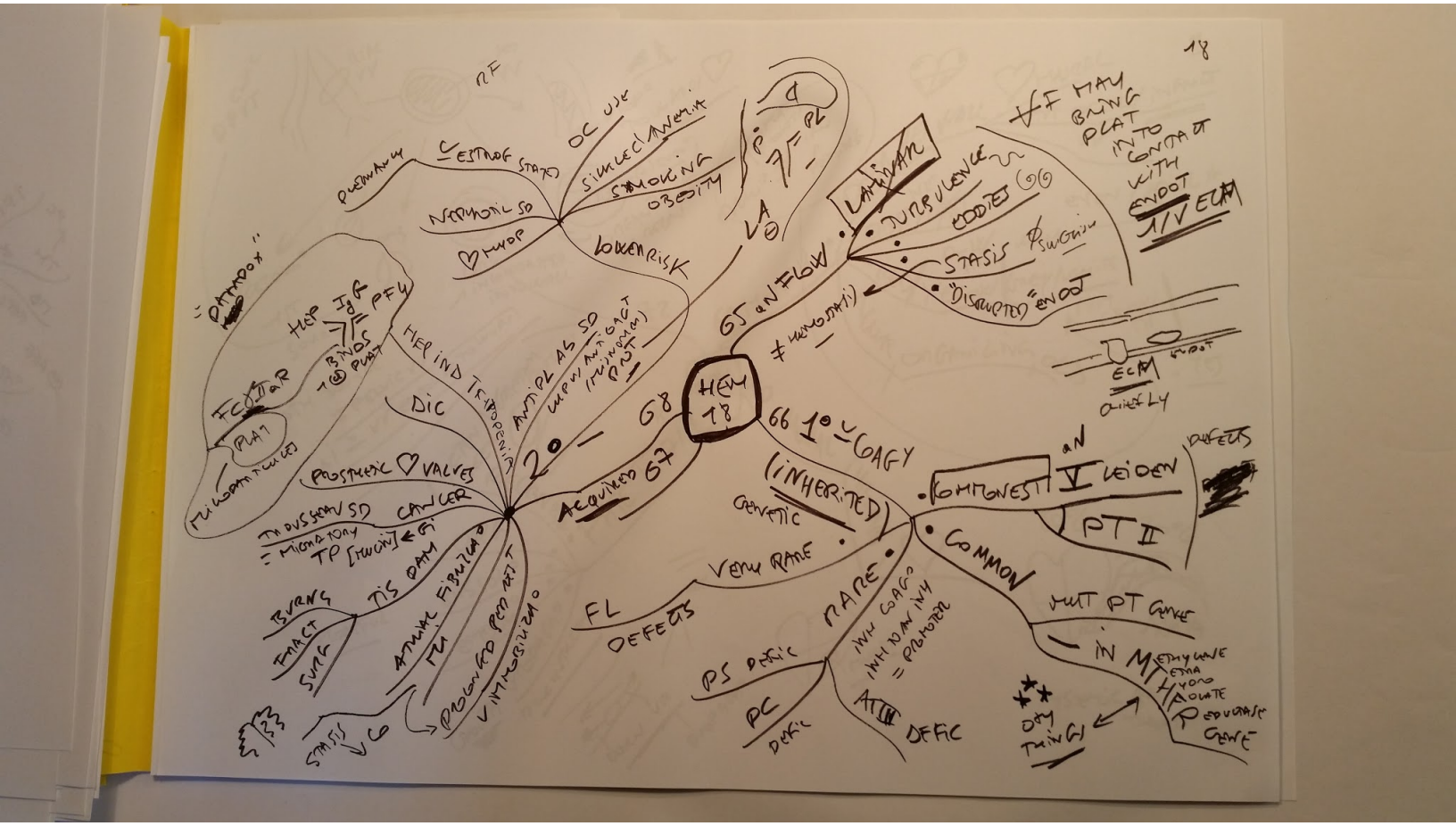

PSEUDO
SUB ENDOT
ECM
COLLAGEN
REGULATED
PLAT
IYTO.
SUBENDOT ECM
PADH 53
48 PLAT
GRANULES
LIGHT
DAMAGE
DENSE BODIES
FG
FIBRONECTIN
V
VIII
PF4
CXC CHEMOK
TGF β
ADP/ATP
AGGREG°
SEC°
ADHES°
P PHASE S2
S1
S0
G1
KEY T2
TF
PINCHING
DERMA
ENDOT FORM
HEPARIN
LIKE
IN G-PL

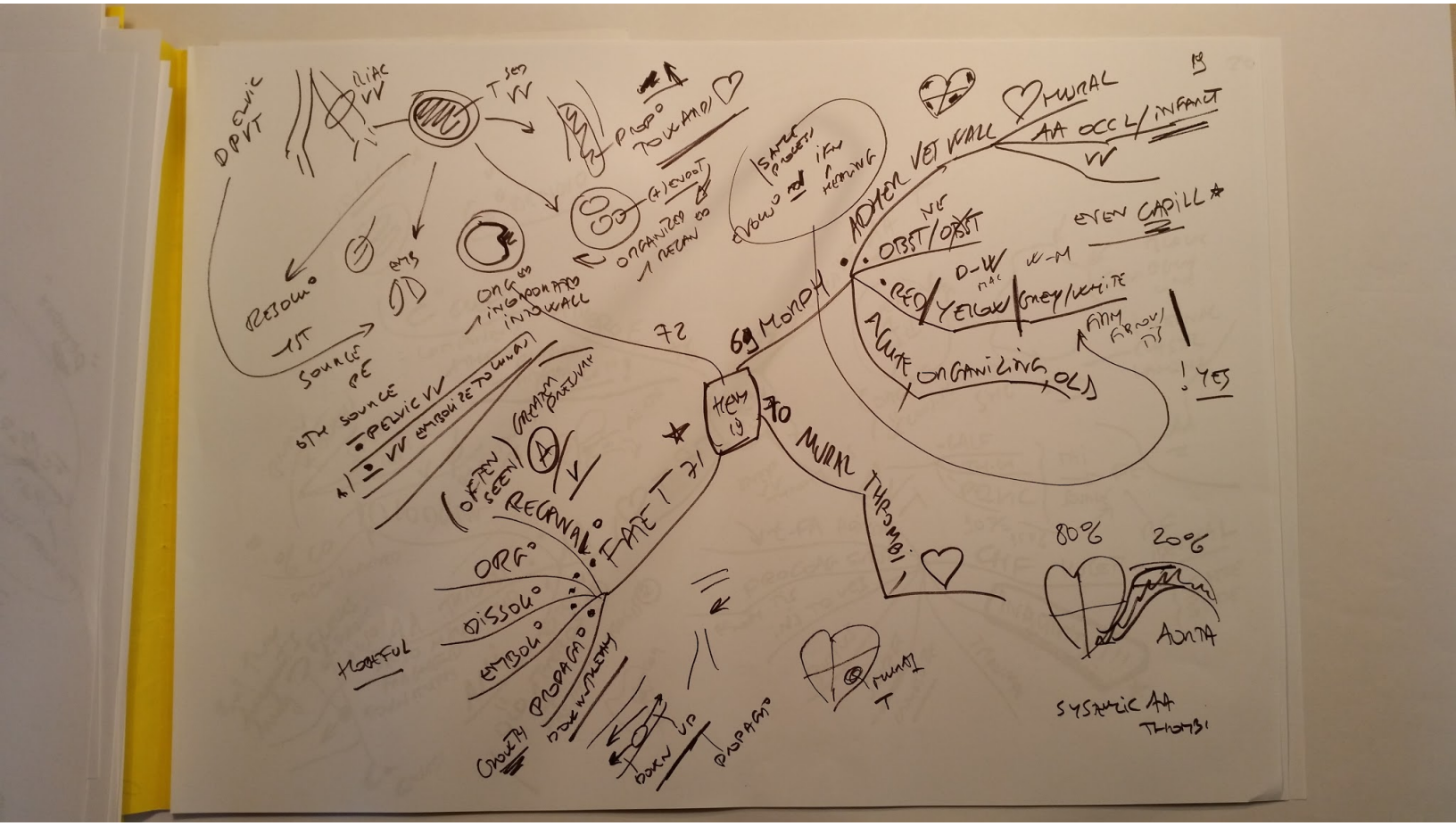

TF-VII
TNF
TF
SUBENDOT
TF
TF
EXPRESS
EXPOSE
PL
TXA2
ADP
SEC
ADH ECM
PLATELET
ADP
ADENOSINE
ECTO
ADP
TXA2
PGI
PLAT
ADP
SMOOTH
MUSCLE
CONTRACT
PLUG
SEC
AGG
INTIMA
ADP
PAGG

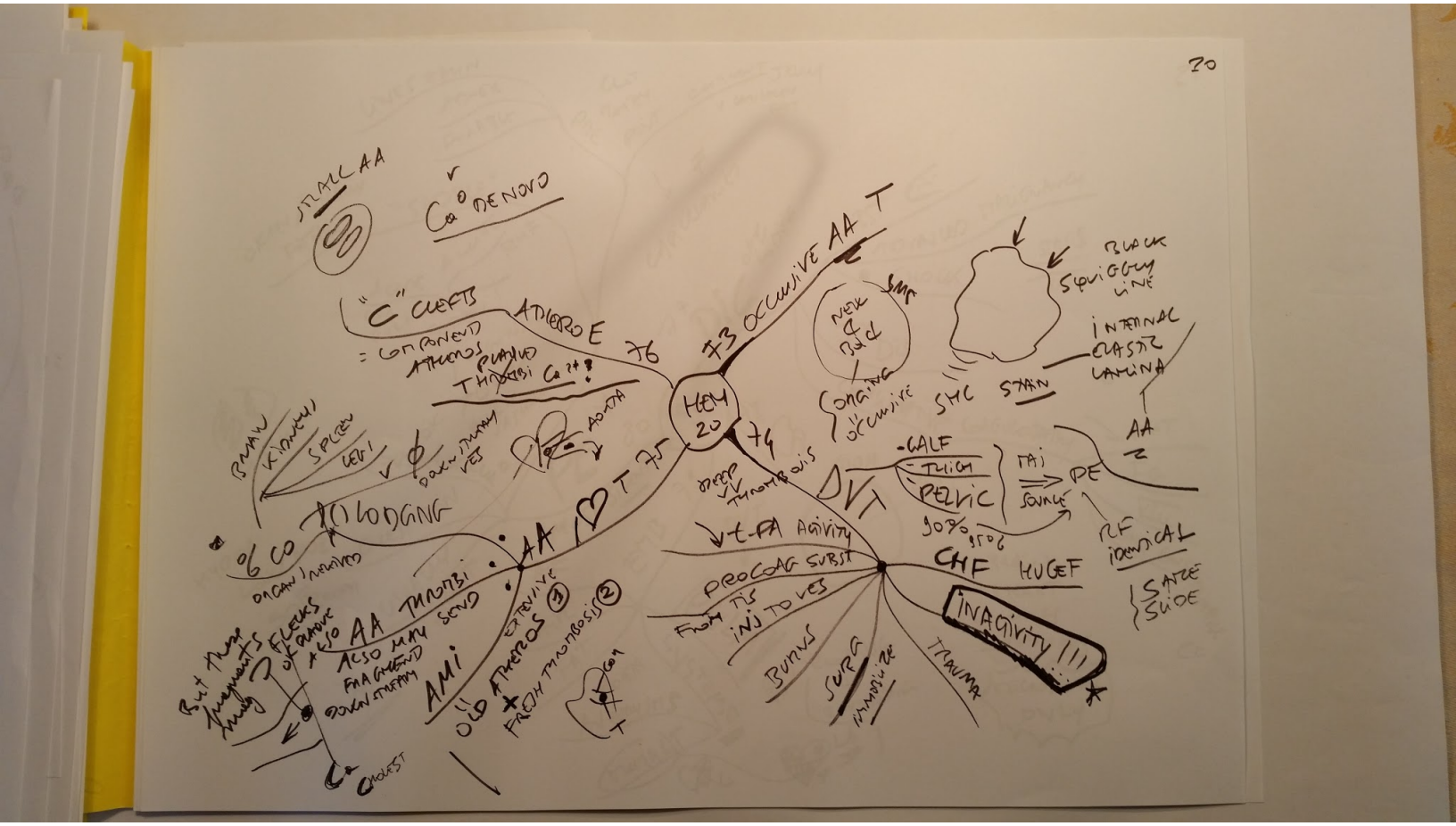

INT GMAT
COF
PL FROM
VITK
II
I
II
I

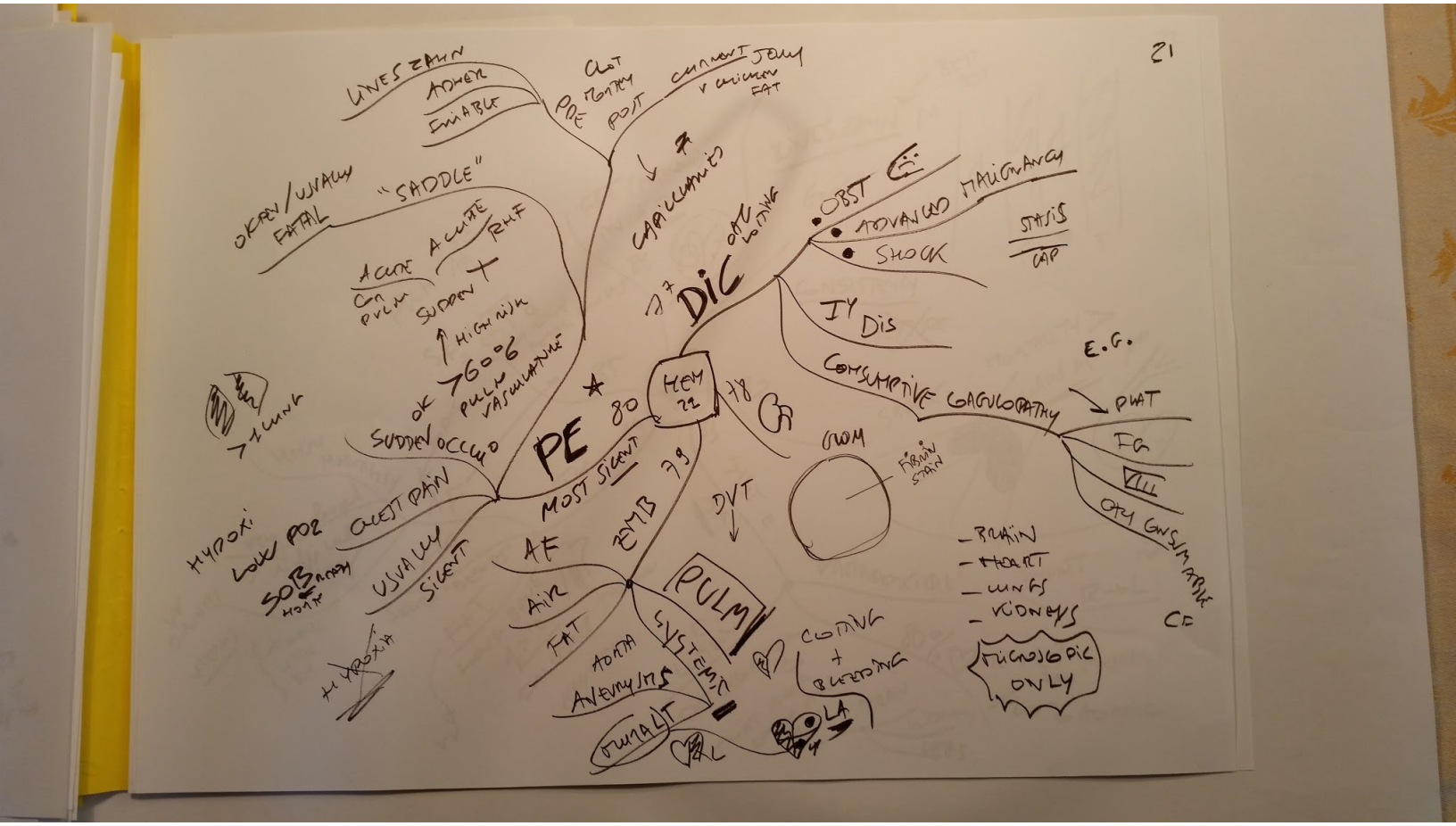

HEM
16
COAG
TESTS
aPTT
INT (HEPRx)
PT (INR) EXT (GUMRx)
Bleeding PLAT 2-9 MIN
PLAT CNT
FG
TT
ASSAYS
150,000
400,000/MM3

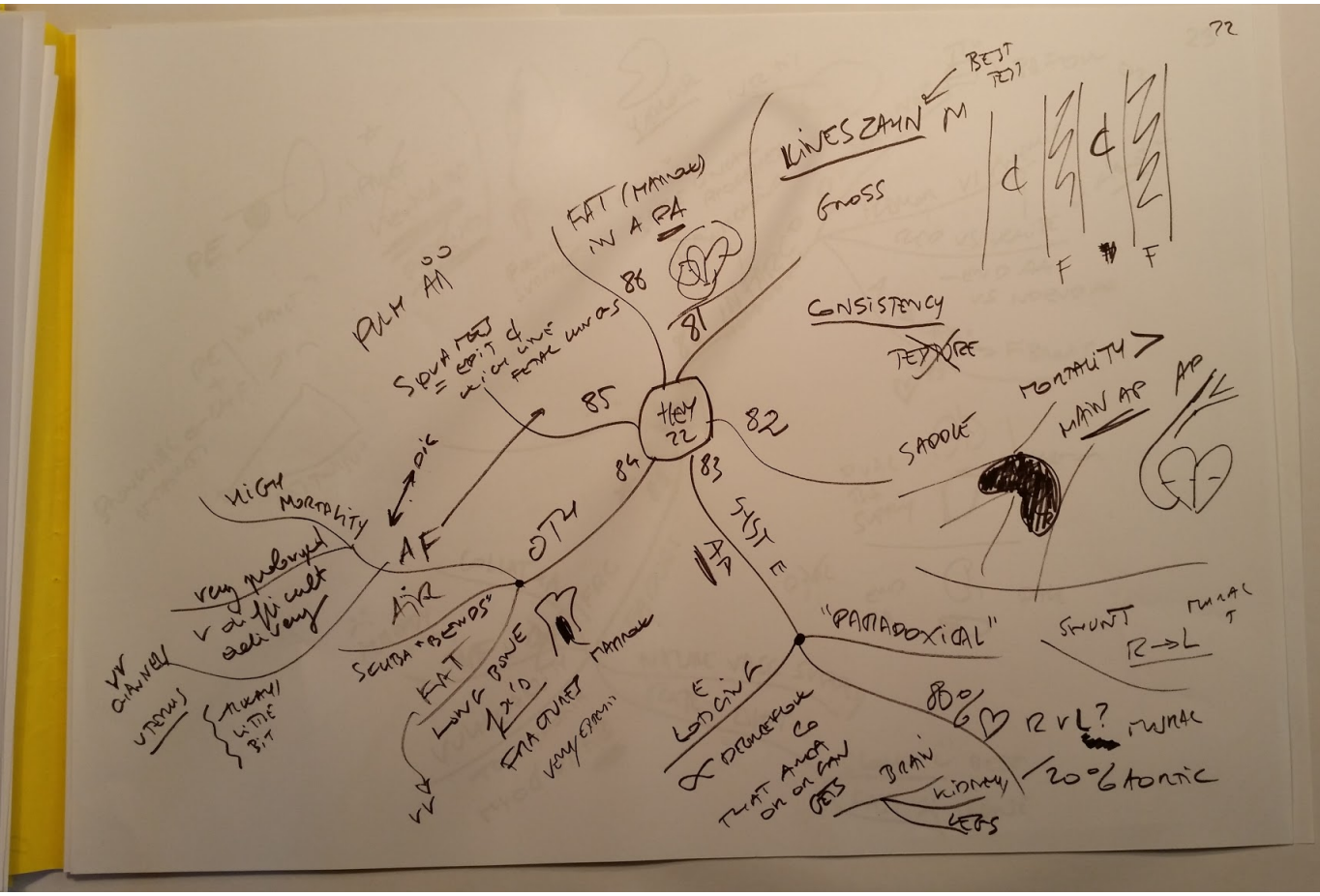

HEM
1ª
PROT
ENDOT
ANTI P
ANTI T
PG
LR
RA
TL
TFPi
TF
PLASMINOGEN
INHIBITORS
VWF
GOL
MARGT
ENDOT INJ
ACTO FGE
MORPH
FATE
CLI
LVL
(RENAL)
ENDOT INJ
ANTI ADP
AGG
PC
TM
TRP
ANTI T
LR
RA

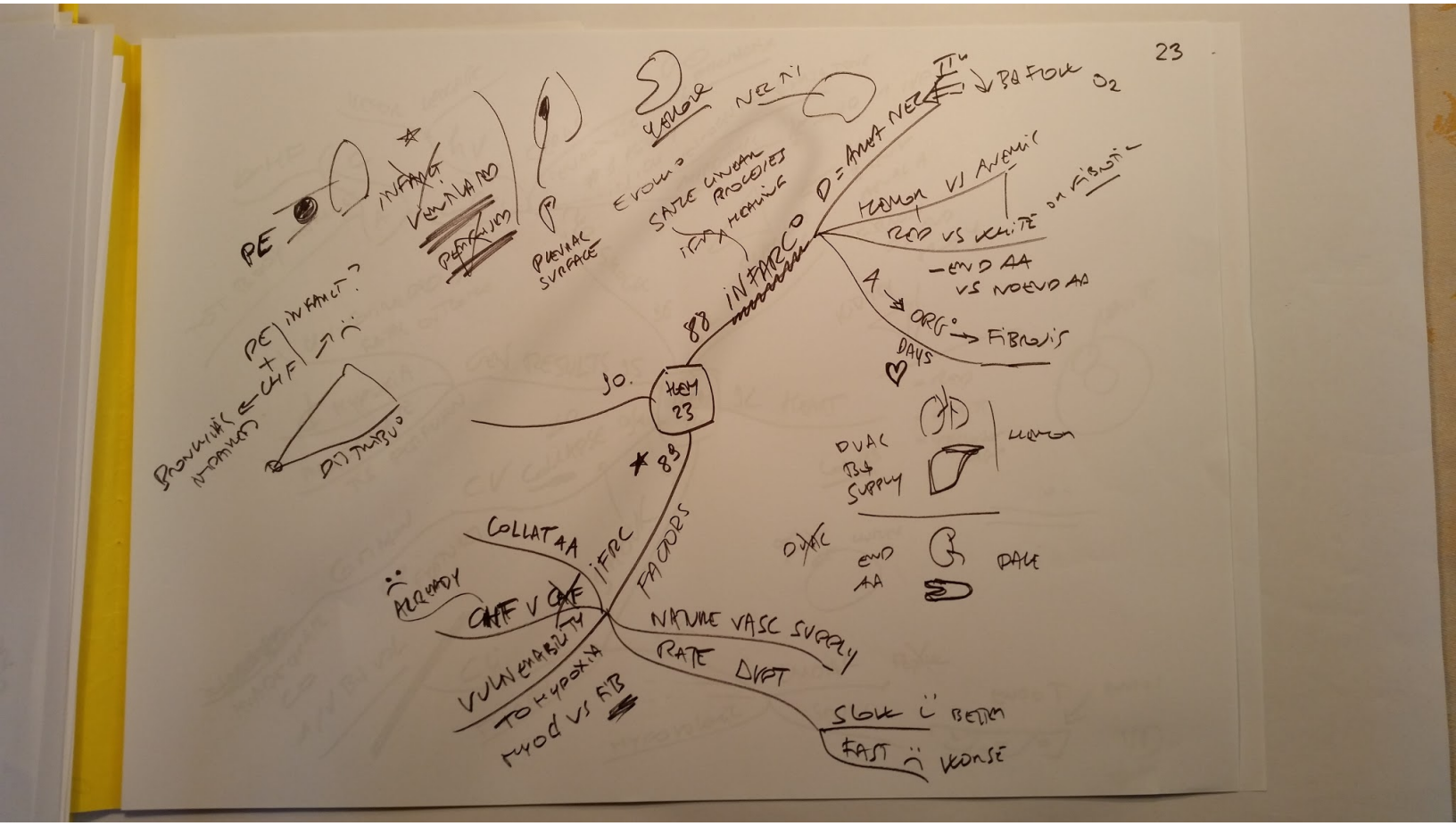

HEM
18
65 aN FLOW
66 1° COAGY
62
67
LOW RISK
ACQUIRED
(INHERITED)
GENETIC
COMMON
RARE
VERY RARE
FL
DEFECTS
PS DEFIC
PC
DEFIC
ATIII DEFIC
PT II
V LEIDEN
aN
DEFECTS
HYPERCOAGULABLE
THROMBOPHILIA
ANTIPL AB SD
DIC
PROSTHETIC VALVES
CANCER
TIS DAM
ATRIAL FIBRILLATION
PE
PROLONGED IMMOBILITY
STASIS
VD
BYPASS
INFARCT
SURG
PREGNANCY
ESTROG STATS
OC USE
SILICLE ANEURYM
SMOKING
OBESITY
NEPHROTIC SD
HHCP
LA
PL
LAMINAR
TURBULENCE
EDDIES
STASIS
"DISRUPTED" ENDOT
HF MAY BRING PLAT INTO CONTACT WITH ENDOT
I/V ECM
ECM
ENDOT
OLIVER LY
HEP
IgG
PF4
HEPARIN
PLAT
ECMAP
"ANTIBOX"
18

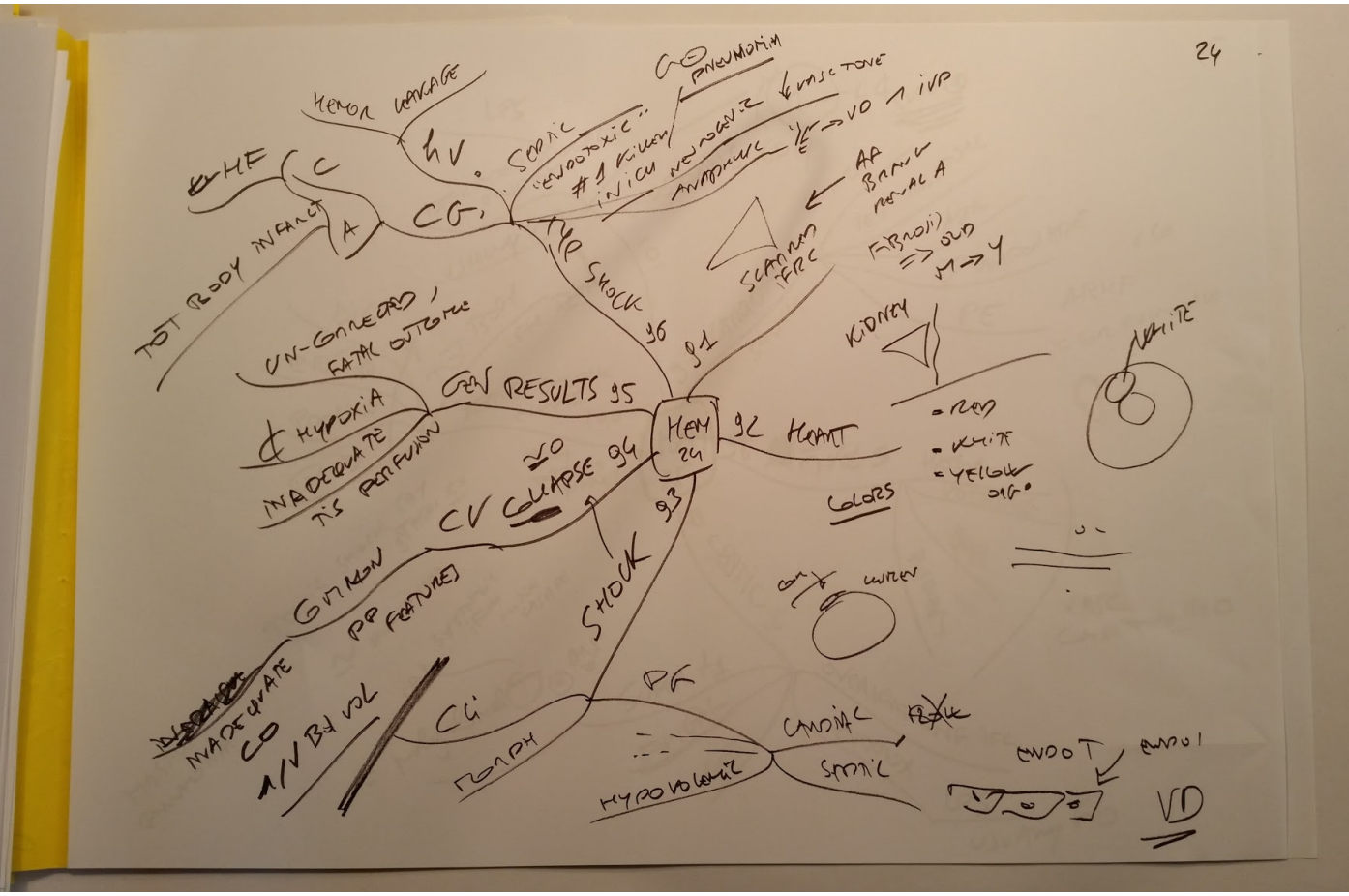

PELVIC
DPVT
ILIAC
VV
T SEG
VV
PHLEBO
THROMBOSIS
ORGANIZED
RECAN
RESOLV
IT
SOURCE
OF
6TH SOURCE
PELVIC VV
EMBOLIZE TO LUNG
72
69 MONTH
ADMIT VEIN WALL
OBST/OBST
RED/YELLOW/GREY/WHITE
EVEN CAPILL
NUMB ORGANIZING
YES
HEM
19
70 MURAL THROMBI
(OFTEN SEEN)
RECANAL
FATE T 71
OPP
DISSOLV
EMBOLI
PROPAGATE
GROWTH
DOWN VP
PROPAGATE
PULM
T
80%
20%
AORTA
SYSTEMIC AA
THROMBI

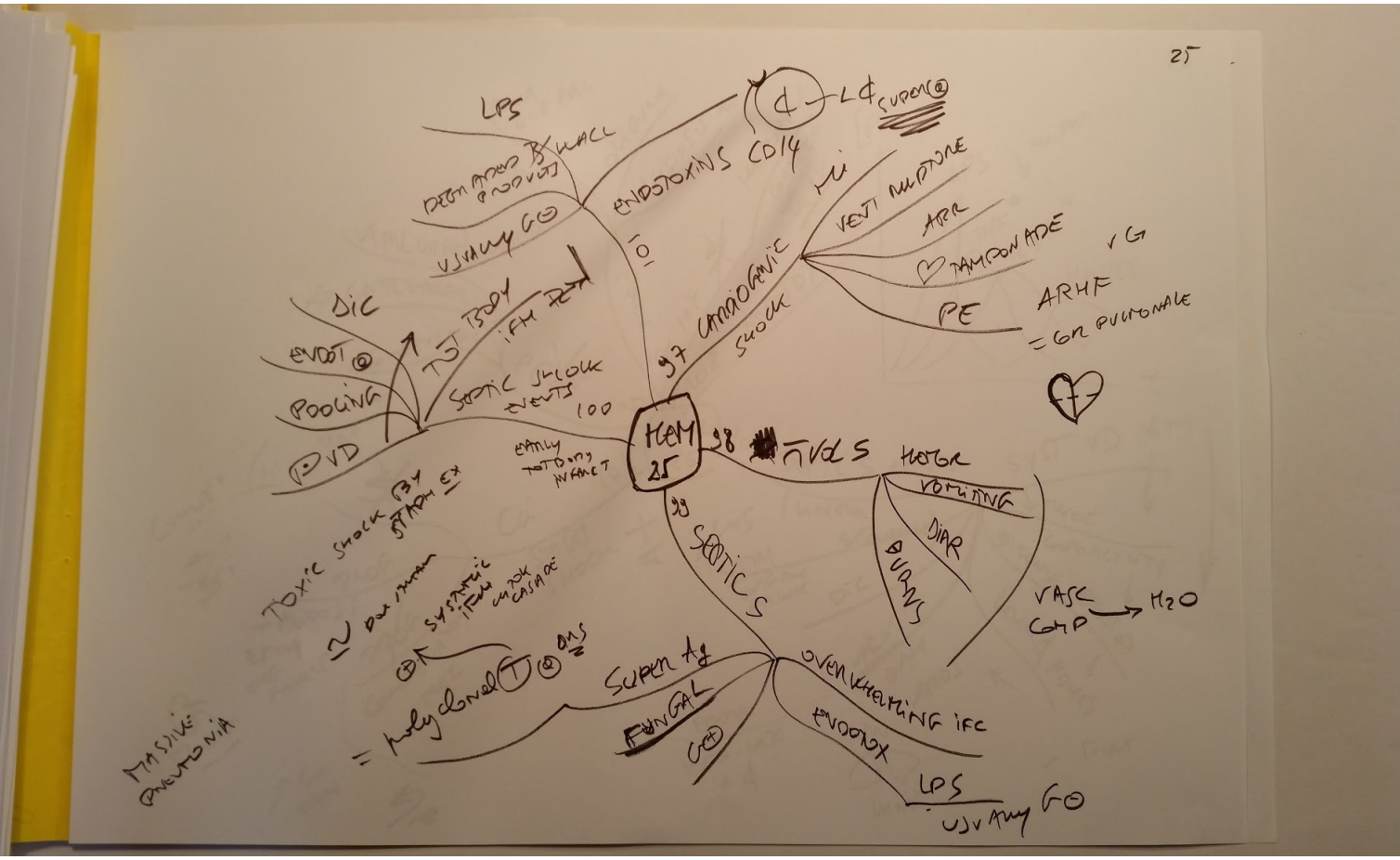

HEM 20
"C" CLEFTS
ATHEROSCLEROSIS
= CONTAINED ATHEROMA
PLAQUE THROMBOSIS
76
73 OCCLUSIVE AA T
NEW AF
ONGOING OCCLUSIVE
SMC
SMA
SMA
BLACK SQUIGGLY LINE
INTIMAL CRACK LAMINA
AA
PE
DVT
-CALF
-THIGH
-PELVIC
PAi
SOURCE
90%
RF IDENTICAL
SAME SLIDE
CHF
HUGE
INACTIVITY!!!
PROCOAG SUBST
FROM TIS INJ TO VES
BURNS
SURG
TRAUMA
74
DEEP IV THROMBOSIS
Vt-PA ACTIVITY
AA
AMI
ATHEROS
THROMBOSIS
ACUTE
CHRONIC
FRESH THROMBOSIS
OLD ATHEROS
ACUTE MAY SOMO FRAGMENTED DOWNSTREAM
FLEXUS OF ARTERIES
But these fragments usually?
% CO
ORGAN DAMAGE
ACTIVATION
SPLEEN
KIDNEYS
BRAIN
VEIN
ARTERY
DOWNSTREAM VEIN

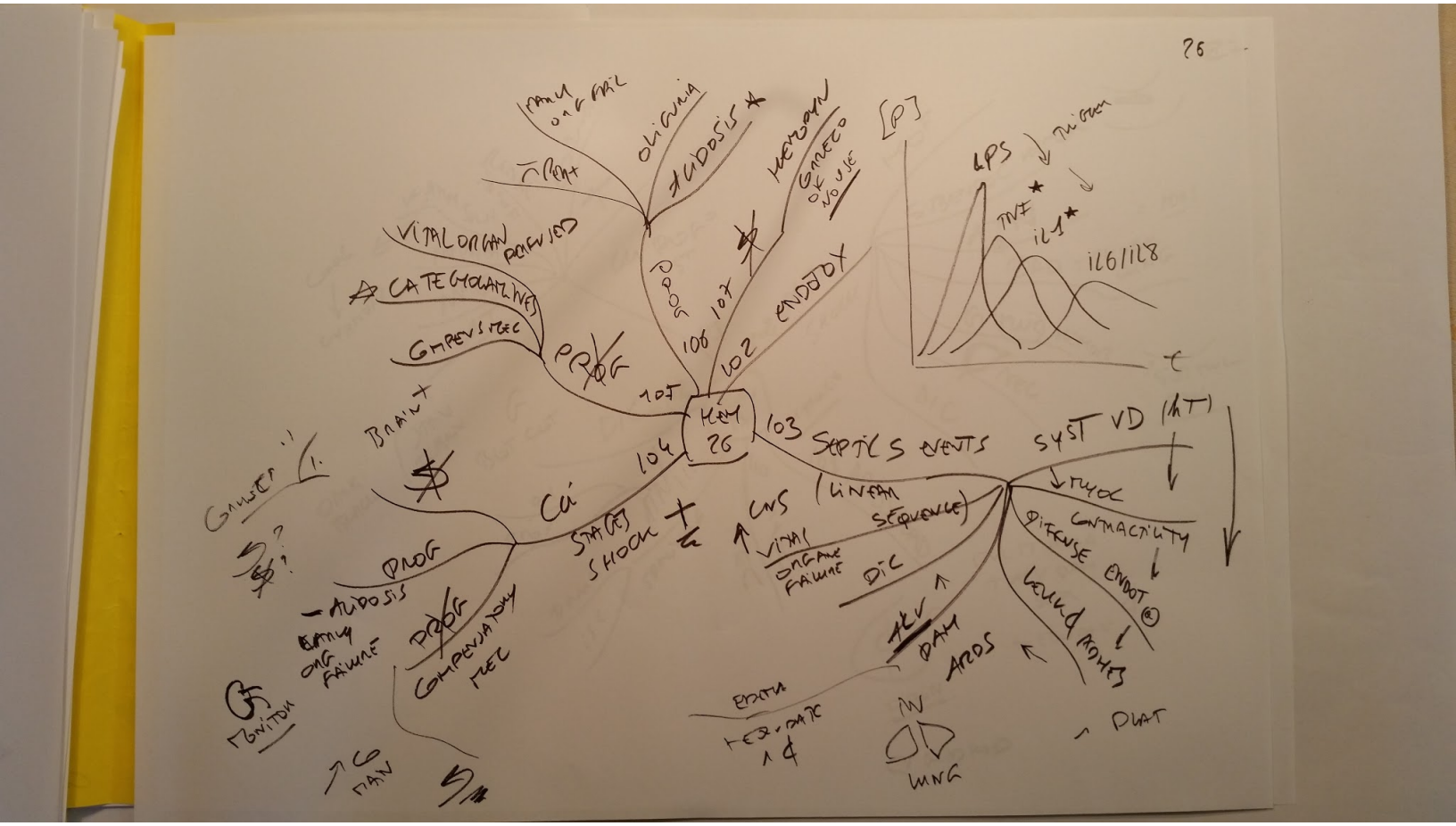

21
PE
DIC
HEM 22
80
78
79
CR
MOST SILENT
DVT
PULM
SYSTEMIC
CLOTTING + BLEEDING
CROM
FIBRIN STAIN
E.G.
PHAT
FG
OTH CONSUMPTIVE
CONSUMPTIVE COAGULOPATHY
OBST
ADVANCED MALIGNANCY
SHOCK
STASIS
IV DIS
SUDDEN OCCLUD
CHEST PAIN
USUALLY SILENT
AF
AIR
FAT
AORTA
"SADDLE"
ACUTE
RHF
SUDDEN
HYPOX
LOW PO2
SOB
BRAIN
HEART
LUNGS
KIDNEYS
HISTOLOGIC ONLY

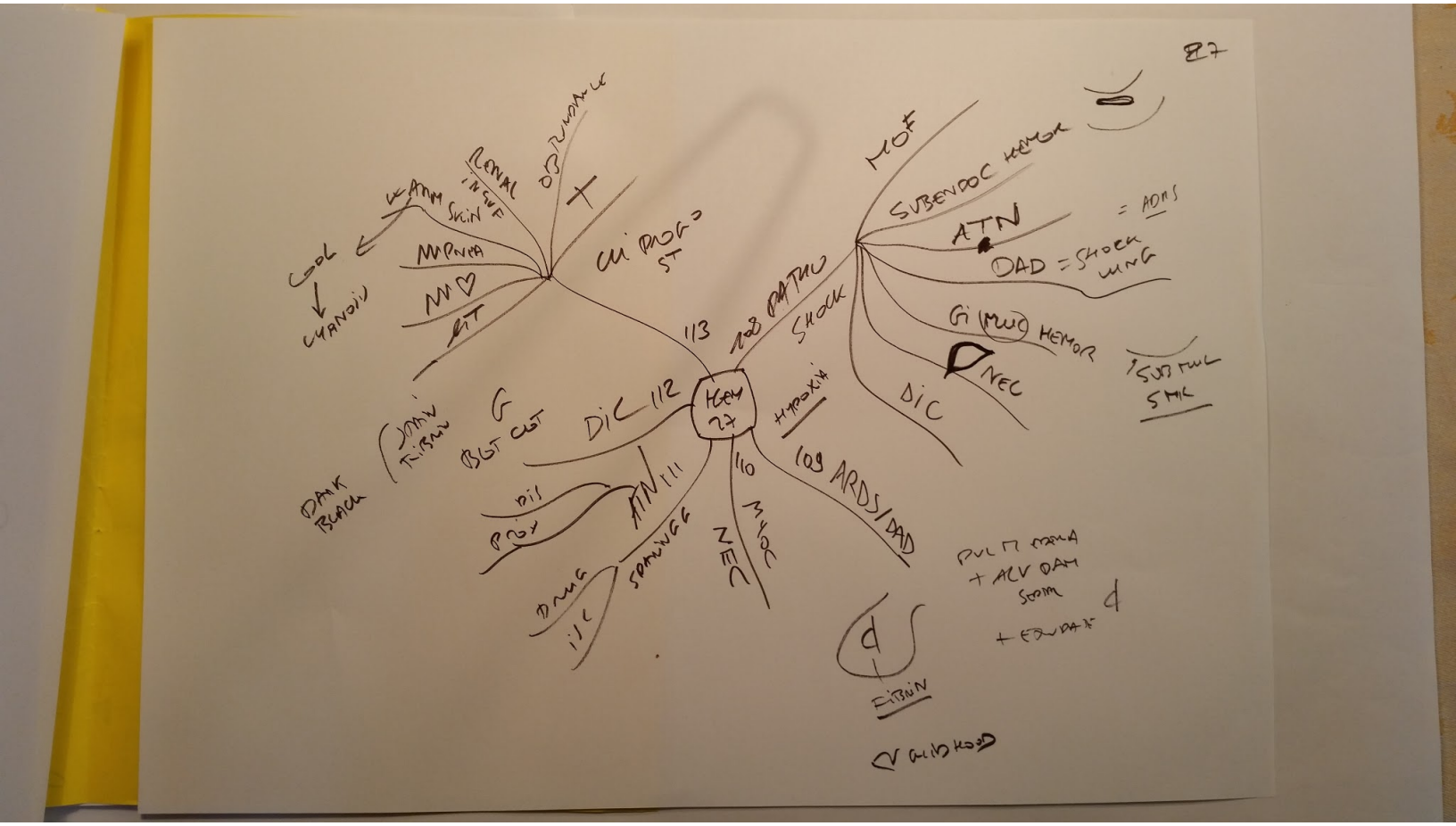

BEST TEST
KLIVESZAHN M
GROSS
F
F
FAT (MANUAL) IN A PA
86
81
POLY AN
SOLVATED = EPIT of LIVE FEMAL LIMBS
85
CONSISTENCY
TEXTURE
MORTALITY
MAIN AP
AP
HEM 22
82
SADDLE
HIGH MORTALITY
AF
84
OTH
83
SYST E
AP
VERY QUALIFYD
V difficult delivery
AIR
SEGMENTS
FAT
LONG BONE
FRACTURED
"PARADOXICAL"
SHUNT
R→L
VV CHANNEL
VTONUS
E LOADING
80%
R V L?
PULMAC
20% ATONIC
BRAIN
KIDNEY
LEGS


PE
INFARCT
VENTILATING
PERFUSION
PLEURAL SURFACE
EVOLVE
SAME CHEMICAL PROCESSES
ITS MECHANIC
88 INFARCT
D = ANAEMIA
HEMOGLOBIN
RBC VS RETICS
-ENDO AA VS NOENDO AA
4 → ORG° → FIBROSIS
DAYS
JO.
HOW 23
88
COLLATAA
ALREADY
CAFF V CAF
VULNERABILITY TO HYPOXIA
MYOCYTES
INFARC
FACTORS
NATURE VASC SUPPLY
RATE DEPT
SLOW
FAST
DUAL BLD SUPPLY
END AA
PALE
PE + CAF
INFARCT?
PROXIMAL OBSTRUCTION
DISTRIBUTION


HEMOR HEMORRHAGE
HV
SEPTIC
NEUROTOXIC
#1 KILLER
INITIAL NEUROGENIC
ANAPHYLAXIS
PNEUMONIA
HF
C
A
CO
TOT BODY INFARCT
SHOCK
96
91
SCAMMON
AA
BAND
NORMAL A
FIBROID
OLD
KIDNEY
UN-CONTROLLED
FATAL OUTCOME
HYPOXIA
INADEQUATE
TIS PERFUSION
CRV RESULTS 95
HEM
24
92
HEART
= RED
= WHITE
= YELLOW
VO
CV COLLAPSE 94
93
COLORS
SHOCK
GITMAN
PP FEATURES
CI
PE
RUPTH
HYPOVOLEMIC
SEPTIC
CARDIAC
ENDO T
VD


25
LPS
ENDOTOXINS CD14
LC
SUPEN
VENT MALFUNCTION
ARR
TAMPONADE
PE
ARHF
= GR PULMONALE
CARDIOGENIC SHOCK
SEPTIC SHOCK EVENTS
100
TCJ BODY
IFM RX
DIC
ENDOT
POOLING
VD
TOXIC SHOCK BY STAPH EX
SYSTEMIC ITEM
= hydrocortisone
SUPEN Ag
FUNGAL
OVERWHELMING IFC
ENDOTOX
LPS
VJV ALWAYS GO
VASC
GMP
H2O
VOMITING
DIAR
BURNS
HEMORR
MASSIVE PNEUMONIA


[P]
LPS
TNF
IL1
IL6/IL8
t
ENDOTOX
HEM
26
SEPTICS EVENTS
SYST VD
CVS
STARTS SHOCK
DIC
ARDS
DIFFUSE ENDOT
CONTRACTILITY
PROC
COMPENSATORY MEC
BRAIN
RENITON


MOF
SUBENDOC HEMOR
ATN
= ADNS
DAD = SHOCK LUNG
GI (MUC) HEMOR
NEC
DIC
SUST MUC SMC
108 PATHO SHOCK
HYPOXIA
109 ARDS/DAD
110 MyoC
NEC
DIC 112
HGM 77
113
CV PROG ST
RENAL INSUF
OLIGURIA
MAPNIA
MI
RT
COOL
CYANOSIS
DARK BLACK
BLST CLOT
DIL
PROX
ATN 111
DRUG
ILL
SOMNOLG
PULM EDEMA
+ ACU DAM
SOBR
+ EQUIPMENT
EITBIN
87