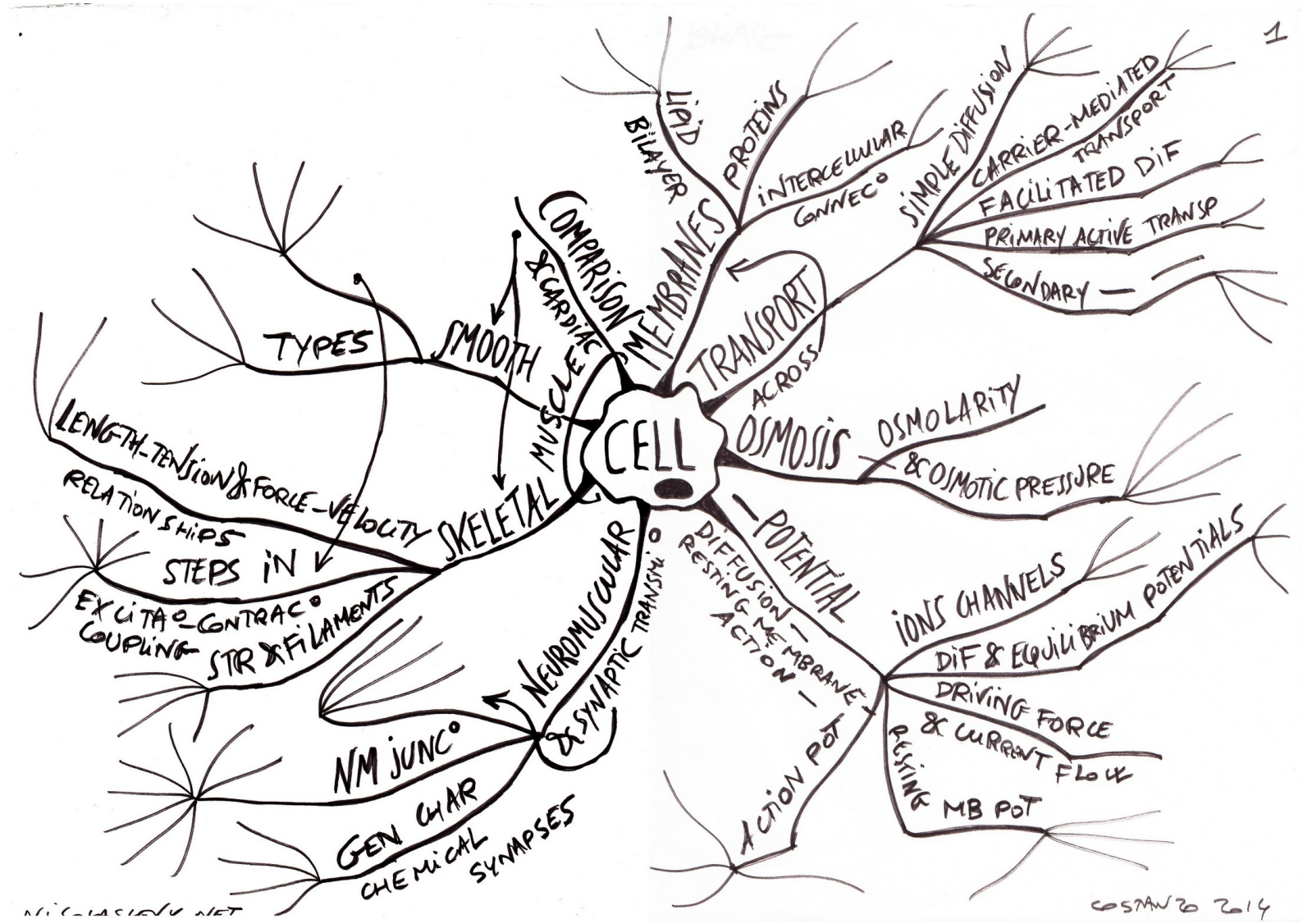
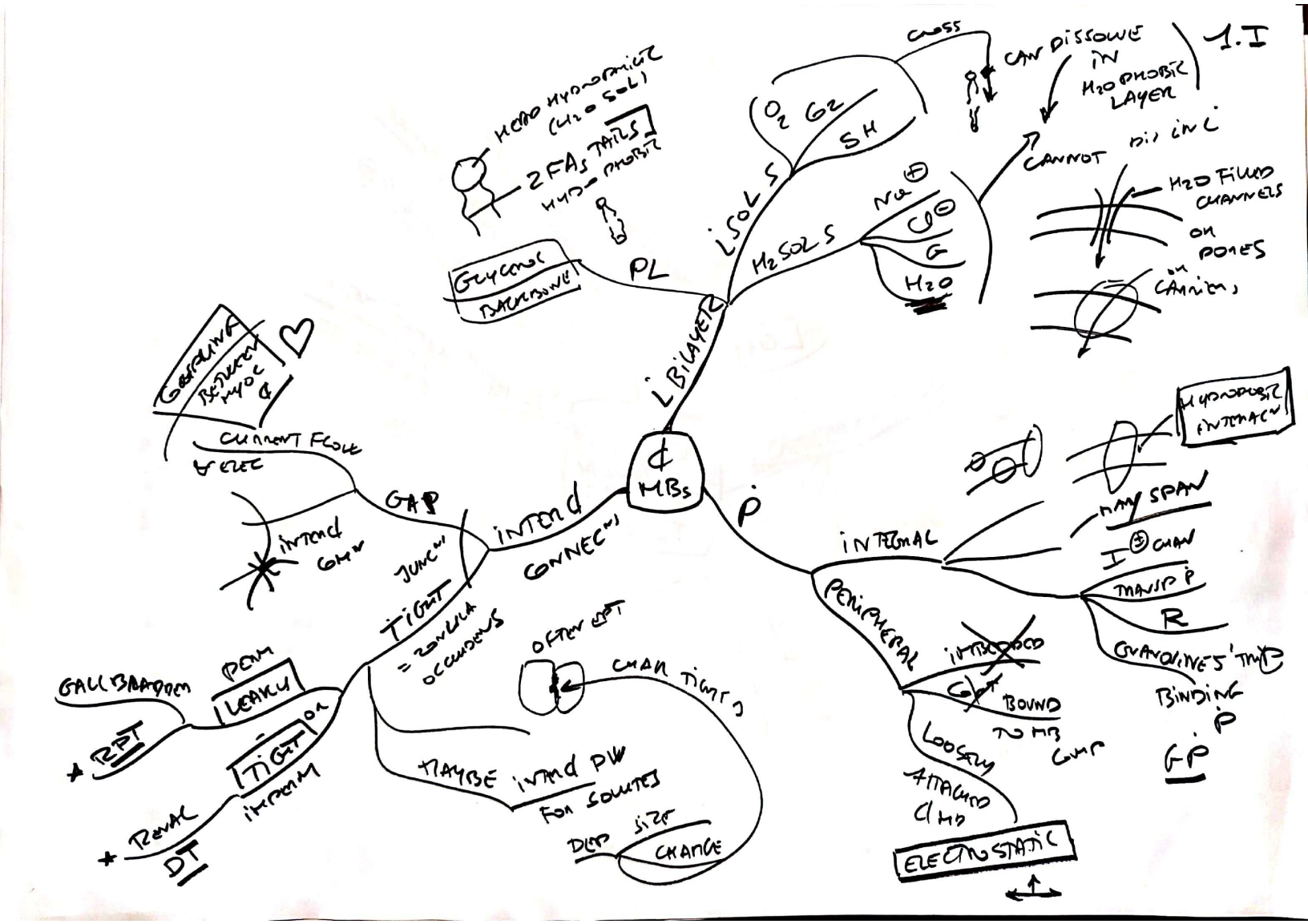
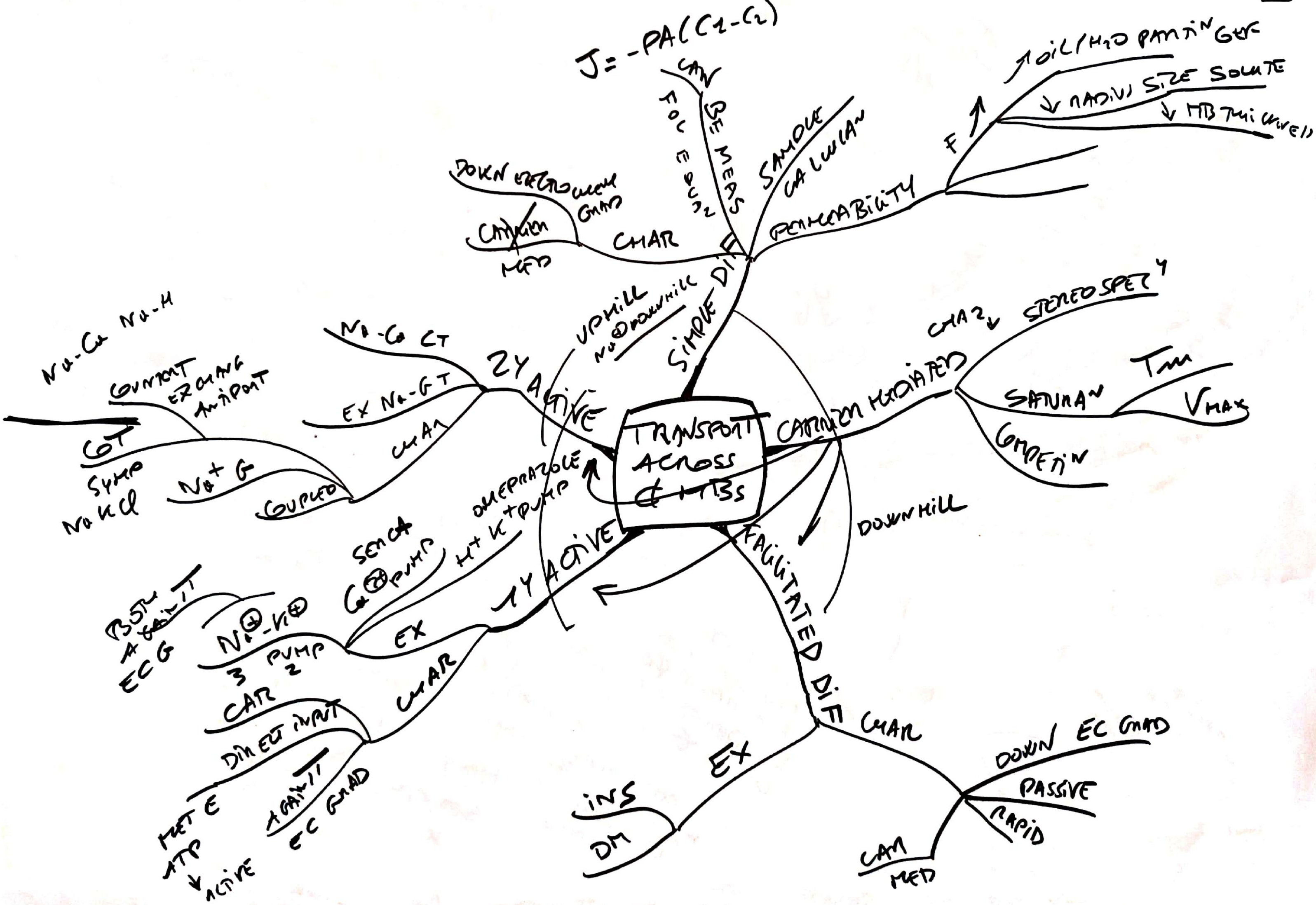

CELL
MEMBRANES
LIPID BILAYER
PROTEINS
INTERCELLULAR CONNEC°
TRANSPORT ACROSS
SIMPLE DIFFUSION
CARRIER-MEDIATED TRANSPORT
FACILITATED DIF
PRIMARY ACTIVE TRANSP
SECONDARY
OSMOSIS
OSMOLARITY & OSMOTIC PRESSURE
POTENTIAL
DIFFUSION POTENTIAL
RESTING MEMBRANE POT
ACTION POT
IONS CHANNELS
DIF & EQUILIBRIUM POTENTIALS
DRIVING FORCE & CURRENT FLOW
RESTING MB POT
MUSCLE
COMPARISON & CARDIAC
SMOOTH
TYPES
SKELETAL
LENGTH-TENSION & FORCE-VELOCITY RELATIONSHIPS
STEPS IN EXCITA°-CONTRAC° COUPLING
STR & FILAMENTS
NEUROMUSCULAR & SYNAPTIC TRANSMI°
NM JUNC°
GEN CHAR
CHEMICAL SYNAPSES


1.I
CAN DISSOLVE IN H2O PHOBIC LAYER
CROSS
O2 CO2
SH
LSOLS
H2SOLS
NO
CO
G
H2O
CANNOT
H2O FILLED CHANNELS OR PORES
HEAD HYDROPHILIC (CH2 O SOL)
2 FAs TAILS HYDROPHOBIC
GLYCOLIPIDS
PL
LBILAYER
GRAPHENE REDUCED HYDRO
CURRENT FLOW & ELEC
GAP
INTERCONNECT
JUNCT
TIGHT = ZONULA OCCLUDENS
INTERCAL CHAN
PERM LEAKY
TIGHT IMPERM
GALLBLADDER
RPT
RENAL
DT
OFTEN OPT
CHAN TIGHTS
MAYBE INTERCAL PW FOR SOLUTES
DIFF SIZE CHANGE
Φ MBs
P
INTRACAL
PERIPHERAL
INTRACOCO
COAT
BOUND TO MB
LOOSELY ATTACHED CMP
ELECTROSTATIC
HYDROPHOBIC INTERACTN
MAY SPAN
CHAN
TRANSP P
R
GUANOSINE 5' TRIP
BINDING P
GP



TRANSPORT ACROSS C MBs
SIMPLE DIF
J = -PA(C1-C2)
CAN BE MEAS
FOR E QUA
SAMPLE
CALULAN
PERMEABILITY
F
↑ OIL/H2O PARTITION COEF
↓ RADIUS SIZE SOLUTE
↓ MB THICKNESS
CHAR
DOWN ELECTROCHEM GRAD
CARRIER MED
UPHILL
DOWNHILL
CARRIER MEDIATED
CHAR
STEREOSPEC
SATURAN
Tm
Vmax
COMPETIN
DOWNHILL
FACILITATED DIF
CHAR
DOWN EC GRAD
PASSIVE
RAPID
CAR MED
EX
INS
DM
1Y ACTIVE
OMEPRAZOLE
H+ K+ PUMP
SERCA
Ca PUMP
EX
Na-K
3 PUMP 2
ECG
CHAR
CAR
DIRECT INPUT
MET E
ATP
ACTIVE
AGAINST
EC GRAD
2Y ACTIVE
Na-Ca CT
EX Na-G T
CHAR
COUPLED
Na+ G
SYMP
Na K Cl
CT
COUNTER EXCHANG ANTIPORT
Na-Ca Na-H

$1 mL = 1 cm^3$

$$\text{Flux} = \left[\frac{1\times10^{-5}}{sec}\right](100\,cm^2)\left(\frac{20\,mg}{100\,mL} - \frac{10\,mg}{100\,mL}\right)$$

10

=

$= \frac{0.1\,mg}{cm^3}$

$= 1\times10^{-4}\,mg/sec$ LUMEN → Bd

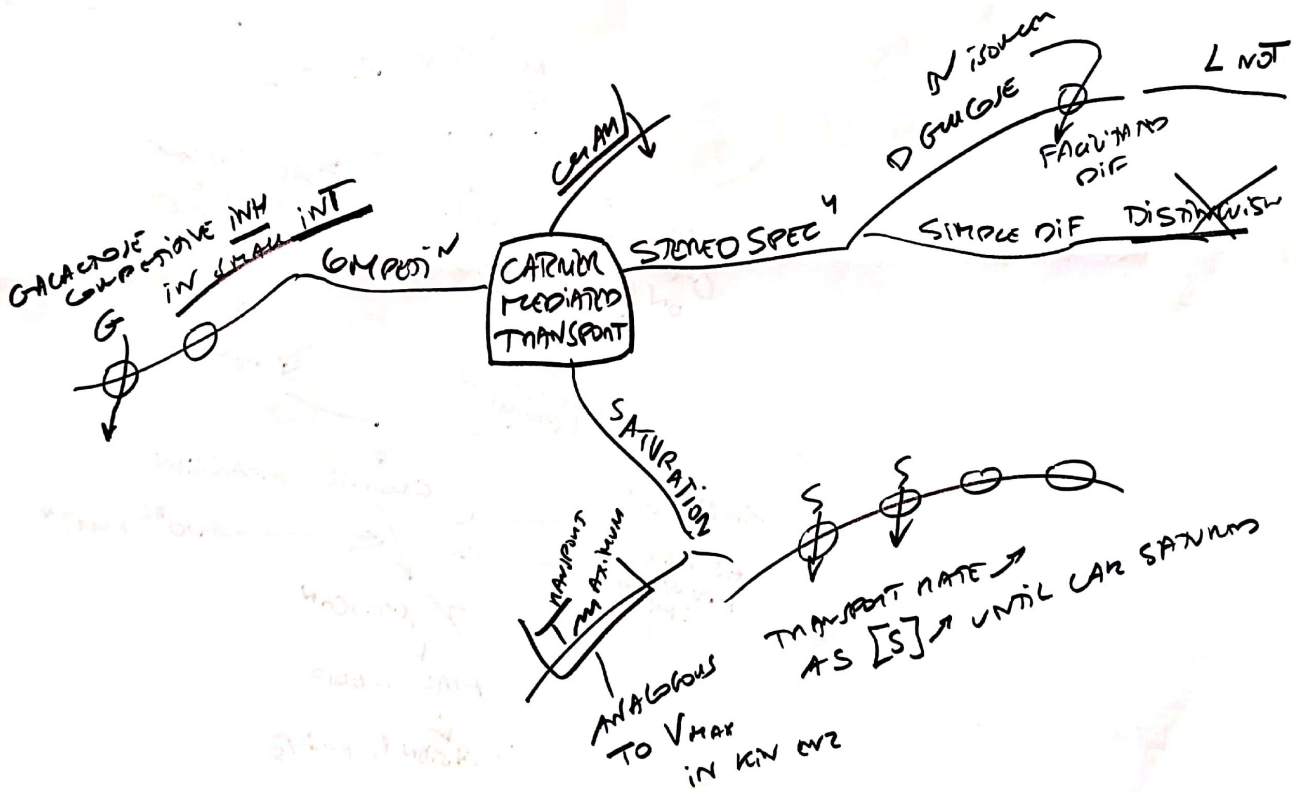
$[UREA]_{Bd} = \frac{10\,mg}{100\,mL}$

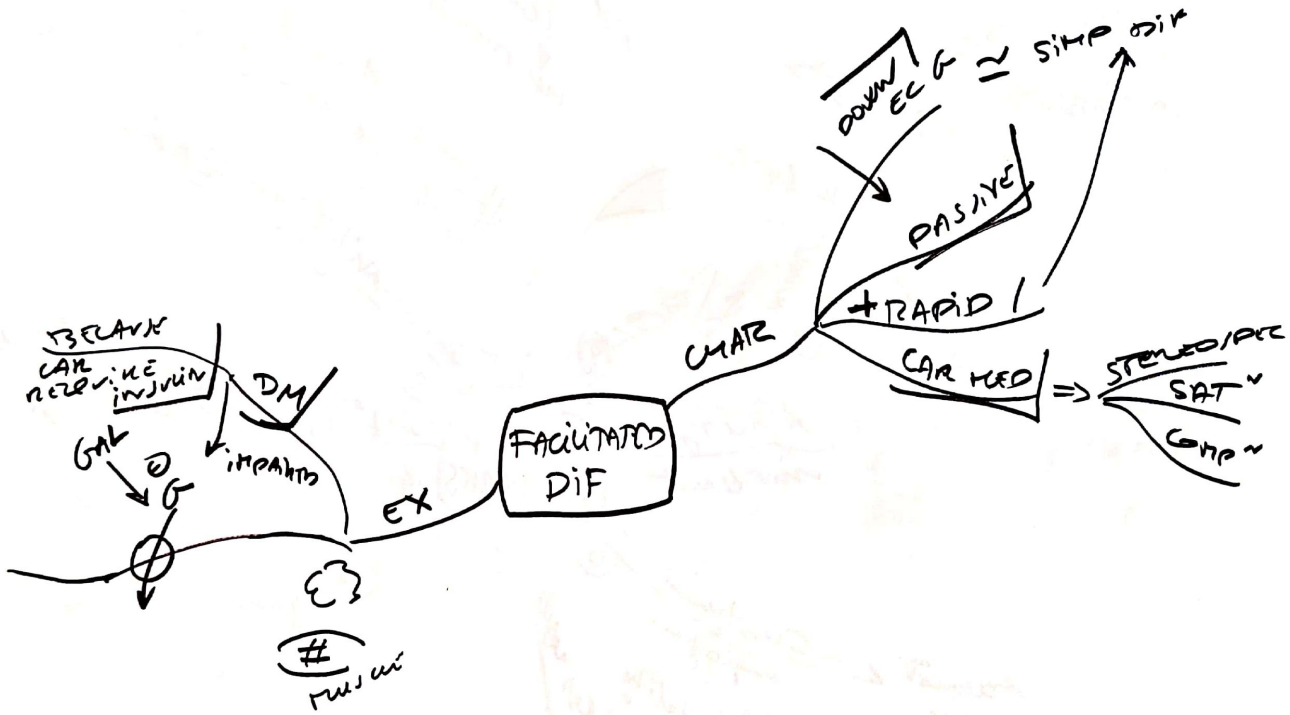
PT $\frac{20\,mg}{100\,mL}$

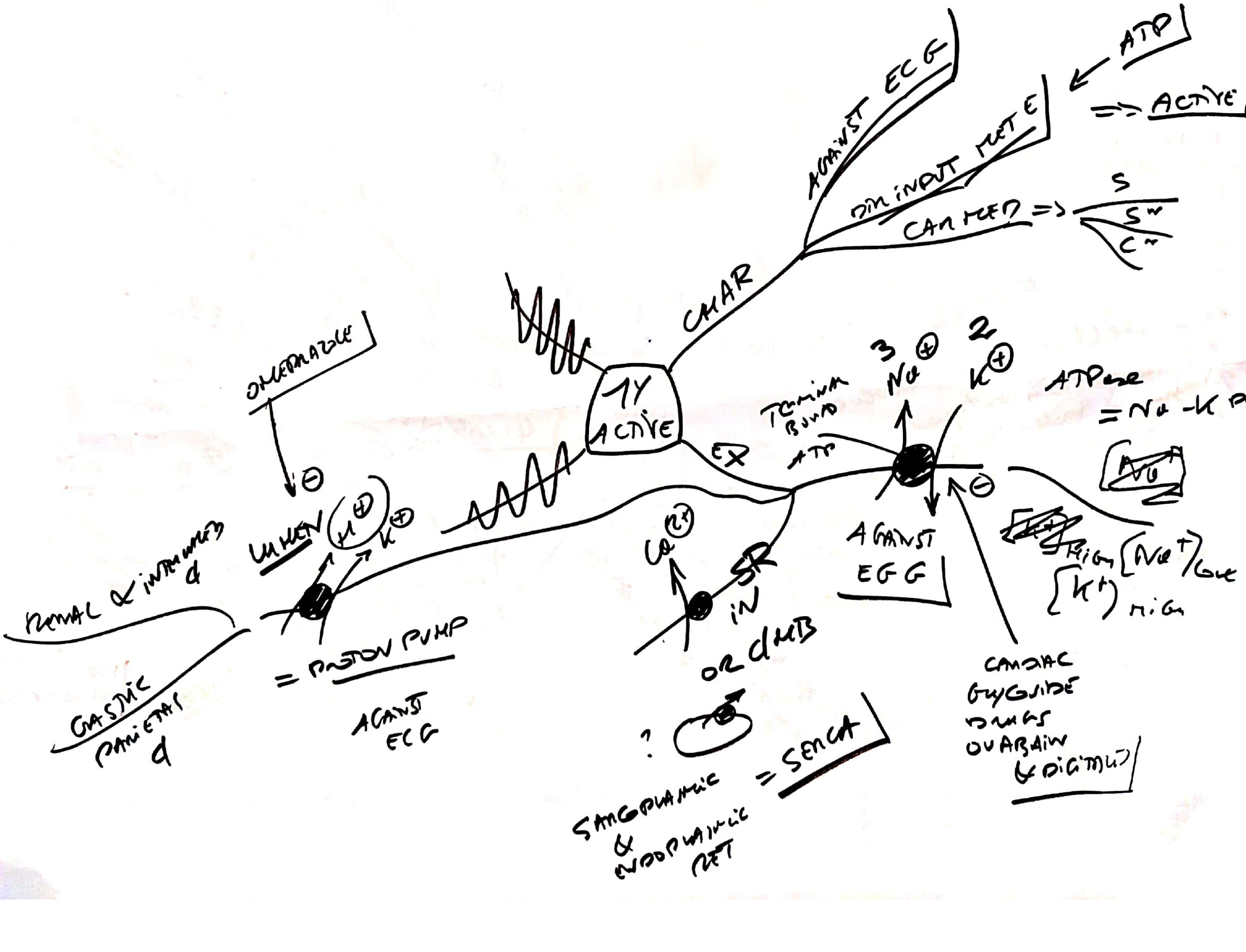
$P_{UREA} = 1\times10^{-5}\,cm/sec$

$A = 100\,cm^2$

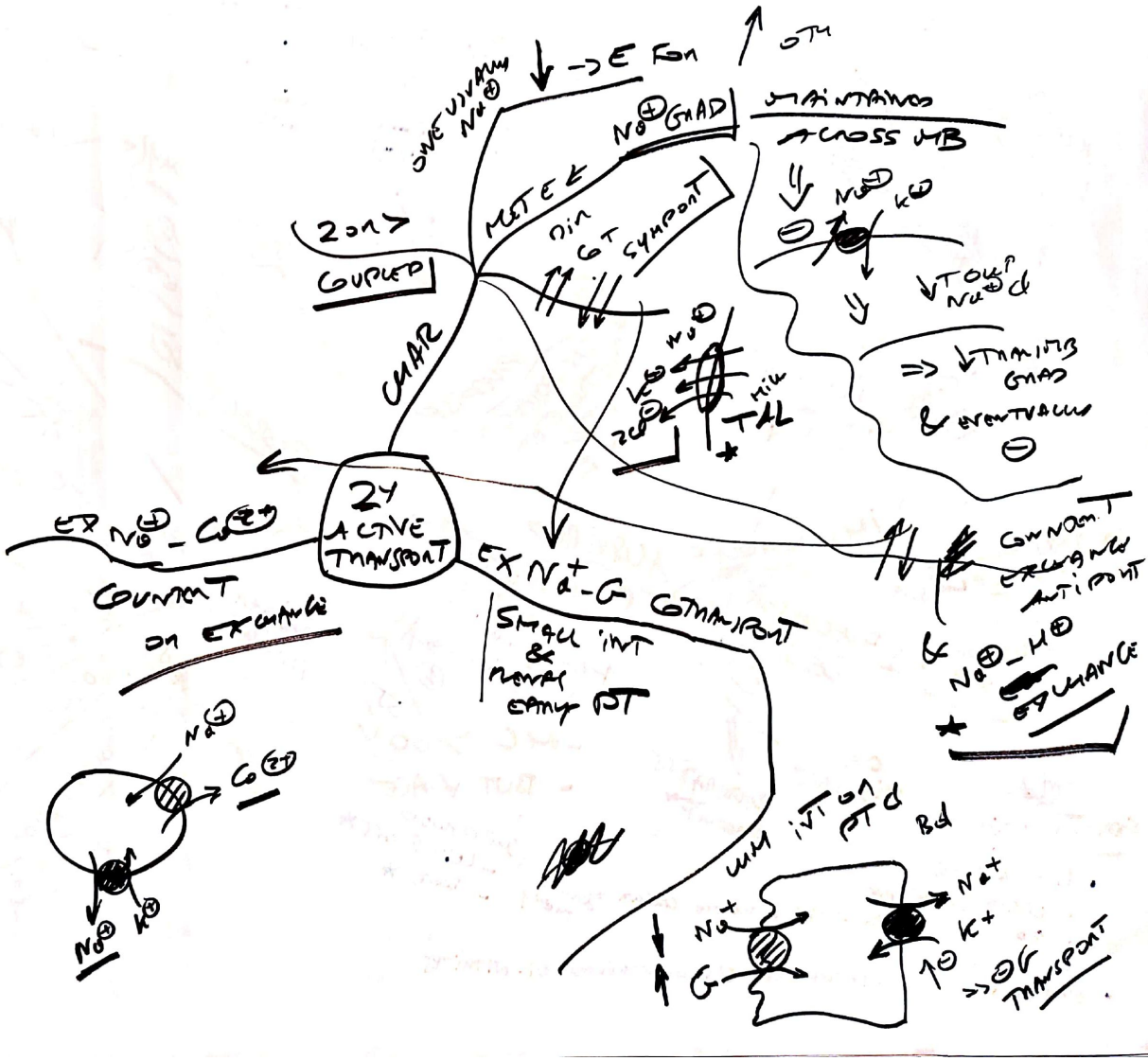
MAGNITUDE & DIRECTION ?


CARRIER MEDIATED TRANSPORT
COMPETITIVE INH
UNCOMPETITIVE INH
COMPETITIVE INT
STEREOSPECIFICITY
D GLUCOSE
L NOT
FACILITATED DIF
SIMPLE DIF
SATURATION
TRANSPORT MAXIMUM
ANALOGOUS TO Vmax IN MICHAELIS MENTEN
TRANSPORT RATE AS [S] UNTIL CARRIER SATURATES

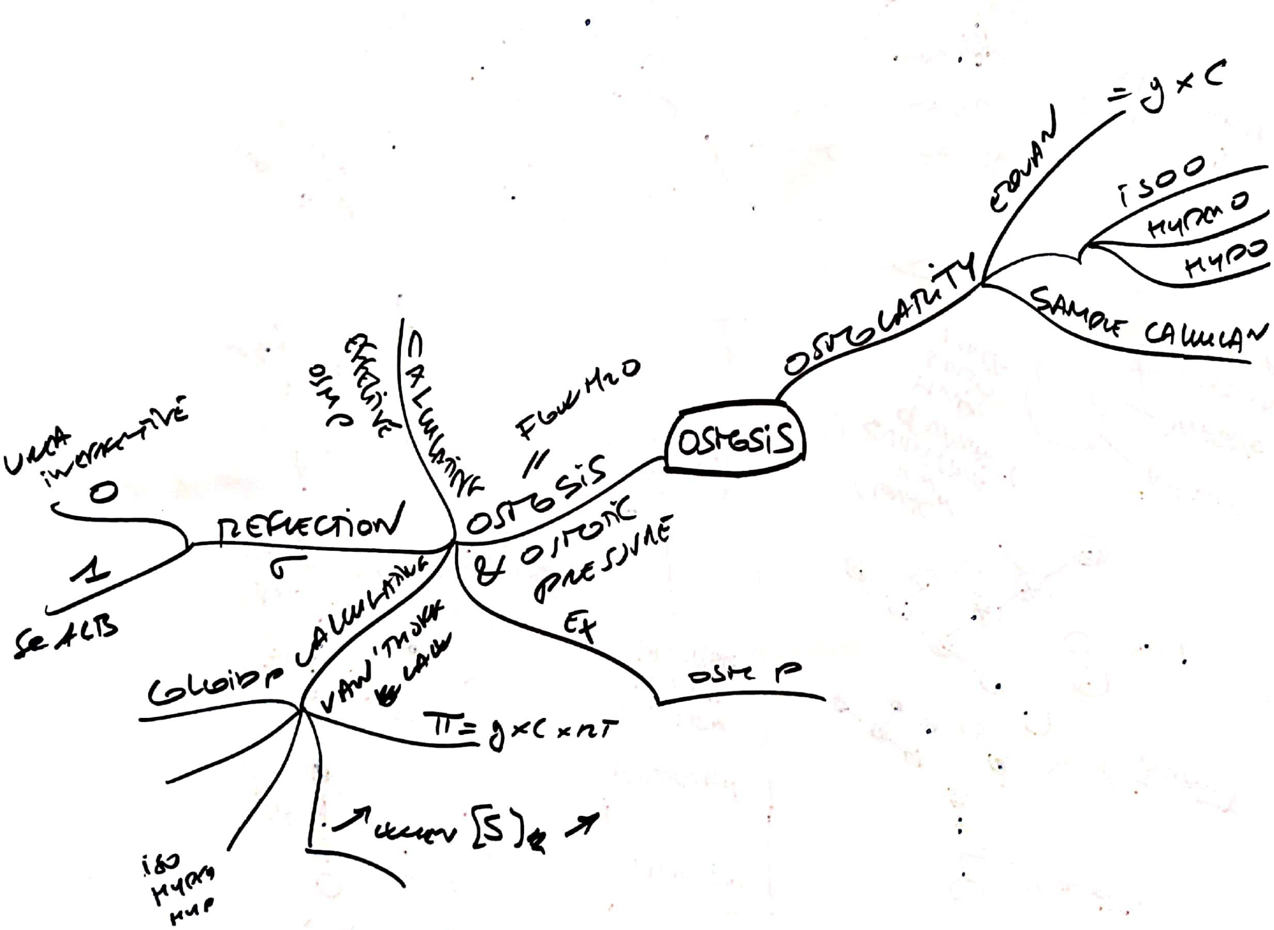

DOWN
ECG ≃ SIMP dir
PASSIVE
+RAPID /
CAR MED
=> STEROIDS/ICE
SAT ~
COMP ~
CHAIR
FACILITATED
DIF
EX
IMPAIRED
DM
GAIT
#

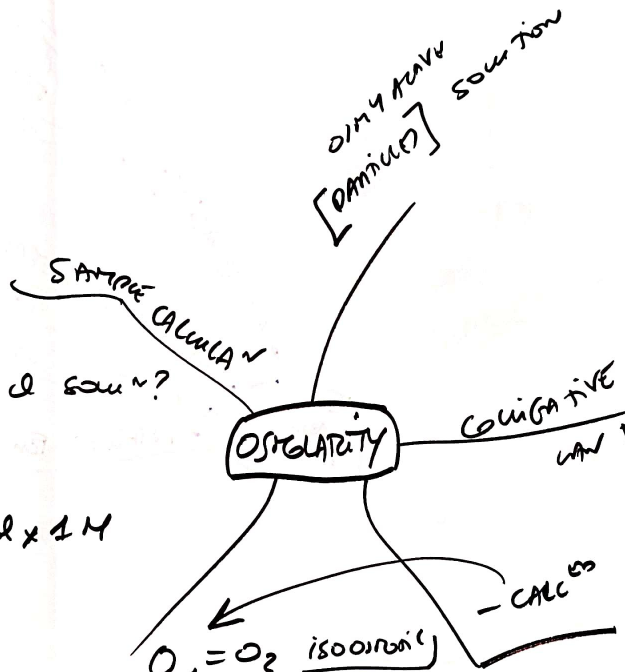


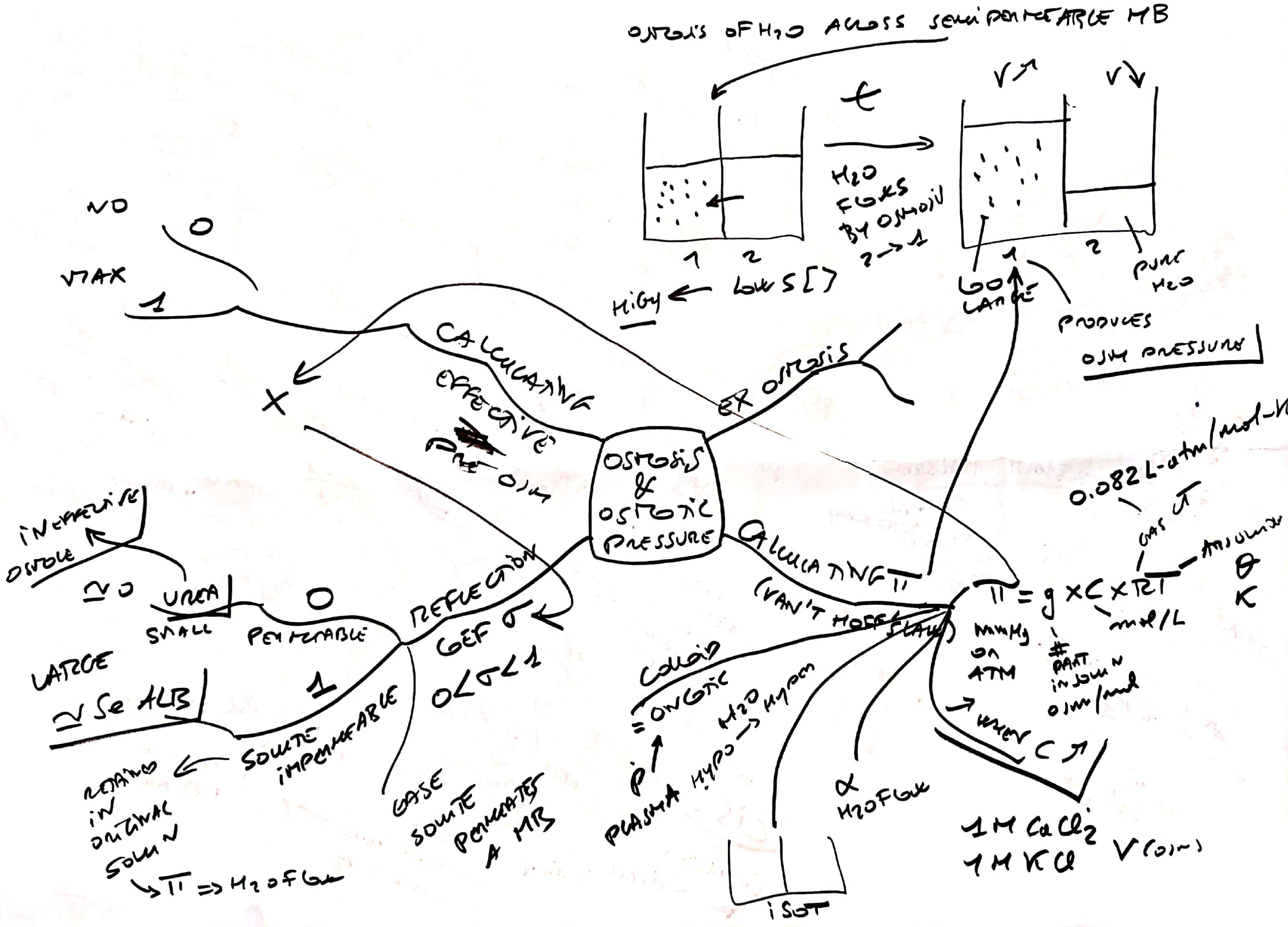
1Y ACTIVE
CHAR
AGAINST ECG
ATP
=> ACTIVE
EX
3 Na+
2 K+
ATP
ATPase = Na-K PUMP
AGAINST EGG
HIGH [Na+] LOW
[K+] HIGH
CARDIAC GLYCOSIDE DRUGS OUABAIN & DIGITALIS
Ca2+
SR IN
= SERCA
OMEPRAZOLE
LUMEN
H+ K+
= PROTON PUMP
AGAINST ECG
GASTRIC PARIETAL

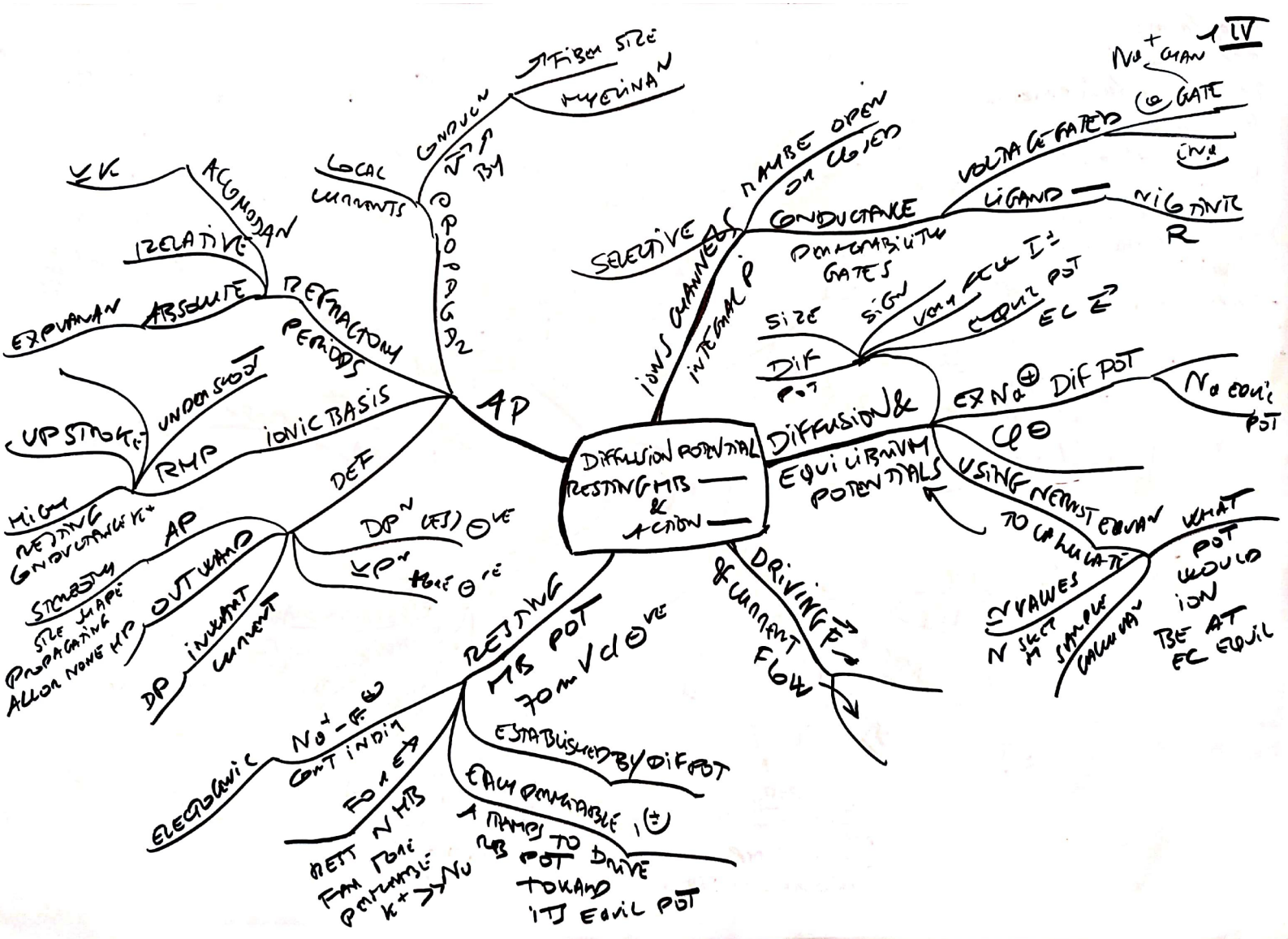

2 ary
COUPLED
CHAR
2y
ACTIVE
TRANSPORT
EX Na⊕ - Ca⊕
COUNTER
EXCHANGE
EX Na+ - G
SMALL INT
&
RENAL
EARLY PT
Na⊕
Ca⊕
Na⊕
K⊕
Na+
G
Na+
K+

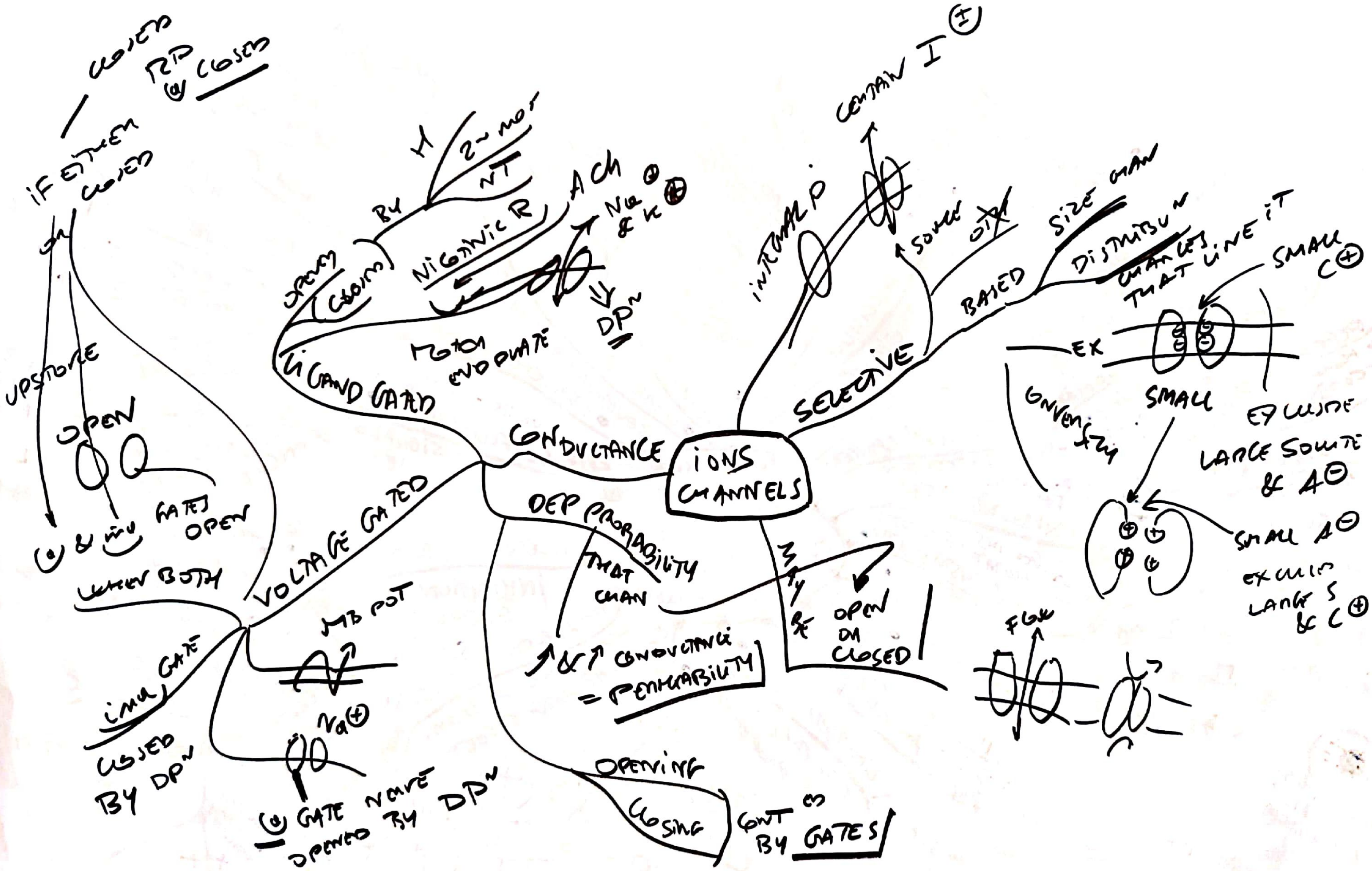
1. III

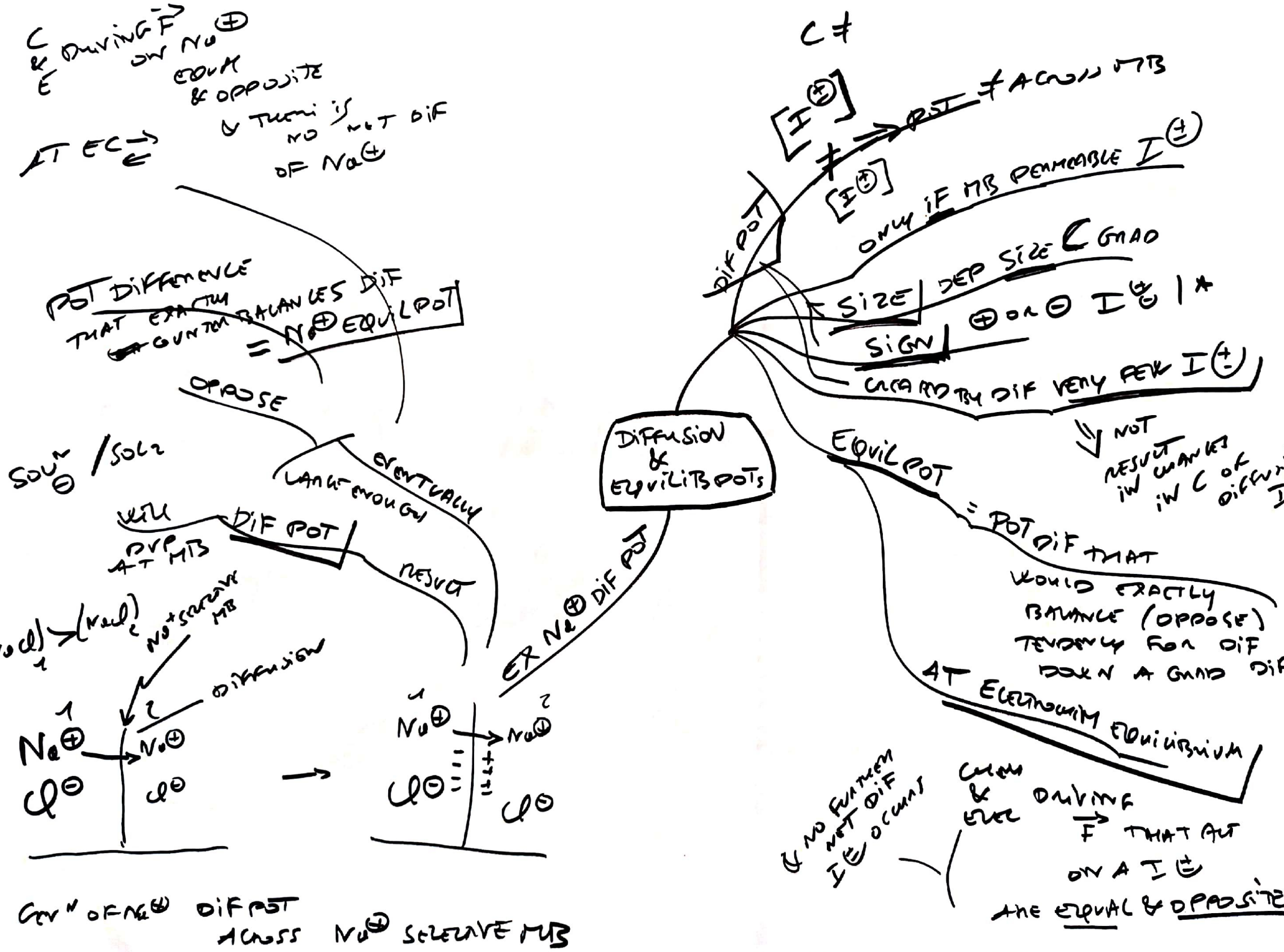

OSMOSIS
OSMOLARITY
= g × C
ISOO
HYPERO
HYPO
SAMPLE CALCULAN
FLOW H2O
//
OSMOSIS & OSMOTIC PRESSURE
CALCULATING
EFFECTIVE
OSM P
REFLECTION
σ
UREA INEFFECTIVE
0
1
SE ALB
COLLOID CALCULATING
VAN'T HOFF'S LAW
π = g × C × RT
EX
OSM P
ISO
HYPER
HYP

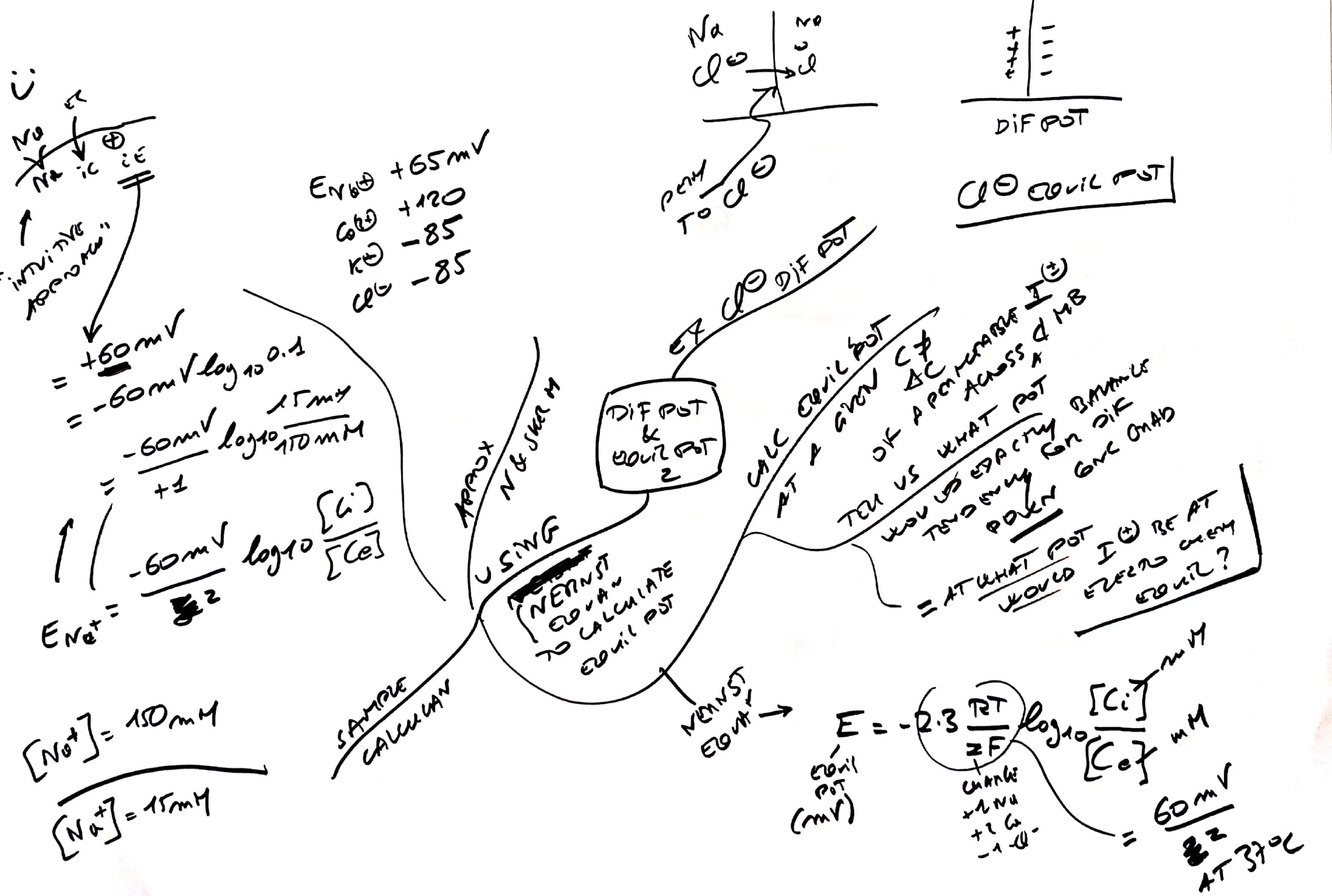

OSMOLARITY
OSMOLARITY
SAMPLE CALCULATION
1M NaCl SOLN?
OSMOLARITY = g x C
= 2 osm/mol x 1 M
= 2 osm/L
OSMOLARITY
[DAMAGED]
SOLUTION
COLLIGATIVE PROPERTY
CAN BE MEAS BY FREEZING POINT DEPRESN
CALCD
O1 = O2 ISOOSMOTIC
HYPER
HYPO
OSMOLARITY = g x C
OSM/L
PART SOLN
OSM/mol
mol/L
gNaCl = 2
gGLUCOSE = 1


OSMOSIS & OSMOTIC PRESSURE
OSMOSIS OF H2O ACROSS SEMIPERMEABLE MB
H2O FLOWS BY OSMOSIS 2 → 1
1 2
HIGH ← LOW S [ ]
PRODUCES OSM PRESSURE
PURE H2O
EX OSMOSIS
CALCULATING EFFECTIVE PRE OSM
NO 0
MAX 1
CALCULATING π (VAN'T HOFF'S LAW)
π = g × C × RT
0.082 L-atm/mol-K
GAS CT
ABSOLUTE T K
mol/L
mmHg OR ATM
PART IN SOLN osm/mol
WHEN C ↑
1 M CaCl2
1 M KCl
REFLECTION COEF σ
0<σ<1
PERMEABLE 0
UREA
SMALL
≈ 0
INEFFECTIVE OSMOLE
LARGE
≈ Se ALB
1
SOLUTE IMPERMEABLE
RETAINED IN ORIGINAL SOLN
↑π ⇒ H2O FLOW
CASE SOLUTE PERMEATES A MB
COLLOID = ONCOTIC
P ↑
PLASMA HYPO → HYPER H2O
∝ H2O FLOW
ISOT

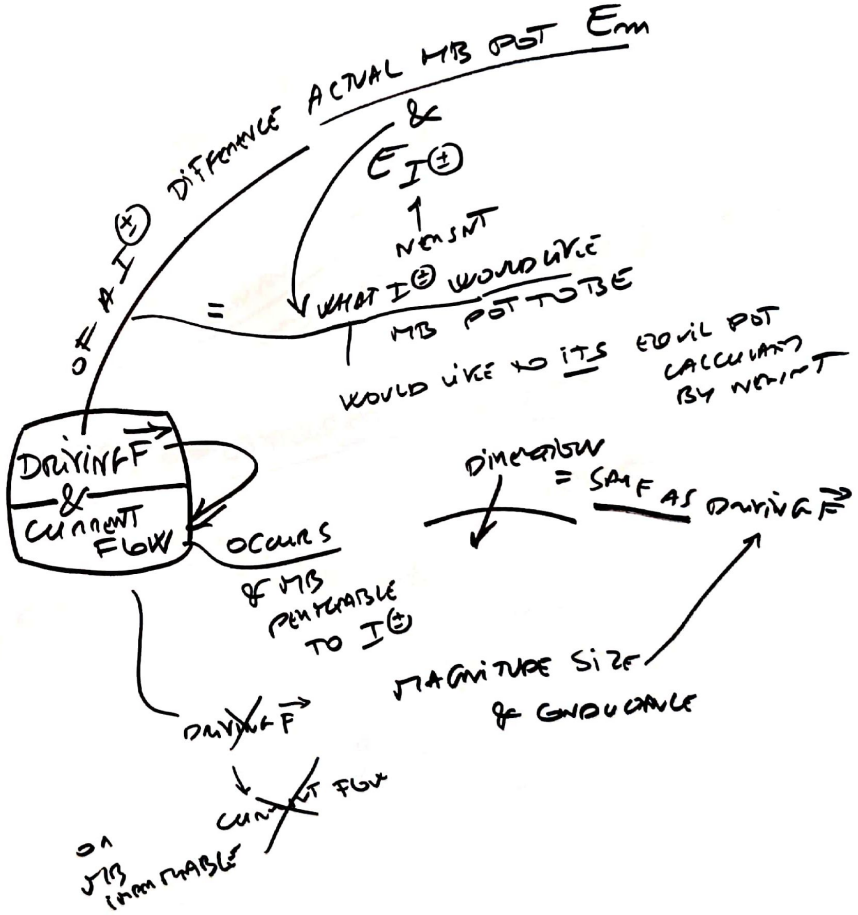

DIFFUSION POTENTIAL
RESTING MB & ACTION
DIFFUSION&
EQUILIBRIUM POTENTIALS
DRIVING F
CURRENT FLOW
AP
IONIC BASIS
DEF
RMP
UNDERSHOOT
OVERSHOOT
REFRACTORY PERIODS
ABSOLUTE
RELATIVE
ACCOMODAN
PROPAGATION
LOCAL CURRENTS
CONDUCTANCE
SELECTIVE
IONS CHANNELS
INTEGRAL P
VOLTAGE GATED
LIGAND
GATE
DIF POT
EC
SIZE
SIGN
RESTING MB POT
FROM Vd
ESTABLISHED BY DIF POT
ELECTROGENIC
CONTINUITY
EQUILIBRIUM POTENTIAL
DRIVE TOWARD EQUIL POT
USING NERNST EQUAN
WHAT POT WOULD ION BE AT EC EQUIL

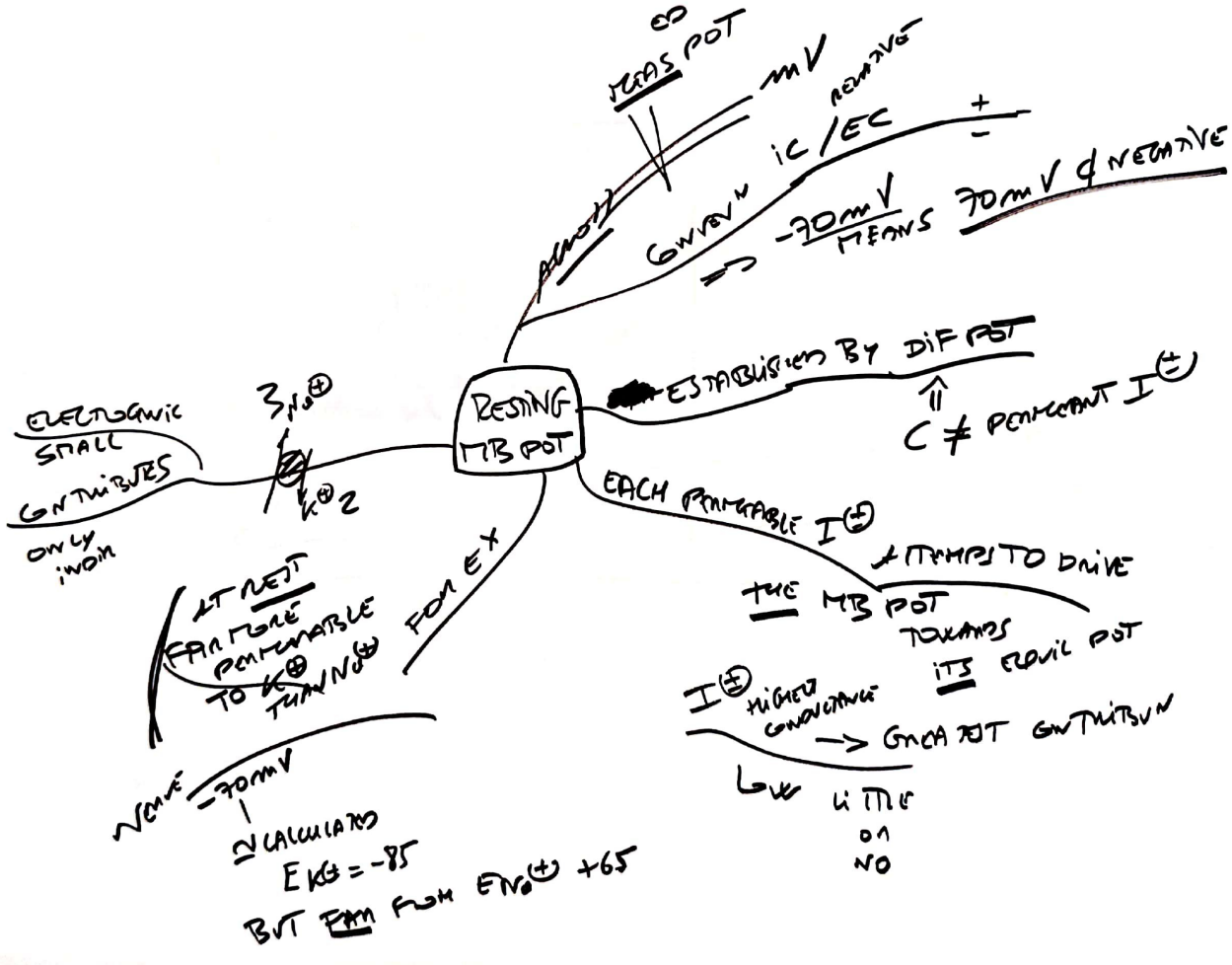

IONS CHANNELS
CONDUCTANCE
SELECTIVE
BASED
SIZE CHAN
DISTRIBUTN
CHARGES THAT LINE IT
SMALL C⊕
EX
SMALL
EXCLUDE LARGE SOLUTE & A⊖
SMALL A⊖
EXCLUD LARGE S & C⊕
INTERNAL P
CERTAIN I
SOME
OTH
DEP PROBABILITY THAT CHAN
MAY BE OPEN OR CLOSED
↑ & ↑ CONDUCTANCE = PERMEABILITY
OPENING
CLOSING
CONT BY GATES
LIGAND GATED
NICOTINIC R
ACH
Na & K ⊕
DPⁿ
MOTOR END PLATE
OPEN
CLOSED
BY
NT
VOLTAGE GATED
MB POT
Na⊕
INACT GATE
CLOSED BY DPⁿ
⊕ GATE NEVER OPENED BY DPⁿ
WHEN BOTH
⊕ & INACT GATES OPEN
OPEN
UPSTROKE
IF EITHER CLOSED
CLOSED
RP
⊕ CLOSED

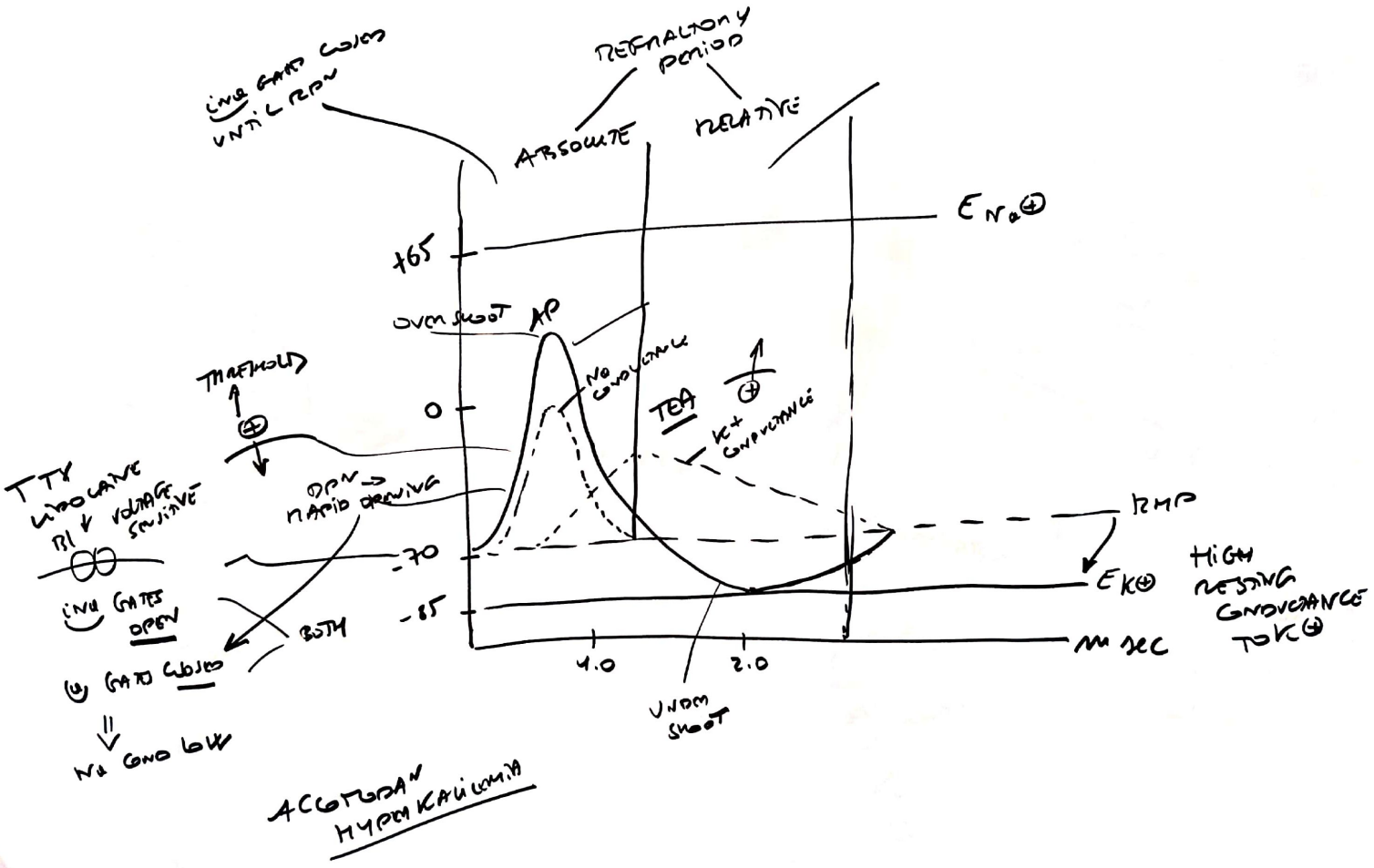

DIFFUSION & EQUILIB POTS
EX Na⊕ DIF POT
DIF POT
C ≠
POT ≠ ACROSS MB
ONLY IF MB PERMEABLE I
DEP SIZE C GRAD
SIZE
SIGN
⊕ OR ⊖
VERY FEW I
NOT RESULT IN CHANGE IN C OF DIFFUSING I
EQUIL POT
= POT DIF THAT WOULD EXACTLY BALANCE (OPPOSE) TENDENCY FOR DIF DOWN A GRAD DIFFERENCE
AT ELECTROCHEM EQUILIBRIUM
DRIVING F THAT ACT ON A I
ARE EQUAL & OPPOSITE
NO FURTHER NET DIF OCCURS
POT DIFFERENCE THAT EXACTLY COUNTERBALANCES DIF = Na⊕ EQUIL POT
OPPOSE
EVENTUALLY
LARGE ENOUGH
DIF POT
RESULT
AT MB
DIFFUSION
Na⊕
Cl⊖
GEN OF Na⊕ DIF POT ACROSS Na⊕ SELECTIVE MB
AT EC
OF Na⊕

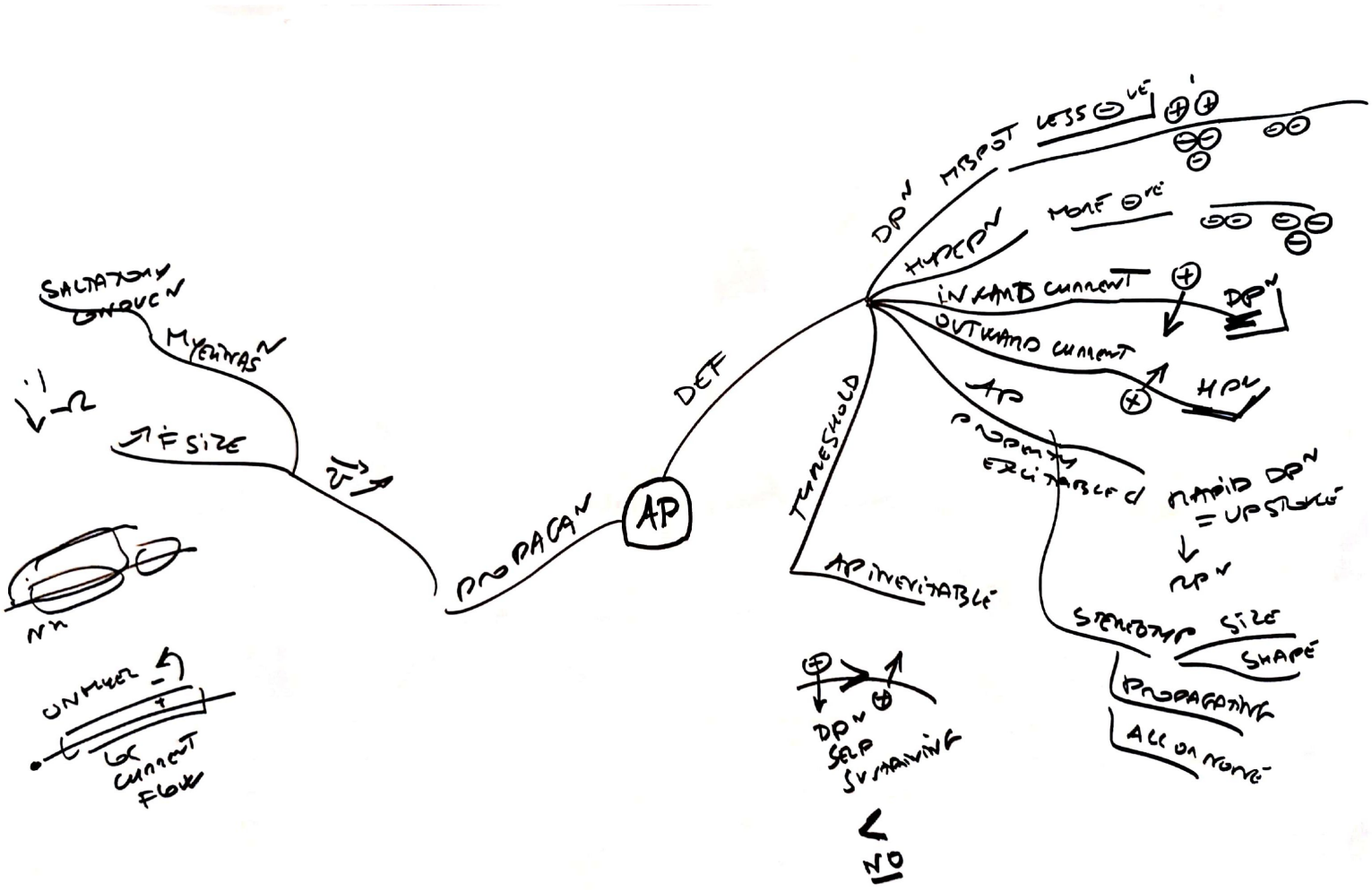


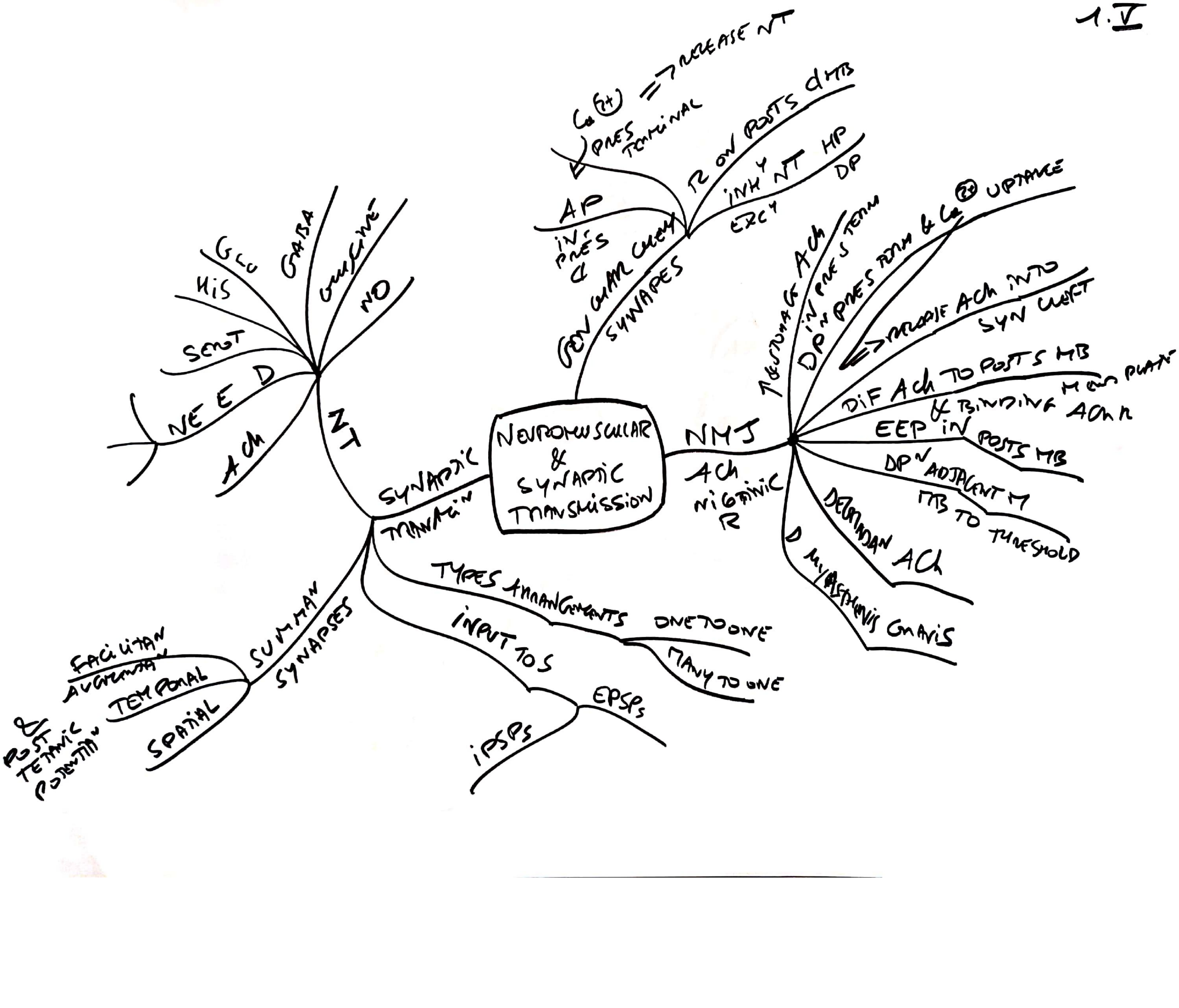
DIF POT & EQUIL POT 2
USING NERNST EQUATION TO CALCULATE EQUIL POT
NERNST EQUAT
$E = -2.3 \frac{RT}{zF} \log_{10} \frac{[C_i]}{[C_e]}$ mM
EQUIL POT (mV)
CHARGE +1 Na +2 Ca -1 Cl-
$= \frac{60\,mV}{z}$ AT 37°C
CALC EQUIL POT AT A GIVEN ΔC OF A PERMEABLE I(±) ACROSS A MB
TELL US WHAT POT WOULD EXACTLY BALANCE TENDENCY FOR DIF POWER
= AT WHAT POT WOULD I(±) BE AT ELECTRO CHEM EQUIL?
EX Cl⊖ DIF POT
Na Cl⊖ Na Cl
PERM TO Cl⊖
DIF POT
Cl⊖ EQUIL POT
APPROX N & SKM
E_{Na^+} +65 mV
Ca^{2+} +120
K^+ -85
Cl^- -85
SAMPLE CALCULATION
$[Na^+] = 150\,mM$
$[Na^+] = 15\,mM$
$E_{Na^+} = \frac{-60\,mV}{z} \log_{10} \frac{[C_i]}{[C_e]}$
$= \frac{-60\,mV}{+1} \log_{10} \frac{15\,mM}{150\,mM}$
$= -60\,mV \log_{10} 0.1$
$= +60\,mV$
"INTUITIVE APPROACH"
Na EC Na IC ⊕ IE

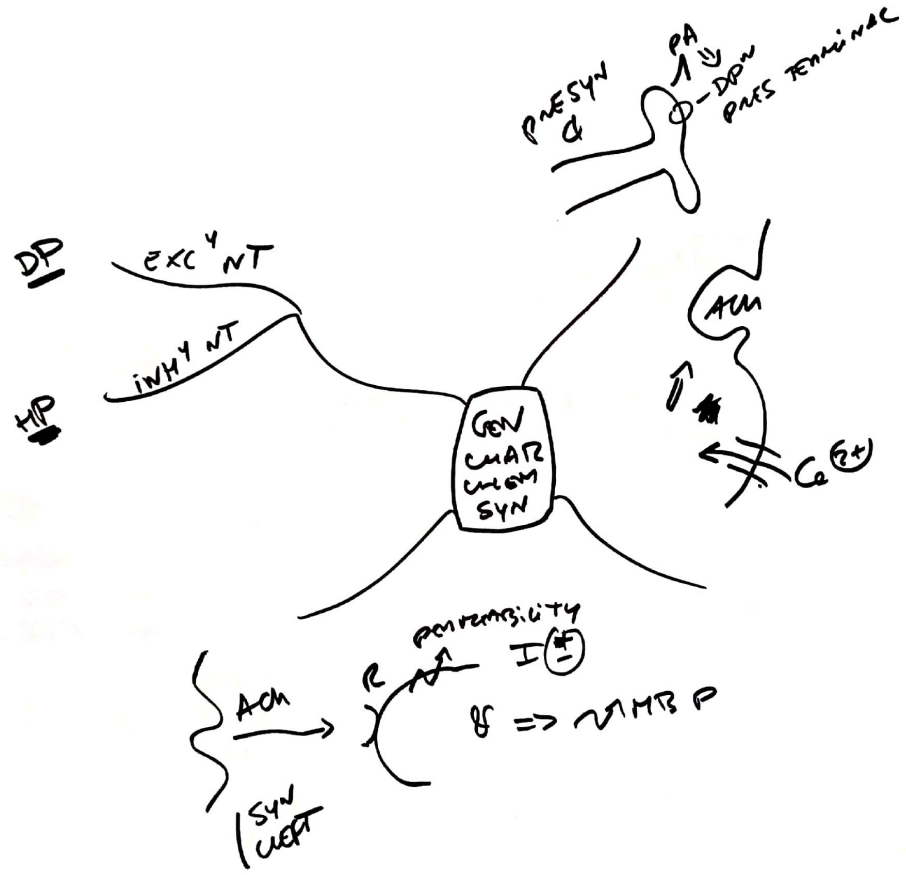

DIFFERENCE ACTUAL MB POT E_{mn}
OF $A_{I\pm}$
&
$E_{I\pm}$
=
WHAT $I\pm$ WOULD LIKE MB POT TO BE
WOULD LIKE TO ITS EQUIL POT CALCULATED BY NEWT
DRIVING F
&
CURRENT FLOW
OCCURS
OF MB PERMEABLE TO $I\pm$
DIMENSIONLESS = SAME AS DRIVING F
MAGNITUDE SIZE & GRADIENT
DRIVING F
CURRENT FLOW
ON MB IMPERMEABLE

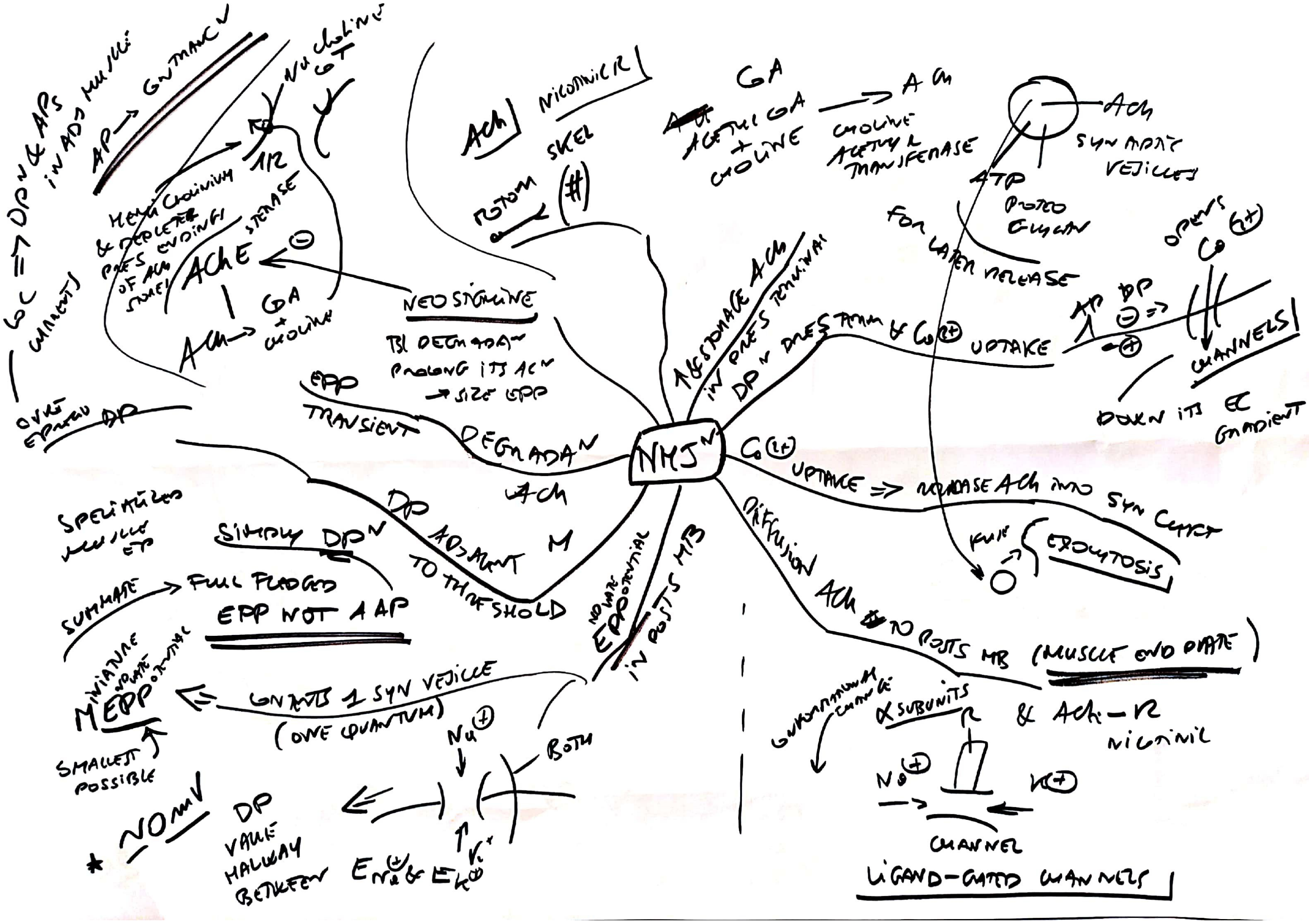

RESTING MB POT
ELECTROGENIC SMALL CONTRIBUTES
ONLY INDIR
3 Na+
K+2
ESTABLISHED BY DIFFUSION
C ≠ PERMEANT I±
EACH PERMEABLE I±
TRANSPORTERS TO DRIVE
THE MB POT TOWARDS ITS EQUIL POT
I± HIGHER CONDUCTANCE → GREATER CONTRIBUTION
LOW LITTLE OR NO
AT LEAST FOR MORE PERMEABLE TO K+ TURNING POINT EX
NORM -70mV
≅ CALCULATED EK+ = -85
BUT FAR FROM ENa+ +65
MEAS POT
mV
NEGATIVE
GOVERN IC/EC
+
-
-70mV MEANS 70mV of NEGATIVE

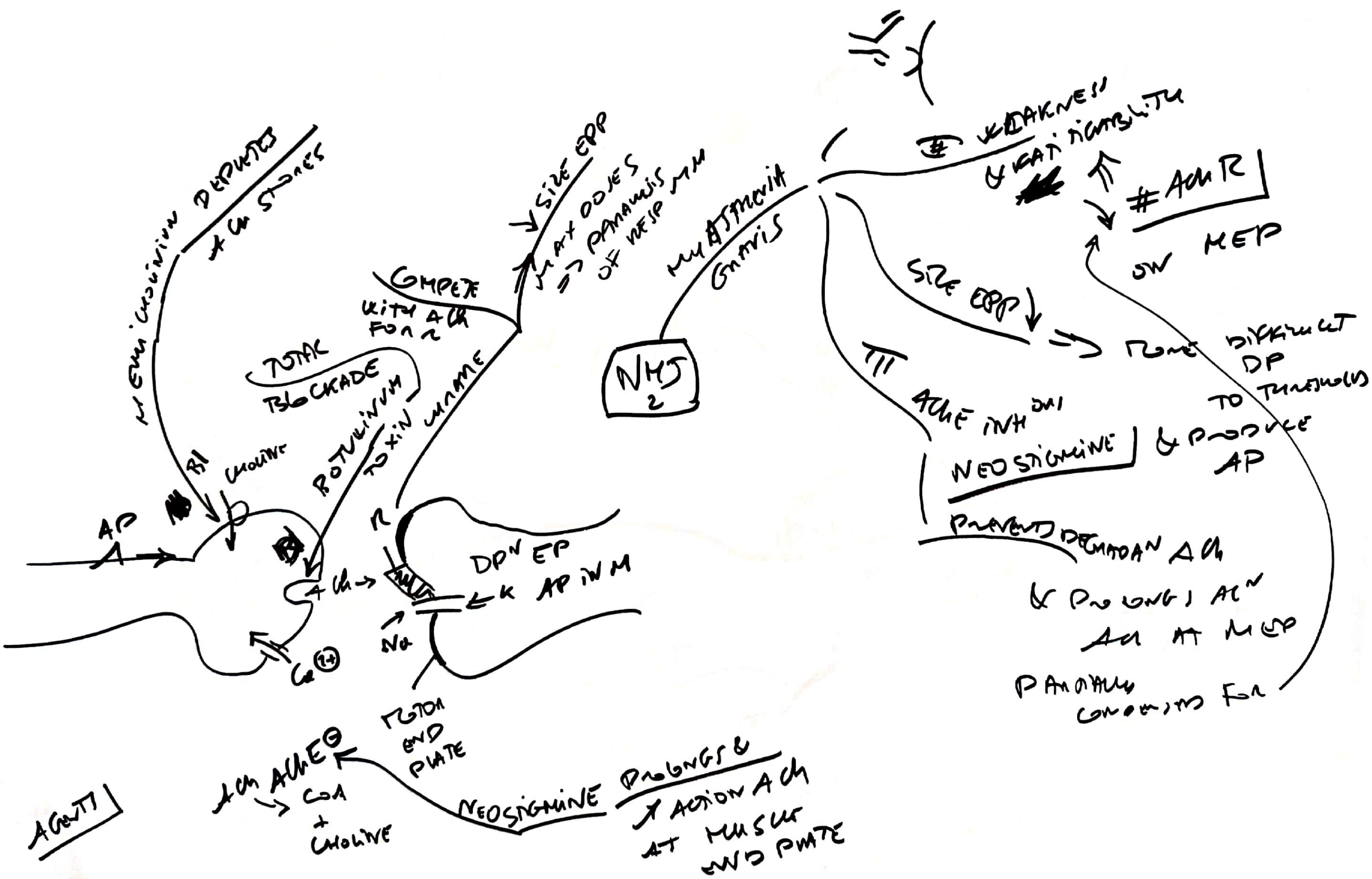

INA GATES CLOSED UNTIL REPOL
REFRACTORY PERIOD
ABSOLUTE
RELATIVE
ENa⊕
+65
OVERSHOOT AP
THRESHOLD
0
NO CONDUCTANCE
TEA
K+ CONDUCTANCE
TTX
VOLTAGE SENSITIVE
OPEN → RAPID DRIVING
RMP
-70
INA GATES OPEN
BOTH
-85
EK⊖
HIGH RESTING CONDUCTANCE TO K⊖
GATES CLOSED
msec
4.0
2.0
UNDERSHOOT
INA GATES CLOSED
ACCOTARD HYPERKALEMIA

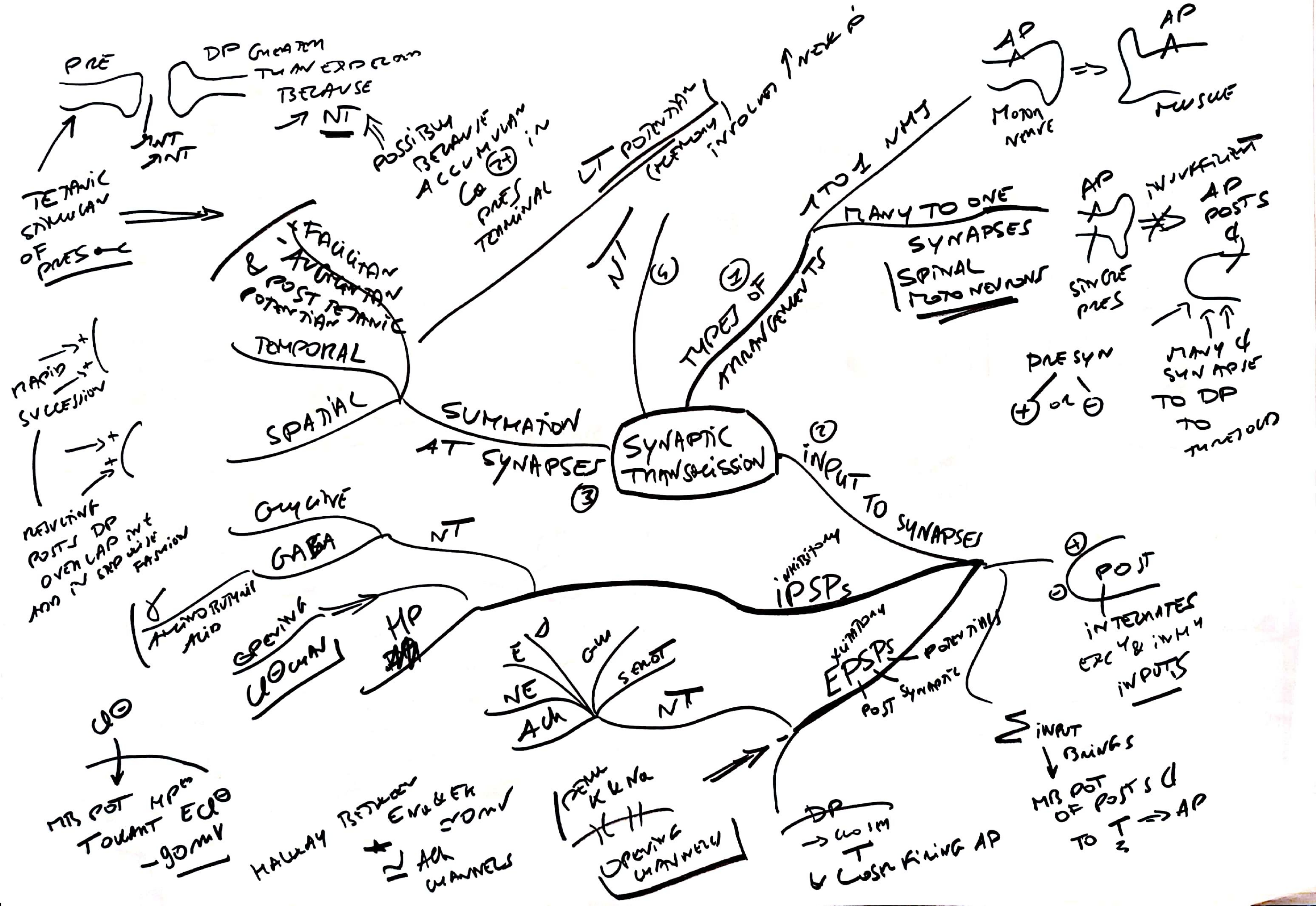

AP
DEF
PROPAGN
DPN MSPOT LESS ⊖ VE
HYPERPN
MORE ⊖VE
INWARD CURRENT
DPN
OUTWARD CURRENT
HPN
AP
PROPERTY EXCITABLE CL
RAPID DPN = UPSTROKE
RPN
STIMULUS SIZE SHAPE
PROPAGATIVE
ALL OR NONE
THRESHOLD
AP INEVITABLE
DPN SELF SUMMING
NO
SALTATORY CONDUCTN
MYELINATN
↑ FIBRE SIZE
↑ ↑
NN
UNMYEL
LOC CURRENT FLOW


NEUROMUSCULAR & SYNAPTIC TRANSMISSION
NMJ
ACh NICOTINIC R
SYNAPTIC TRANSM
SYNAPSES
NT
GLU
GABA
GLYCINE
HIS
NO
SEROT
NE E D
ACh
AP
IN PRES
PRES TERMINAL
R ON POSTS
INHY NT HP
EXCY DP
DP IN PRES TERM & Ca UPTAKE
RELEASE ACh INTO SYN CLEFT
DIF ACh TO POSTS MB
EEP IN POSTS MB
DEGRADATION ACh
TYPES ARRANGEMENTS
ONE TO ONE
MANY TO ONE
INPUT TO S
EPSPs
IPSPs
SUMMATION
FACILITATION
AUGMENTATION
TEMPORAL
SPATIAL
& POST TETANIC POTENTIATION

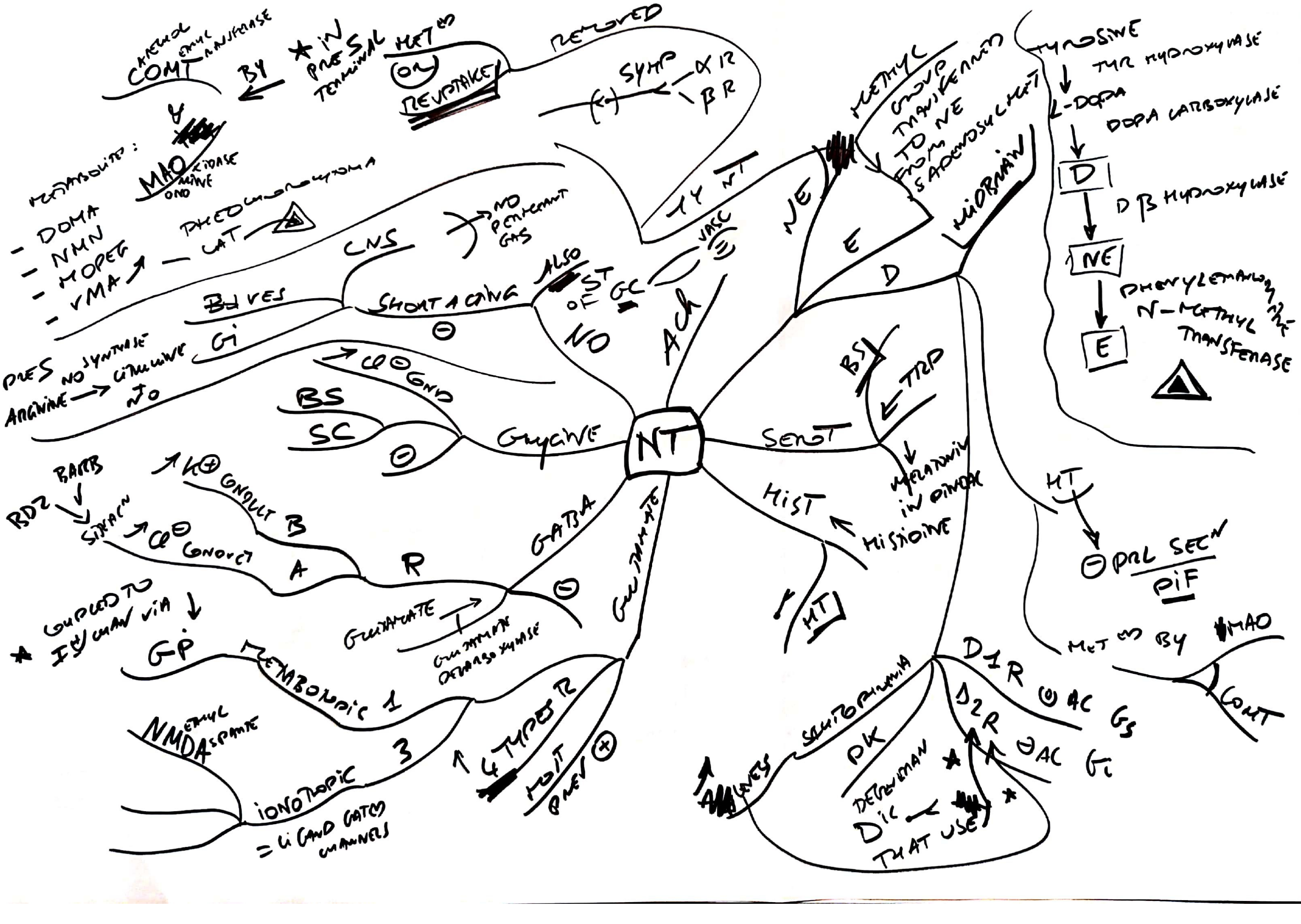

PRESYN
PA
DP
PRES TERMINAL
DP
EXC NT
INH NT
HP
GEN
CHAR
UNCOM
SYN
ACh
Ca++
ACh
R
I
SYN
CLEFT

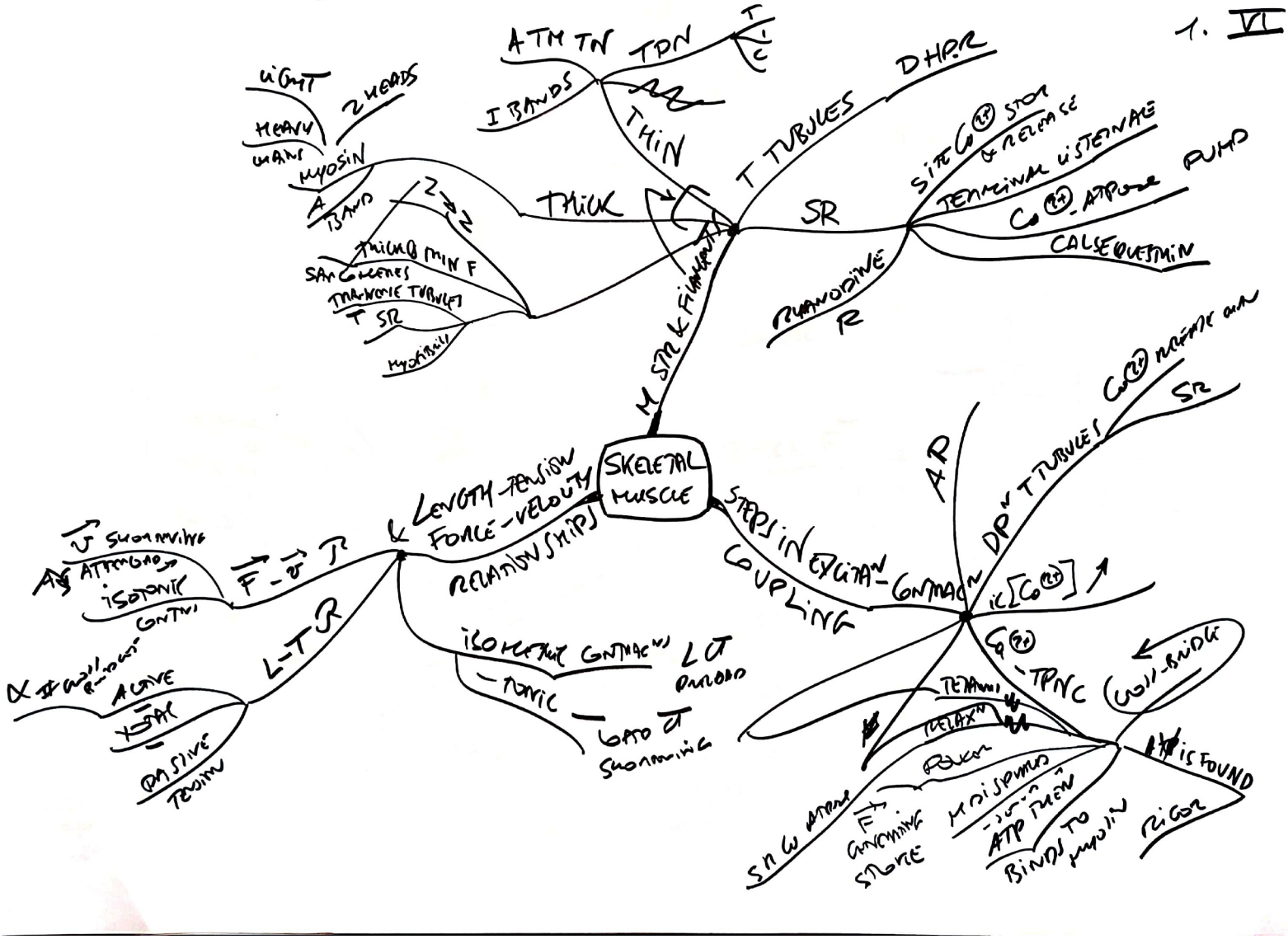

NMJⁿ
ACh | Nicotinic R
Autom Skel
CoA
Acetyl CoA + Choline → ACh
Choline Acetyl Transferase
ACh
Synaptic vesicles
ATP Proteoglycan
For later release
Synthesise ACh in pres terminal
DPⁿ pres term & Ca2+ uptake
AP DP
Opens Ca2+ channels
Down its EC gradient
Ca2+ uptake => release ACh into syn cleft
Fuse
Exocytosis
Diffusion ACh to posts MB (muscle end plate)
α subunits
R
& ACh-R nicotinic
Conformational change
Na+
K+
Channel
Ligand-gated channels
EPP (end plate potential) in posts MB
Counts 1 syn vesicle (one quantum)
MEPP (miniature end plate potential)
Smallest possible
Na+
K+
Both
DP value halfway between E Na+ & E K+
* NOMV
Specialized muscle EP
Simply DPⁿ
Summate → full fledged EPP not a AP
DP adjacent M to threshold
Degradation ACh
EPP transient
Neostigmine
To ↓ degradation, prolong its ACh → ↑ size EPP
AChE ⊖
ACh → CoA + choline
Na choline cot
1/2
Many cholinium & depleted pres endings of ACh stores
Sterase
ChC => DPⁿ & APs in adj muscle
AP → continuation
Over expanded DP

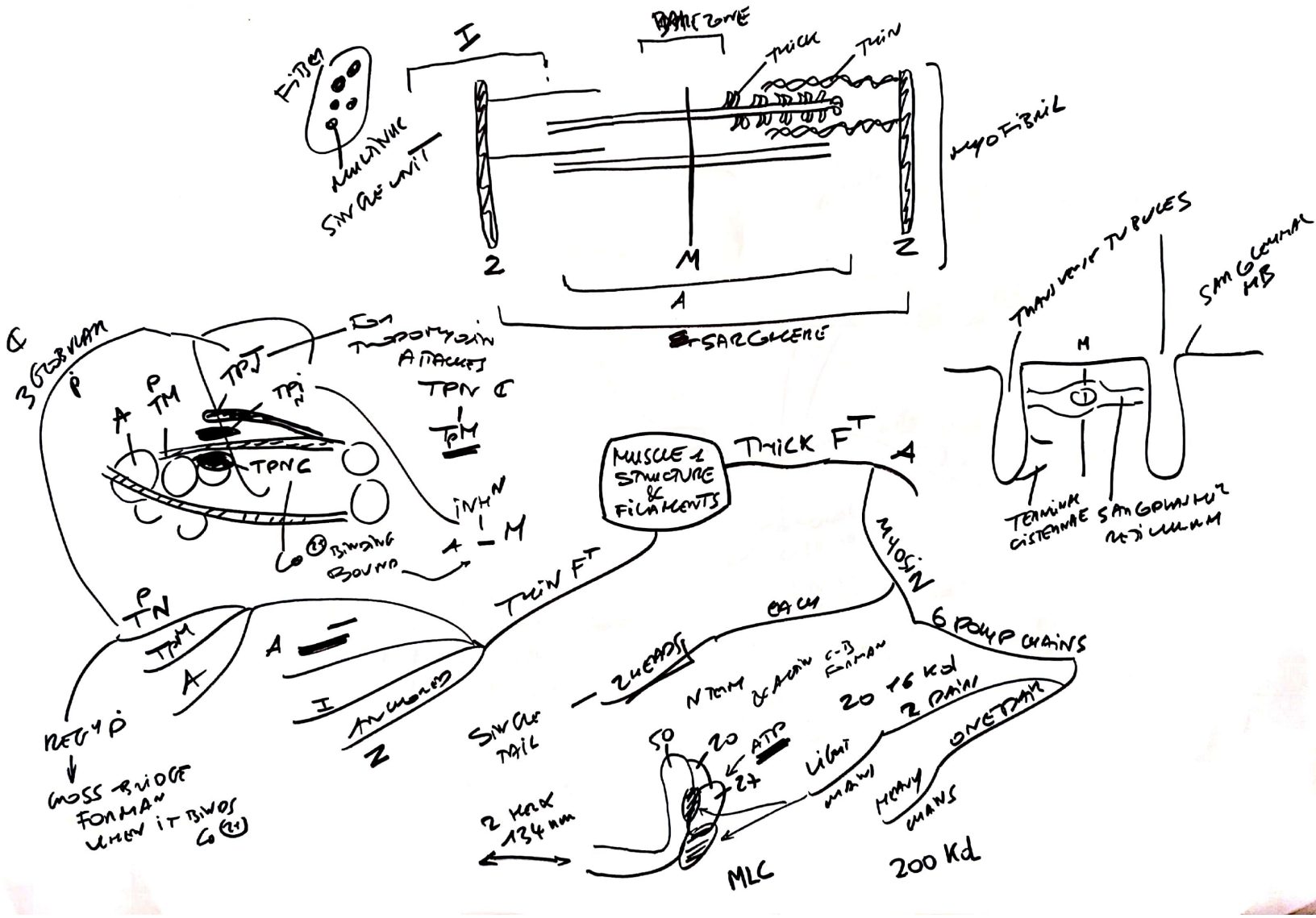

Hemicholinium depletes
ACh stores
AP
Choline
ACh
Ca2+
Na
K
R
DPN EP
AP IN M
Motor end plate
Total blockade
Botulinum toxin
Compete with ACh for R
Curare
↓ size EPP
May cause paralysis of resp mm
Myasthenia gravis
NMJ 2
Weakness & fatigability
AChR on MEP
Size EPP ↓ ⇒ more difficult EP to threshold & produce AP
AChE inh
Neostigmine
Prevent degradation ACh & prolongs action ACh at MEP
Partially compensates for
ACh AChE → CoA + choline
Neostigmine prolongs & ↑ action ACh at muscle end plate
Agents

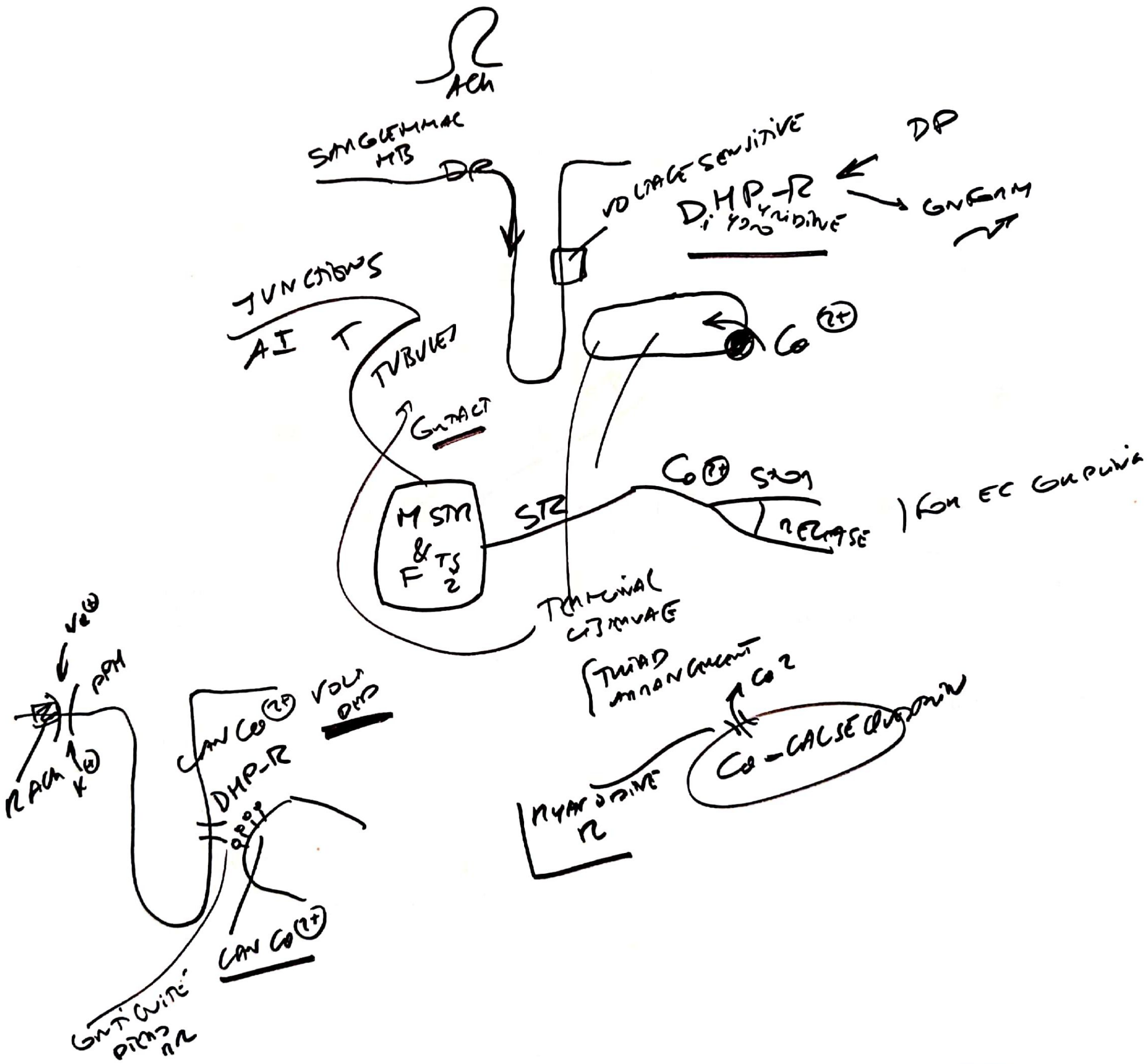


SYNAPTIC TRANSMISSION
TYPES OF ARRANGEMENTS (1)
1 TO 1 NMJ
AP
MOTOR NERVE
AP
MUSCLE
MANY TO ONE SYNAPSES
SPINAL MOTO NEURONS
AP
SINGLE PRES
INSUFFICIENT AP POSTS
MANY SYNAPSE TO DP TO THRESHOLD
PRE SYN
+ or −
INPUT TO SYNAPSES (2)
POST
INTEGRATES EXC & INH INPUTS
INHIBITORY IPSPs
EXCITATORY EPSPs
POTENTIALS
POST SYNAPTIC
Σ INPUT BRINGS MB POT OF POSTS TO T → AP
DP → T → CLOSE FIRING AP
OPENING CHANNELS
NT
Ach
NE
E
D
GLU
SEROT
HALFWAY BETWEEN
~ Ach CHANNELS
NT
GLYCINE
GABA
γ AMINOBUTYRIC ACID
OPENING Cl CHAN
HP
Cl
MB POT MP TOWARDS ECl
−90 mV
SUMMATION AT SYNAPSES (3)
SPATIAL
TEMPORAL
RAPID SUCCESSION
POST TETANIC POTENTIATION
TETANIC STIMULATION OF PRES
PRE
DP GREATER THAN EXPECTED BECAUSE
NT
POSSIBLY BECAUSE ACCUMULATION Ca 2+ IN PRES TERMINAL
LT POTENTIATION
(MEMORY)
NT


NT
NE
E
D
TY NT
METHYL GROUP TRANSFERRED TO NE FROM S ADENOSYLMET
MIDBRAIN
TYROSINE
TYR HYDROXYLASE
L-DOPA
DOPA CARBOXYLASE
D
D β HYDROXYLASE
NE
PHENYLETHANOLAMINE N-METHYL TRANSFERASE
E
SEROT
TRP
BS
MELATONIN IN PINEAL
HIST
HISTIDINE
HT
PRL SECN
PIF
MET BY MAO
COMT
D1R AC Gs
D2R AC Gi
PK
SCHIZOPHRENIA
THAT USE
ACh
NO
ALSO
ST OF GC
VASC
SHORT ACTING
CNS
NO PERMEANT GAS
B/VES
Gi
PRES
NO SYNTHASE
ARGININE → CITRULLINE NO
GLYCINE
BS
SC
GABA
R
A
B
BDZ
BARB
SITE ON
K⊕ CONDUCT
Cl⊖ CONDUCT
COUPLED TO ION CHAN VIA
GP
GLUTAMATE
GLUTAMATE DECARBOXYLASE
GLUTAMATE
4 TYPES R
METABOTROPIC 1
IONOTROPIC 3
NMDA
N METHYL D ASPARTATE
= LIGAND GATED CHANNELS
METABOLITES:
- DOMA
- NMN
- MOPEG
- VMA
PHEOCHROMOCYTOMA
CAT
MAO
MONOAMINE OXIDASE
COMT
CATECHOL O METHYL TRANSFERASE
BY
IN PRES TERMINAL
MET'D OR REUPTAKE
REMOVED
SYMP
α R
β R

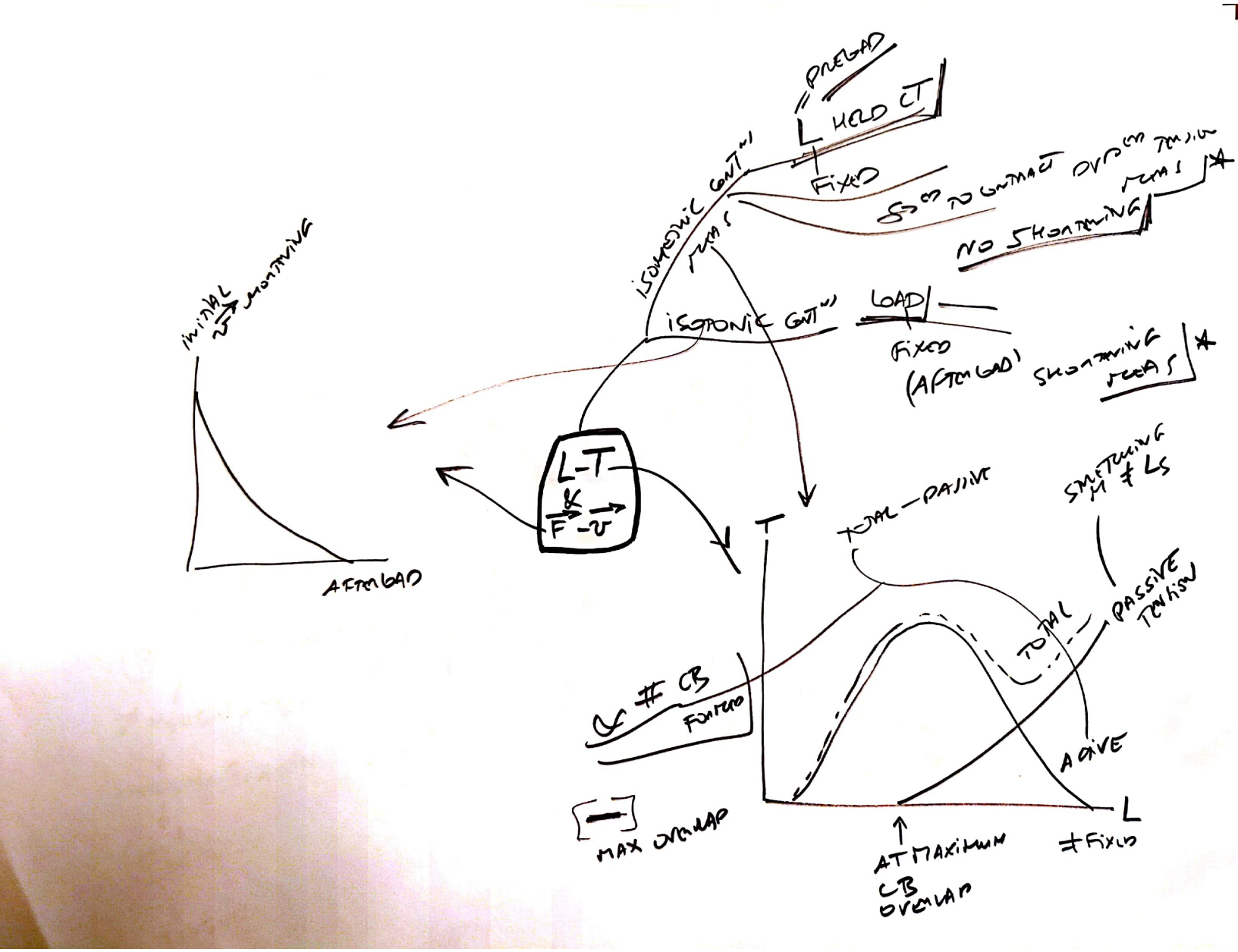

SKELETAL MUSCLE
M STR & FILAMENTS
ATM TN
TPN
T/C
I BANDS
THIN
LIGHT
HEAVY CHAIN
2 HEADS
MYOSIN
A BAND
Z → Z
THICK
THICK MIN F
SARCOLEMES
TRANSVRSE TUBULES
T SR
MYOFIBRILS
T TUBULES
DHPR
SR
SITE Ca++ STOR & RELEASE
TERMINAL CISTERNAE
Ca++ ATPase PUMP
CALSEQUESTRIN
RYANODINE R
LENGTH-TENSION FORCE-VELOCITY RELATIONSHIPS
F-v
ISOTONIC CONTN
ACTIVE
PASSIVE TENSION
L-T R
ISOMETRIC CONTRACN
TONIC
LOAD
STEPS IN EXCITATION-CONTRACTION COUPLING
AP
T TUBULES
Ca++ RELEASE CHAN
SR
DHPR
Ca++ [Ca++] ↑
Ca++ -TPNC
TROPONIN
TROPOMYOSIN
TENSION
RELAXATION
Ca++ BINDS
IS FOUND
RIGOR
ATP THEN
BINDS TO MYOSIN
Ca++ STORAGE
SR W ATPase

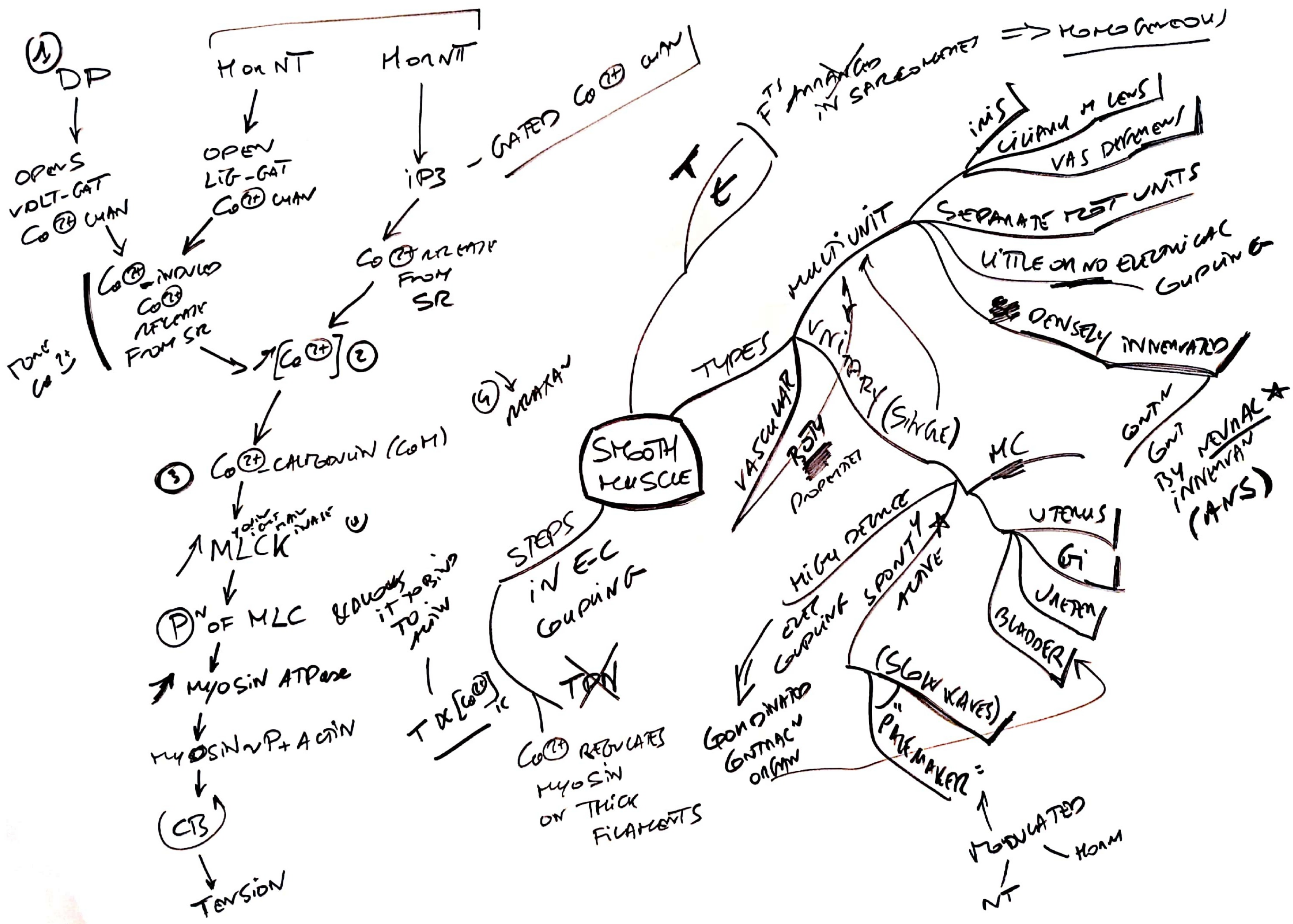

I
A BAND ZONE
THICK
THIN
FIBER
MYOFIBRIL
SINGLE UNIT
Z
M
A
SARCOLEMMA
TRANSVERSE TUBULES
SARCOLEMMAL MB
TERMINAL CISTERNAE
SARCOPLASMIC RETICULUM
MUSCLE STRUCTURE & FILAMENTS
THICK F
THIN F
MYOSIN
6 POLYP CHAINS
2 HEADS
EACH
SINGLE TAIL
20 TO KD
2 PAIRS
ONE PAIR
LIGHT CHAIN
HEAVY CHAINS
200 KD
MLC
2 NM 134 nm
ATP
TROPONIN
TPC
TPI
TPT
TPM
TPN
INHIBITORY
BINDING SOUND
CROSS BRIDGE FORMATION



ACh
DP
VOLTAGE SENSITIVE
DHP-R
DP
JUNCTIONS
T
TUBULES
CONTACT
Ca 2+
Ca 2+ STOR
RELEASE
STR
TERMINAL
TRIAD ARRANGEMENT
Ca-CALSEQUESTRIN
R
AP
K+
CAN Ca 2+
DHP-R
CAN Ca 2+

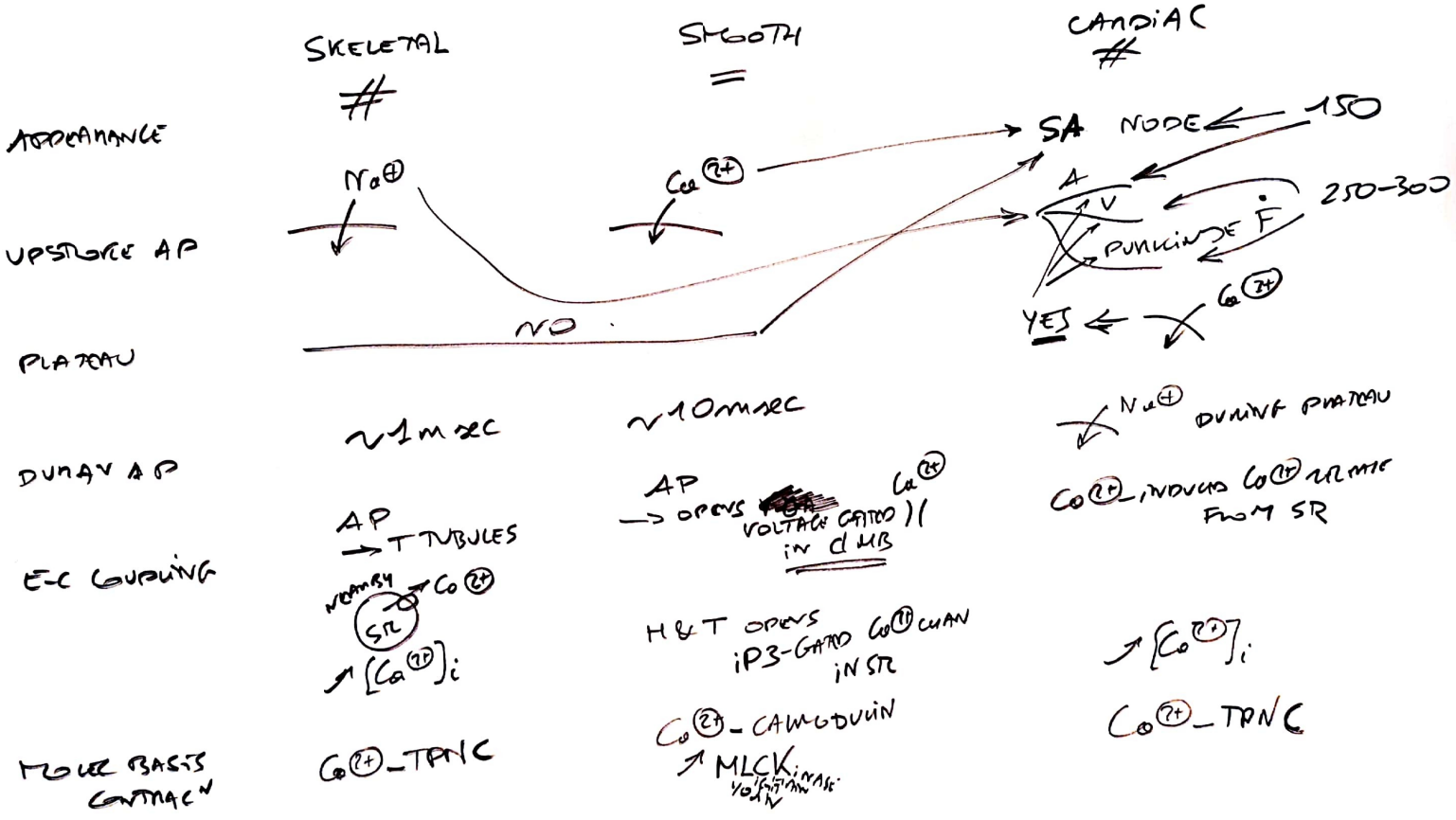

STEPS IN EXCITAT'N CONTRAC'N COUPLING
AP
DP T TUBULES
IC [Ca2+] ↑
Ca2+ - TPNC ⇒ CONFORM ∆ IN TPN THAT MOVES TPM OUT OF THE WAY
CROSS-BRIDGE BEGINS
RELAXATION
TETANUS
MEC
SINGLE TWITCH
SINGLE AP
STANDARD AMOUNT
SERCA
SR
Ca2+
Ca2+ -X- TPNC
TPM BL A
BINDING SITE
CB CANNOT OCCUR
INITIATE
DP
CONFORM ∆
DHP-R
OPENS Ca2+ RELEASE CHAN
RR
IN NEARBY SR
ATP BOUND
RIGOR
ADP
RIGOR STATE
M
NEW SITE
ADP
POWER STROKE
REPEATS AS LONG Ca2+ TPNC
ATP
Pi
DISPLACED
REMAINS ATTACHED
ATP-M
CONFORM ∆ M
M TO BE RELEASED FROM A


PRELOAD
HELD CT
FIXED
ISOMETRIC CONT"
NO SHORTENING
ISOTONIC CONT"
LOAD
FIXED
(AFTERLOAD)
SHORTENING
INITIAL
SHORTENING
AFTERLOAD
L-T
F-V
T
TOTAL - PASSIVE
SHORTENING
PASSIVE TENSION
TOTAL
ACTIVE
CB
FIXED
MAX OVERLAP
AT MAXIMUM CB OVERLAP
L
FIXED



1 DP
OPENS VOLT-GAT Ca2+ CHAN
HOR NT
OPEN LIG-GAT Ca2+ CHAN
HOR NT
IP3 - GATED Ca2+ CHAN
Ca2+ RELEASE FROM SR
Ca2+-INDUCED Ca2+ RELEASE FROM SR
TONIC Ca2+
[Ca2+] 2
4 ↓ RELAXN
3 Ca2+-CALMODULIN (CaM)
MLCK
P^N OF MLC ALLOWS IT TO BIND TO ACTIN
MYOSIN ATPase
MYOSIN~P + ACTIN
CB
TENSION
SMOOTH MUSCLE
STEPS IN E-C COUPLING
T ∝ [Ca2+]ic
TPN
Ca2+ REGULATES MYOSIN ON THICK FILAMENTS
F^TS ARRANGED IN SARCOMERES => HOMOGENEOUS
TYPES
MULTI UNIT
IRIS
CILIARY M LENS
VAS DEFERENS
SEPARATE FXT UNITS
LITTLE OR NO ELECTRICAL COUPLING
DENSELY INNERVATED
CONTN CONT BY NEURAL INNERVATION (ANS)
UNITARY (SINGLE)
VASCULAR
BOTH
MC
UTERUS
GI
URETER
BLADDER
HIGH DEGREE ELEC COUPLING
SPONTY ACTIVE
COORDINATED CONTRACTN ORGAN
(SLOW WAVES)
PACEMAKER
MODULATED
NT
HORM

COMPARISON


SKELETAL
SMOOTH
CARDIAC
APPEARANCE
UPSTROKE AP
PLATEAU
DURATION AP
E-C COUPLING
MOTOR BASIS CONTRACTION
Na+
Ca2+
SA NODE
150
A
V
250-300
PUMPING DE F
YES
NO
~1msec
~10msec
AP
→ T TUBULES
VOLTAGE GATED
IN d MB
Ca2+ INDUCED Ca2+ RELEASE FROM SR
SR
[Ca2+]i
Ca2+-TPNC
Ca2+-CALMODULIN
MLCK