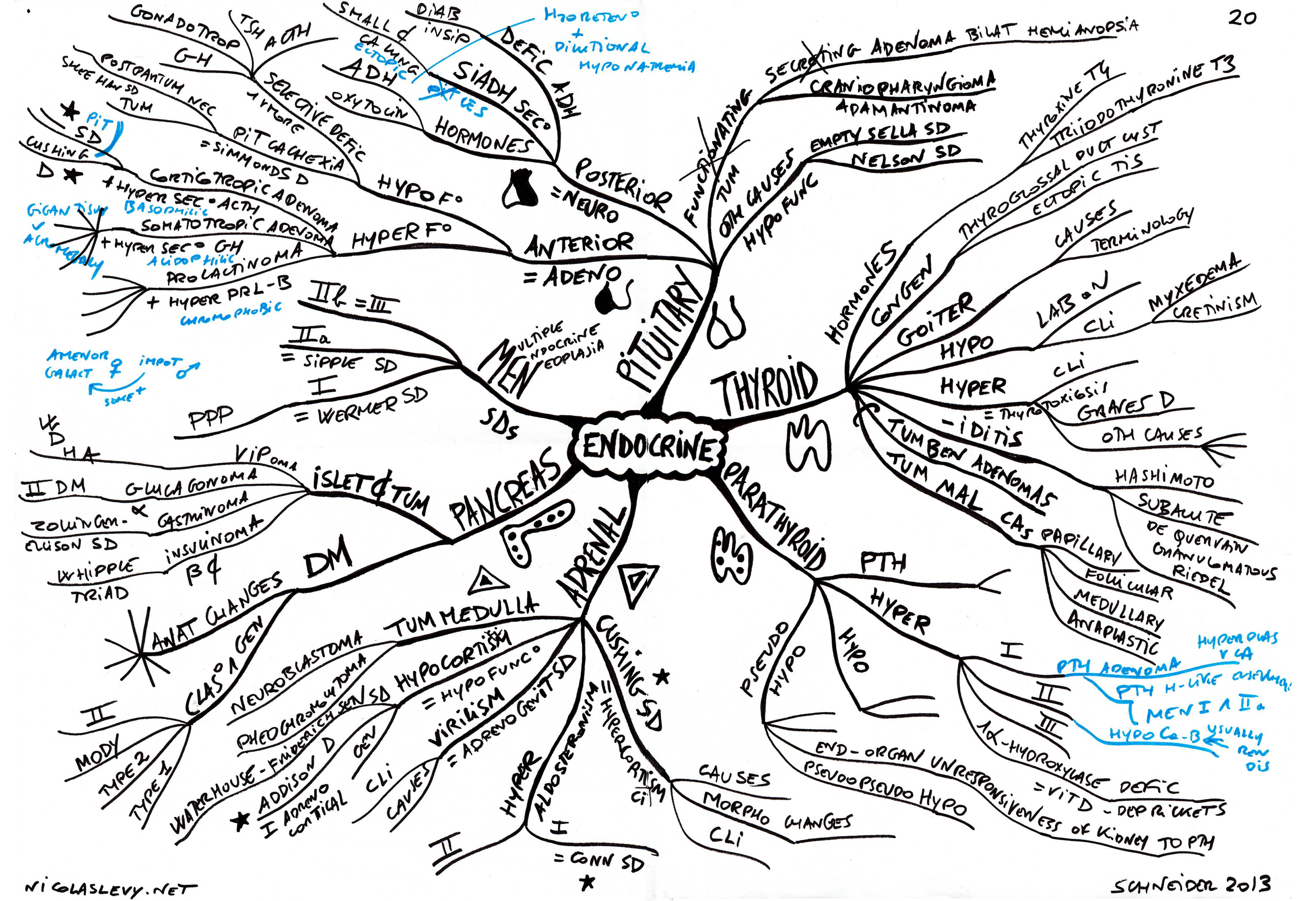
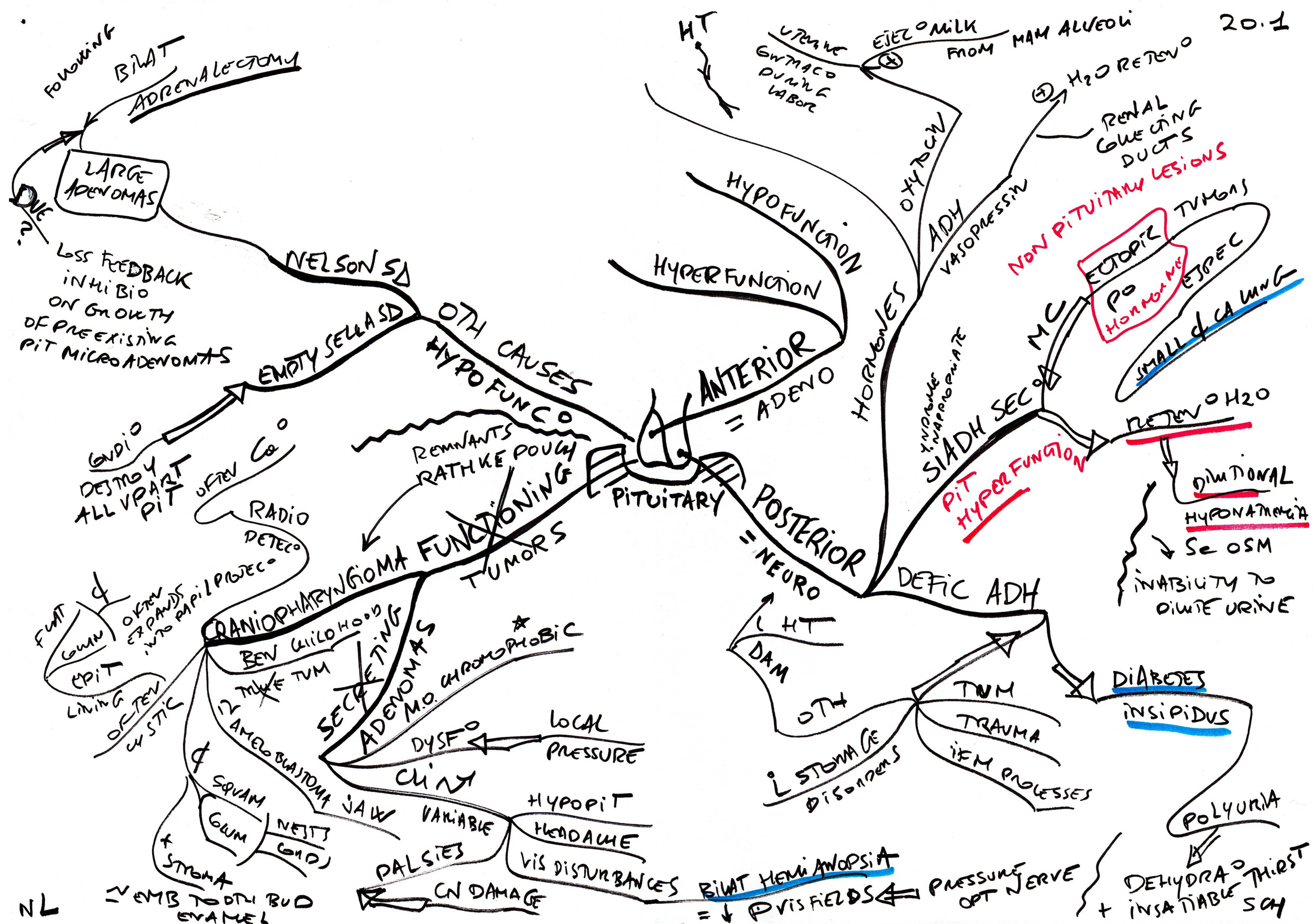
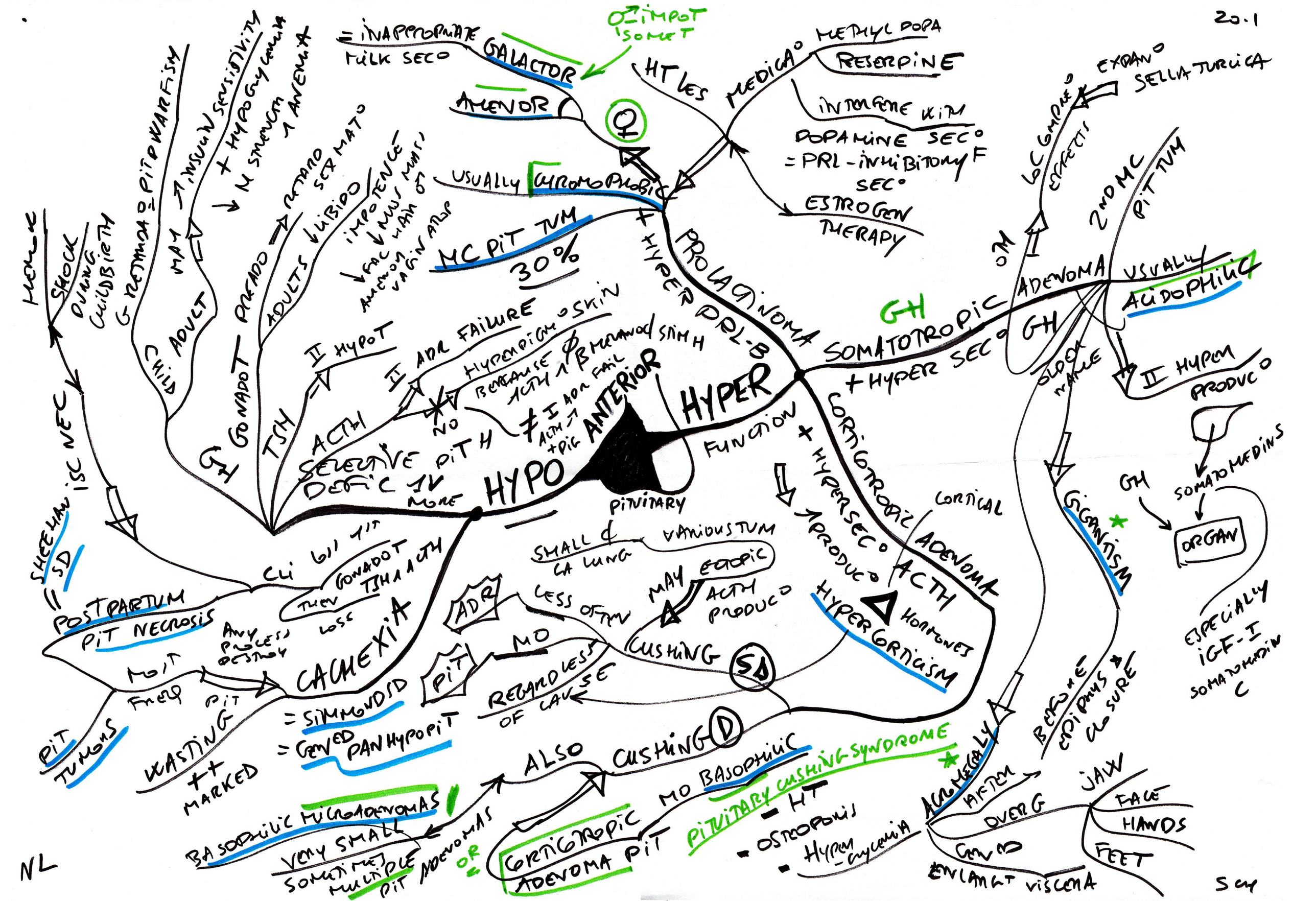
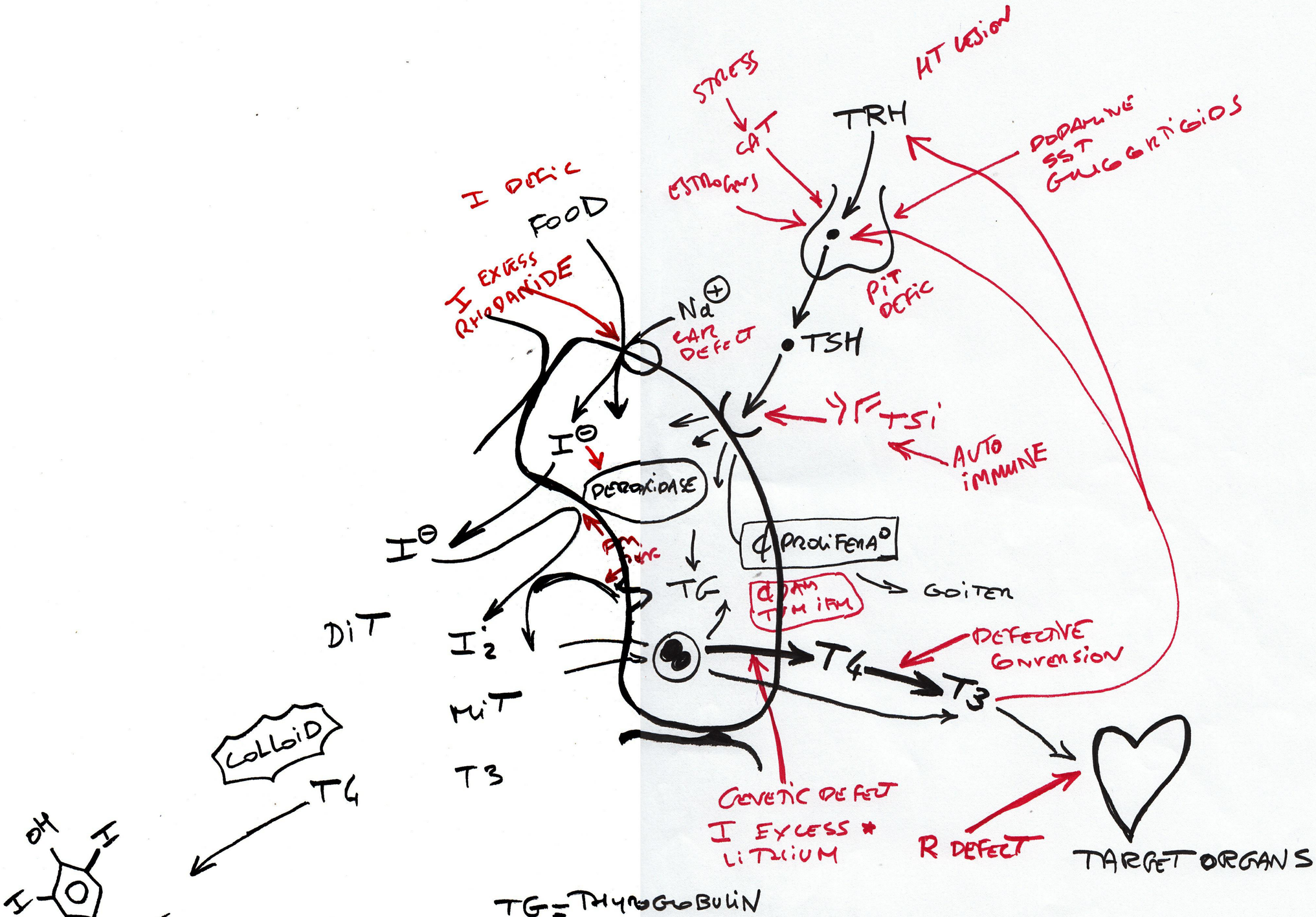


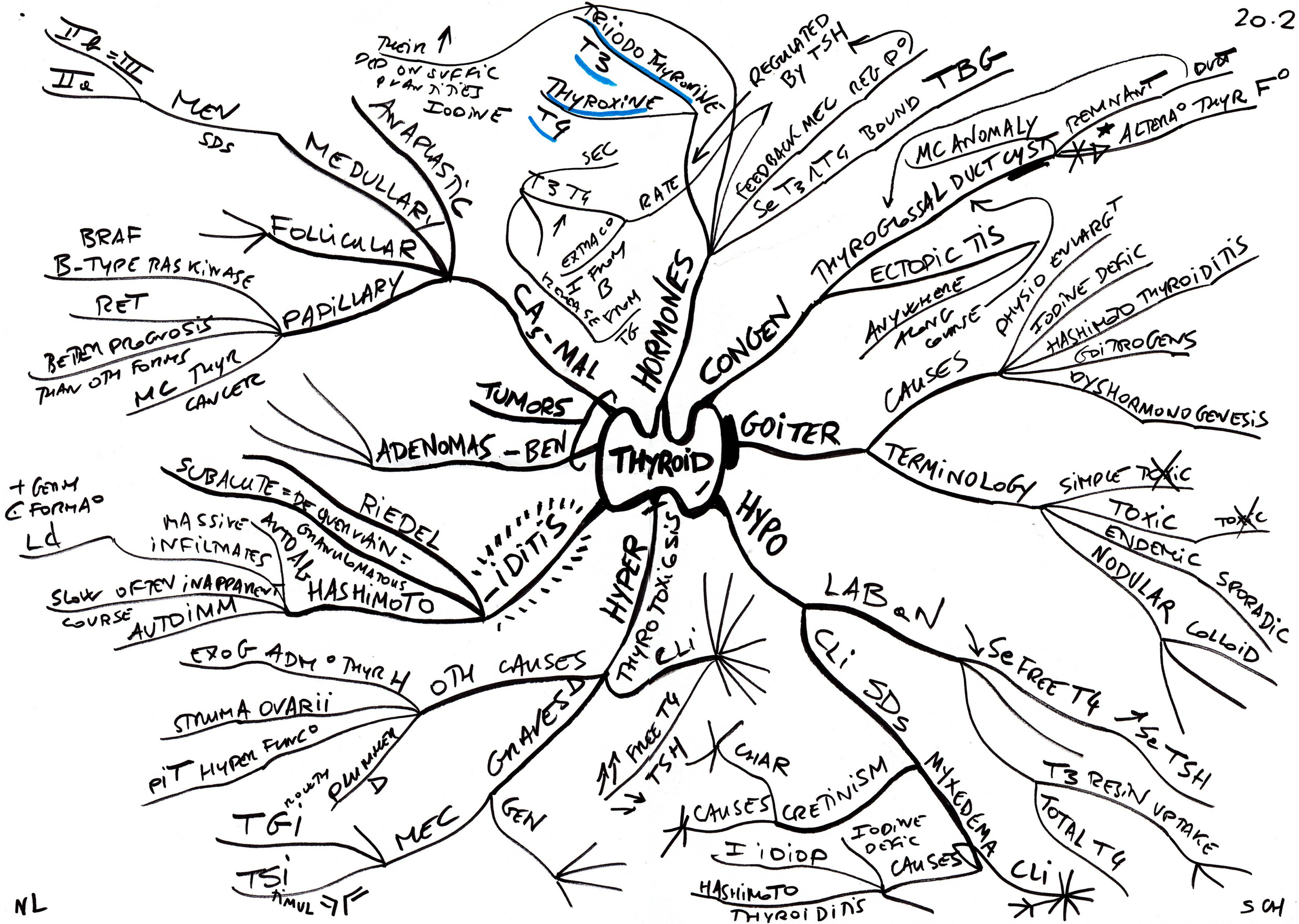
NICOLASLEVY.NET

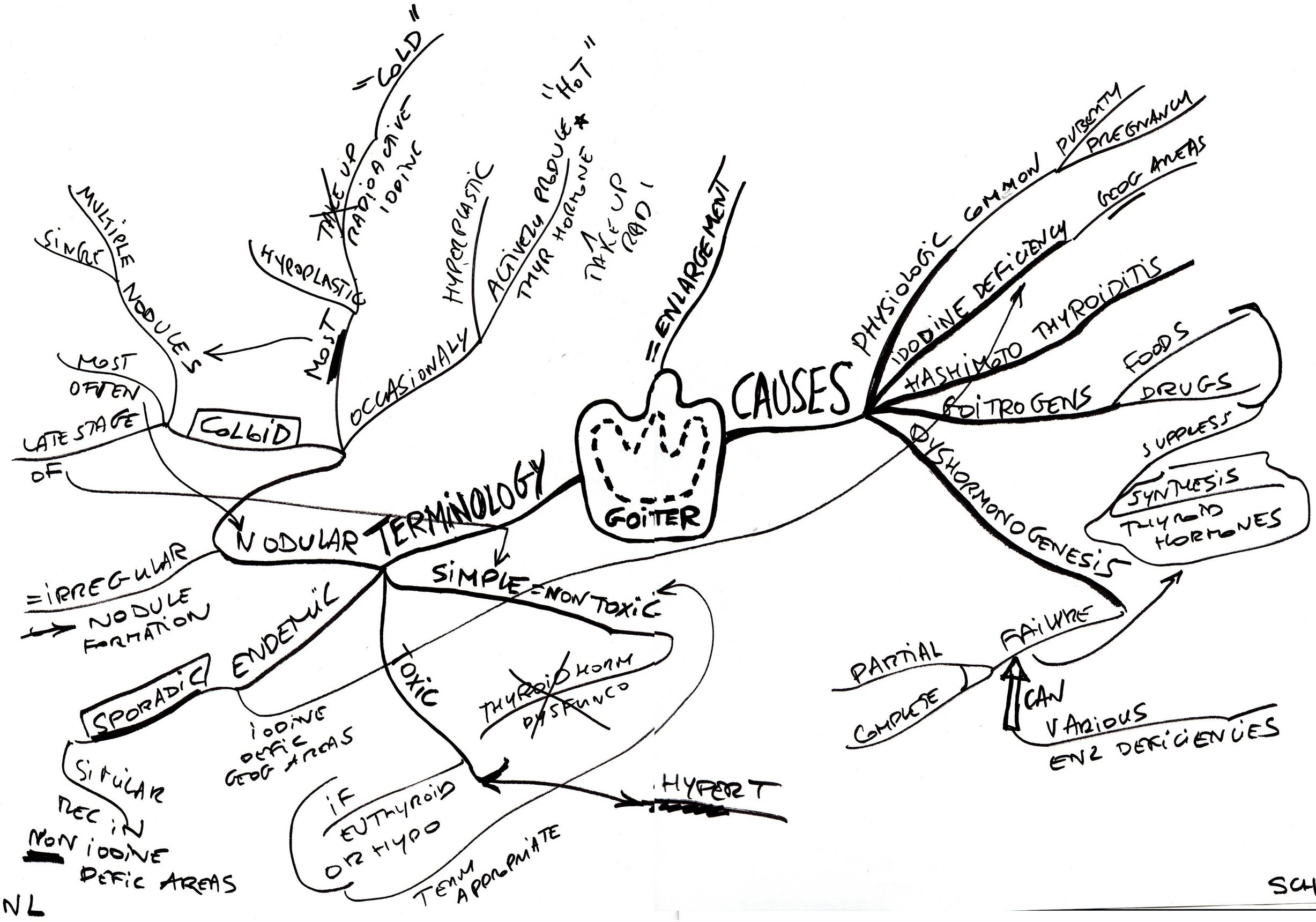
SCHNEIDER 2013

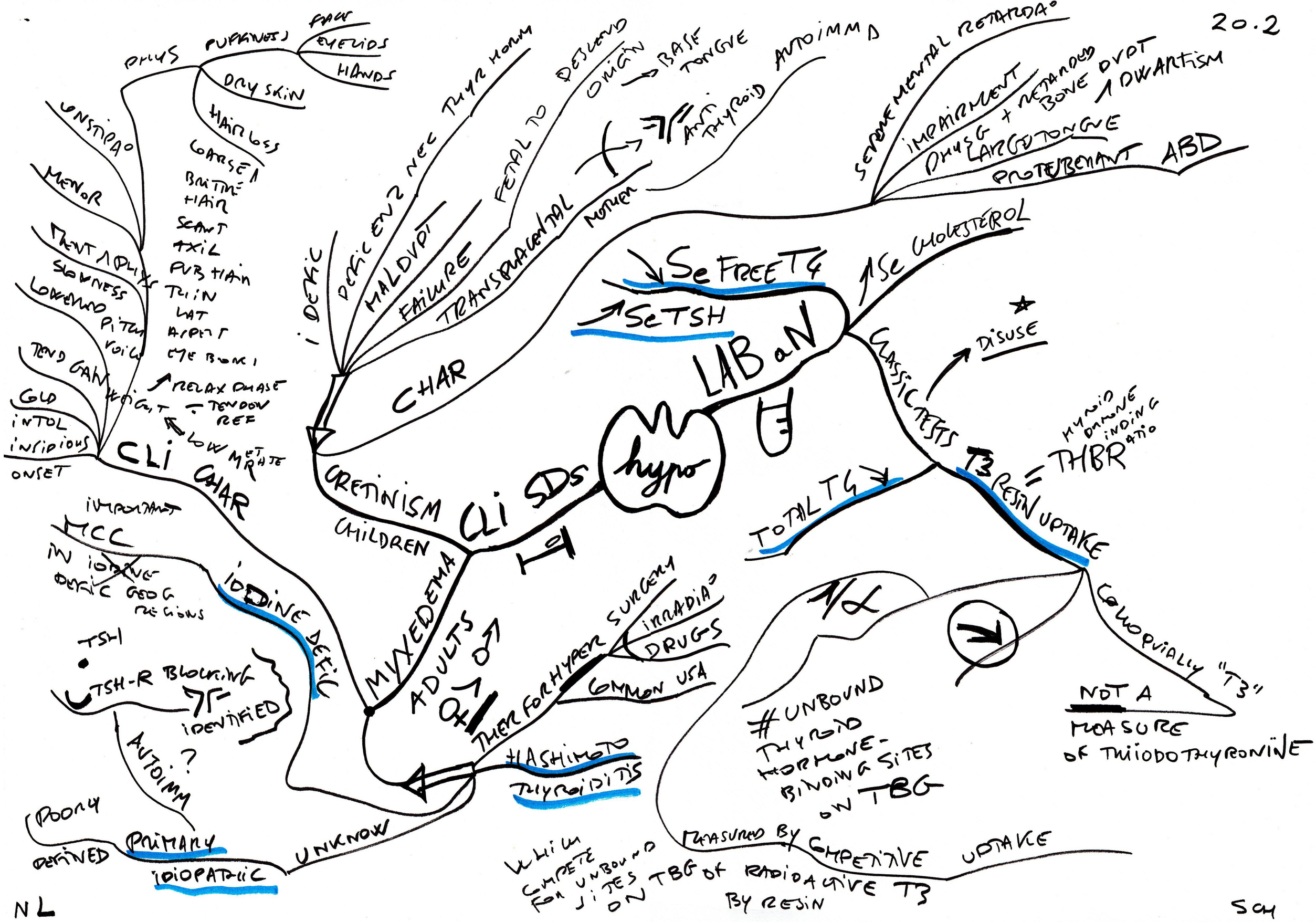

FOLLOWING
BILAT ADRENALECTOMY
LARGE ADENOMAS
DUE
LOSS FEEDBACK INHIBIO ON GROWTH OF PREEXISTING PIT MICROADENOMAS
NELSON'S
EMPTY SELLA SD
GNDIO
DESTROY ALL VPART PIT
OFTEN Co
RADIO
DETECO
OTH CAUSES HYPOFUNCO
REMNANTS RATHKE POUCH
CRANIOPHARYNGIOMA
FUNCTIONING TUMORS
FUNT
GLAND
EPIT
OFTEN EXPANDS INTO PAPILPROJECO
LINING
OFTEN CYSTIC
BEN CHILDHOOD
MIXE TUM
SECRETING ADENOMAS
M.O. CHROMOPHOBIC
AMELOBLASTOMA JAW
SQUAM
GLAND
NEJTS
GNDJ
STROMA
EMB TO DTH BUD ENAMEL
DYSFO
LOCAL PRESSURE
CLINY
VARIABLE
HYPOPIT
HEADACHE
VIS DISTURBANCES
PALSIES
CN DAMAGE
BILAT HEMIANOPSIA
= ↓ P VISFIELDS
PRESSURE OPT NERVE
ANTERIOR = ADENO
HYPOFUNCTION
HYPERFUNCTION
PITUITARY
POSTERIOR = NEURO
HT
DAM
OTH
↓ STOMACE DISORDERS
DEFIC ADH
TUM
TRAUMA
IEM PROCESSES
DIABETES INSIPIDUS
POLYURIA
DEHYDRAO + INSATIABLE THIRST SCH
HORMONES
OTYTOCIN
ADH VASOPRESSIN
UTERINE CONTACO DURING LABOR
EJEC° MILK FROM MAM ALVEOLI
H2O RETENO
RENAL COLLECTING DUCTS
NON PITUITARY LESIONS
ECTOPIC PO HORMONE
TUMORS
ESPEC SMALL CA LUNG
MC
SIADH SEC°
SYNDROME INAPPROPRIATE
PIT HYPERFUNCTION
RETENO H2O
DILUTIONAL HYPONATREMIA
Se OSM
INABILITY TO DILUTE URINE
NL


HYPER
HYPO
ANTERIOR
PITUITARY
PROLACTINOMA
HYPER PRL-B
HT LES MEDICA°
METHYL DOPA
RESERPINE
INTOLERENCE KITM
DOPAMINE SEC°
= PRL-INHIBITORY F
SEC°
ESTROGEN THERAPY
GALACTOR
= INAPPROPRIATE MILK SECO
AMENOR
USUALLY GYNOMOPHOBIC
MC PIT TUM
30%
IMPOT
ISOMET
SOMATOTROPIC ADENOMA
GH
+ HYPER SEC°
USUALLY ACIDOPHILIC
II HYPOM
PRODUC°
SOMATOMEDINS
ORGAN
GH
ESPECIALLY IGF-I
SOMATOMEDIN C
GIGANTISM
BEFORE EPIPHYS CLOSURE
ACROMEGALY
JAW
FACE
HANDS
FEET
OVERG
GENID
ENLARGT VISCERA
AFTER
CORTICOTROPIC ADENOMA
HYPERSEC° ACTH
PRODUC°
HYPERCORTICISM
HORMONES
CORTICAL
MO BASOPHILIC
PITUITARY CUSHING SYNDROME
- HT
- OSTEOPOROSIS
- HYPERGLYCEMIA
CUSHING D
SD
ADR
MO
REGARDLESS OF CAUSE
MAY ECTOPIC ACTH PRODUC°
LESS OFTEN
SMALL CA LUNG
VARIOUS TUM
LOC COMPR°
EXPAN°
SELLA TURCICA
EFFECTS
2ND MC PIT TUM
SLOW GROWTH
II HYPOM
II ADR FAILURE
HYPERPIGM° SKIN
BECAUSE ACTH ↑ β MELANOCYTE STIMH
II HYPOT
ACTH
TSH
GH
GONADOT
PREADO
ADULTS
LIBIDO
IMPOTENCE
FAC HAIR
AMENOR
VAGIN ATROP
SEX MAT°
RETARD
INSULIN SENSITIVITY
HYPOGLYCEMIA
ANEMIA
SMENOR
RETINOPATHY
PITUITARY
CHILD
ADULT
MAT
SHOCK
DURING CHILDBIRTH
HEMOR
SHEEHAN ISC NEC
SD
POSTPARTUM
PIT NECROSIS
CACHEXIA
MOST
FREQ
PIT
ANY PROCESS DESTROY
LOSS
THEY GONADOT TSH ACTH
CLI GOI FIT
WASTING
++ MARKED
= SIMMONDS D
= GENED PANHYPOPIT
PIT TUMORS
BASOPHILIC MICROADENOMAS
VERY SMALL
SOMETIMES MULTIPLE PIT ADENOMAS
OR
CORTICOTROPIC ADENOMA PIT
ALSO CUSHING D
NO
BECAUSE ACTH ↑ + PIG
≠ I ADR FAIL
ACTH ↑
SELECTIVE PIT H DEFIC IV
MORE
NL
SCH


HT LESION
STRESS
CAT
TRH
DOPAMINE
SST
GLUCOCORTICOIDS
ESTROGENS
I DEFIC
FOOD
PIT DEFIC
I EXCESS
RHODANIDE
Na⊕
CAR DEFECT
TSH
TSI
AUTO IMMUNE
I⊖
PEROXIDASE
I⊖
PROLIFERA°
TG
GOITER
DiT
I2
T4
DEFECTIVE CONVERSION
T3
MiT
T3
COLLOID
TG
GENETIC DEFECT
I EXCESS *
LITHIUM
R DEFECT
TARGET ORGANS
TG=THYROGLOBULIN
OH
I
I
O
I
I
TG

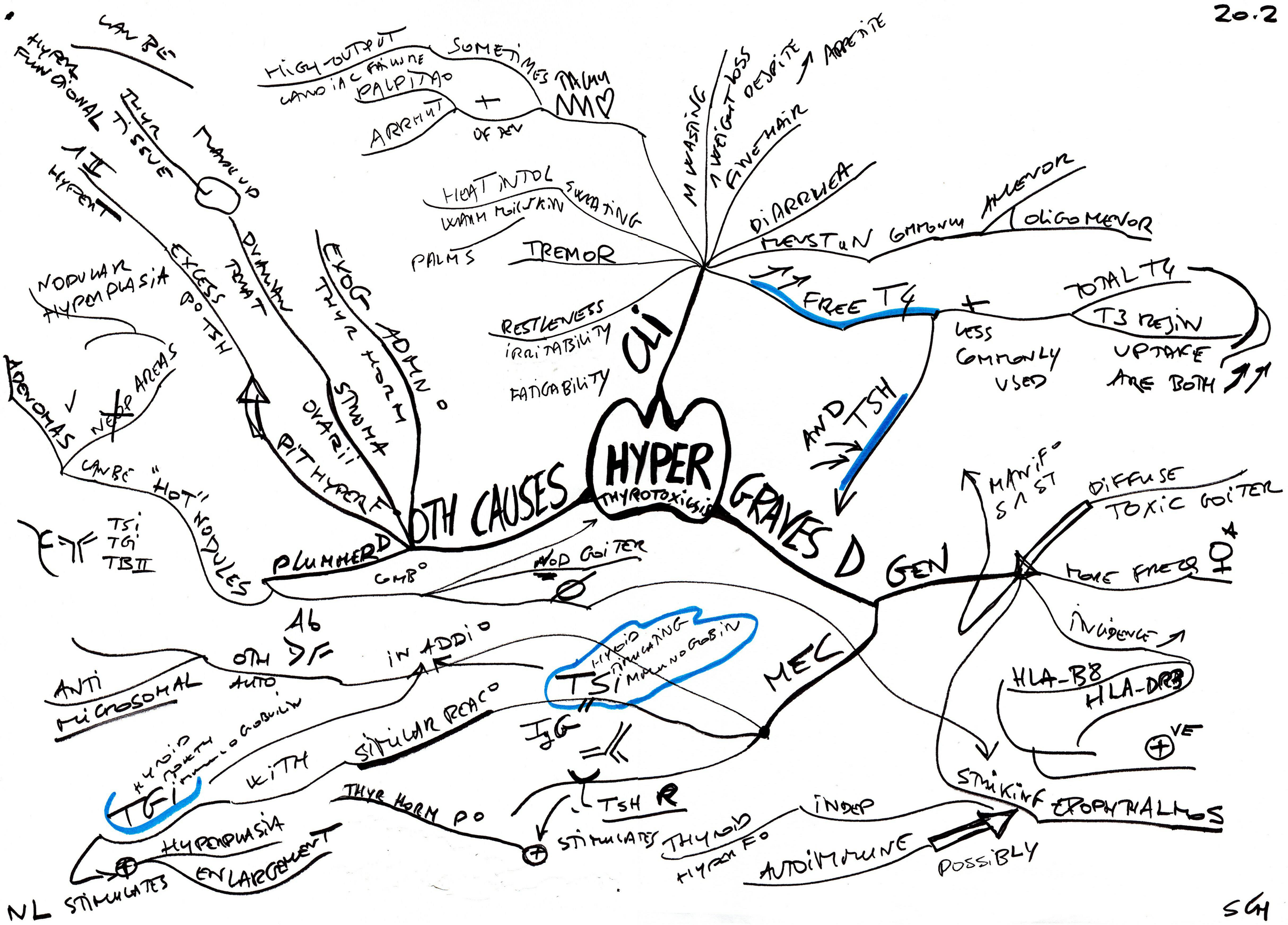

THYROID
HORMONES
TRIIODO THYRONINE
T3
THYROXINE
T4
REGULATED BY TSH
FEEDBACK MEC REG P°
TBG
Se T3 & T4 BOUND
SEC
T3 T4
RATE
EXTRA C°
FROM B
INCREASE FROM TG
CAS-MAL
CONGEN
THYROGLOSSAL DUCT CYST
MC ANOMALY
REMNANT
DUCT
ALTERN° THYR F°
ECTOPIC TIS
ANYWHERE ALONG COURSE
CAUSES
PHYSIO ENLARG^T
IODINE DEFIC
HASHIMOTO THYROIDITIS
GOITROGENS
DYSHORMONOGENESIS
GOITER
TERMINOLOGY
SIMPLE TOXIC
TOXIC
ENDEMIC
SPORADIC
NODULAR
COLLOID
HYPO
LAB & N
CLI
SDs
Se FREE T4
Se TSH
T3 RESIN UPTAKE
TOTAL T4
MYXEDEMA
CLI
CAUSES
IODINE DEFIC CAUSES
I IDIOP
HASHIMOTO THYROIDITIS
CRETINISM
CHAR
HYPER
THYROTOXICOSIS
CLI
↑↑ FREE T4
↓ TSH
GRAVES D
GEN
MEC
TGI
TSI
MOUTH
PLUMMER D
OTH CAUSES
EXOG ADM ° THYR H
STRUMA OVARII
PIT HYPER FUNC°
TUMORS
ADENOMAS - BEN
RIEDEL
SUBACUTE = DE QUERVAIN = GRANULOMATOUS
MASSIVE INFILTRATES
AUTO AB
HASHIMOTO
SLOW OFTEN INAPPARENT COURSE
AUTOIMM
IDITIS
ANAPLASTIC
MEDULLARY
MEN
SDs
IIa = III
IIa
FOLLICULAR
BRAF
B-TYPE RAS KINASE
RET
PAPILLARY
BETTER PROGNOSIS THAN OTH FORMS
MC THYR CANCER
Prein ↑
DIP ON SURFAC PLAN DIET
IODINE
+ GENM
C FORMA°
Ld
NL
SCH


GOITER
=ENLARGEMENT
CAUSES
PHYSIOLOGIC
COMMON
PUBERTY
PREGNANCY
GEOG AREAS
IODINE DEFICIENCY
HASHIMOTO THYROIDITIS
FOODS
DRUGS
GOITROGENS
DYSHORMONOGENESIS
SUPPRESS
SYNTHESIS
THYROID HORMONES
FAILURE
PARTIAL
COMPLETE
CAN
VARIOUS
ENZ DEFICIENCIES
NODULAR TERMINOLOGY
=IRREGULAR
NODULE FORMATION
ENDEMIC
SPORADIC
SIMPLE =NONTOXIC
TOXIC
THYROID HORM DYSFUNCT
IF EUTHYROID OR HYPO
TERM APPROPRIATE
HYPER T
IODINE DEFIC GEOG AREAS
SIMILAR REC IN NON IODINE DEFIC AREAS
COLLOID
MULTIPLE NODULES
SINGLE
MOST OFTEN
LATE STAGE OF
MOST
HYPOPLASTIC
TAKE UP RADIOACTIVE IODINE
=COLD"
OCCASIONALLY
HYPERPLASTIC
ACTIVELY PRODUCE THYR HORMONE
"HOT"
TAKE UP RAD I
NL
SCH

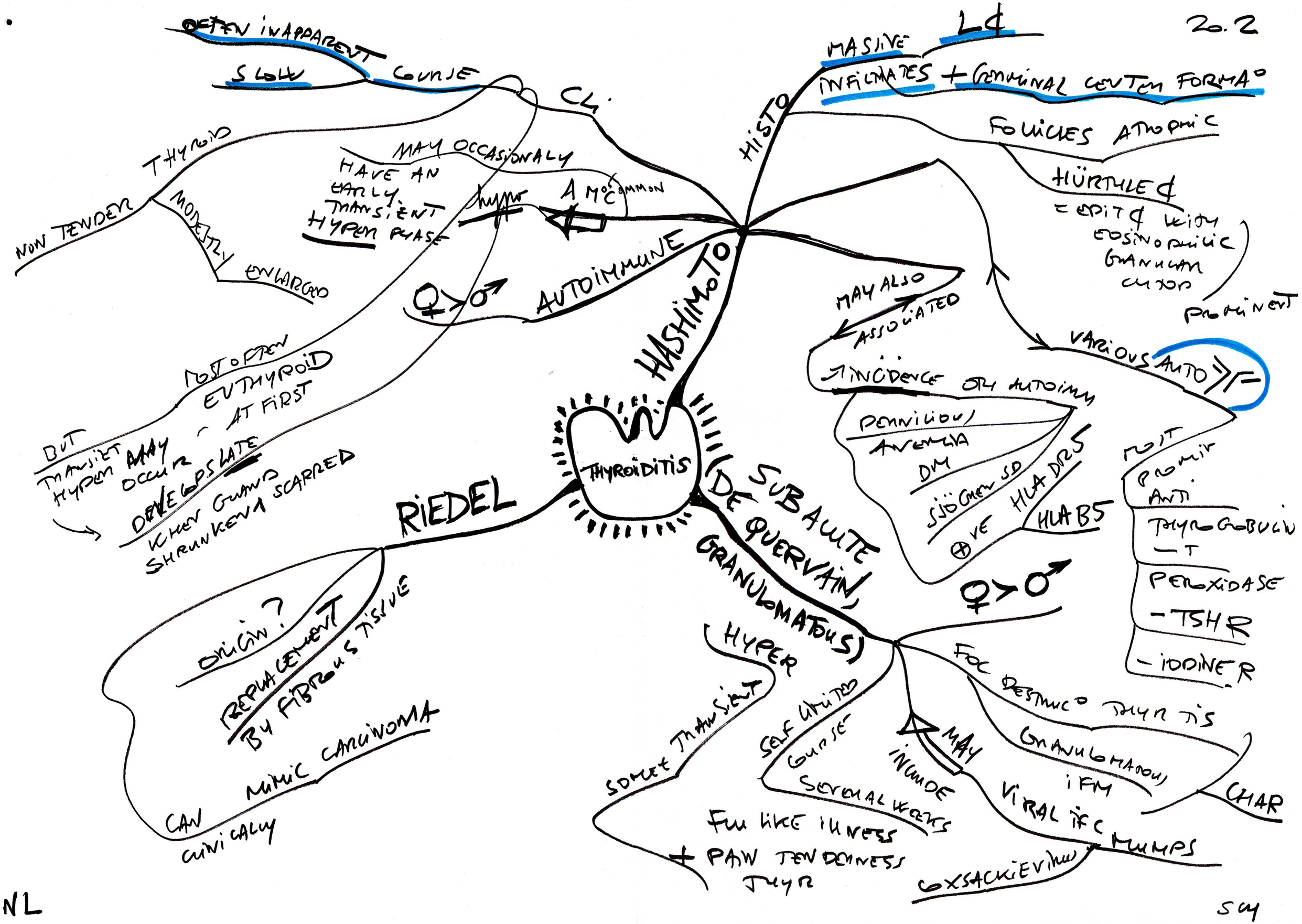

PHYS
PUFFINESS
FACE
EYELIDS
HANDS
DRY SKIN
HAIR LOSS
COARSE & BRITTLE HAIR
SCANT AXIL PUB HAIR
TRIN LAT
EYEBROWS
CONSTIPAO
MENOR
MENT APLIAS
SLOWNESS
LOW BLOOD PITCH VOICE
TEND GAIN
GLD
INTOL
INSIDIOUS ONSET
CLI CHAR
RELAX PHASE
TENDON REF
LOW MET RATE
IDENTIC
DEFIC ENZ NEC THYR HORM
MALDVPT
FAILURE
TRANSPLACENTAL
FETAL TO DESCEND ORIGIN
BASE TONGUE
MOTHER
ANTI THYROID
AUTOIMM D
SEVERE MENTAL RETARDAO
IMPAIRMENT
PHYSG + RETARDED BONE DVPT
DWARFISM
LARGE TONGUE
PROTUBERANT ABD
CHAR
SeFREET4
SeTSH
LAB aN
SeCHOLESTEROL
CLASSICAL TESTS
DISUSE
hypo
CRETINISM
CHILDREN
CLI SDS
TOTAL T4
T3 RESIN UPTAKE
HYPOID DAMONE INDING RATIO
= THBR
IMPORTANT
MCC
IN IODINE DEFIC GEOG REGIONS
IODINE DEFIC
TSH
TSH-R BLOCKING Ab IDENTIFIED
AUTOIMM ?
MYXEDEMA
ADULTS
THERFORHYPER SURGERY
IRRADIA°
DRUGS
COMMON USA
HASHIMOTO THYROIDITIS
COMMONLY "T3"
NOT A MEASURE OF TRIIODOTHYRONINE
UNBOUND THYROID HORMONE-BINDING SITES ON TBG
MEASURED BY COMPETITIVE UPTAKE
POORLY DERIVED
PRIMARY
IDIOPATHIC
UNKNOW
WHICH COMPETE FOR UNBOUND SITES ON TBG OF RADIOACTIVE T3 BY RESIN

NL

SCH

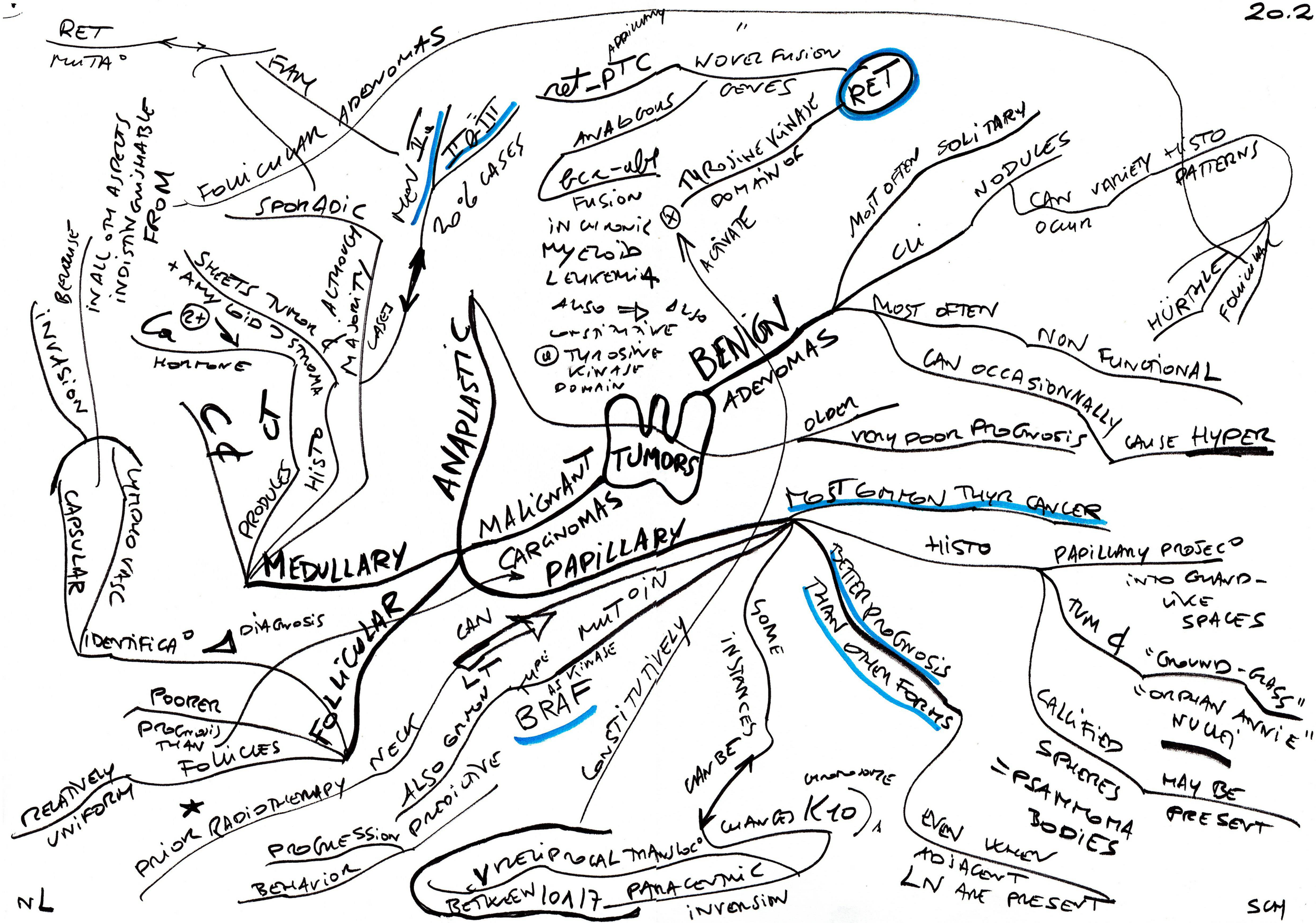

HYPER
THYROTOXICOSIS
GRAVES D
GEN
MEC
OTH CAUSES
HIGH OUTPUT
CARDIAC FAILURE
PALPITATIONS
SOMETIMES
TACHY
ARRHYT
+
OF ATR
HEAT INTOL
SWEATING
WARM MOIST SKIN
PALMS
TREMOR
RESTLENESS
IRRITABILITY
FATIGABILITY
WEASING
WEIGHT LOSS DESPITE ↑ APPETITE
FINE HAIR
DIARRHEA
MENSTRUAL COMPLAINTS
AMENOR
OLIGOMENOR
↑↑ FREE T4
+
LESS COMMONLY USED
TOTAL T4
T3 RESIN
UPTAKE ARE BOTH ↑↑
AND TSH
↑↑
MANIFESTS
DIFFUSE TOXIC GOITER
♀
MORE FREQ
INCIDENCE ↑
HLA-B8
HLA-DR3
⊕ VE
STRIKING EXOPHTHALMOS
POSSIBLY
AUTOIMMUNE
INDEP
THYROID HYPERFO
STIMULATES
⊕
TSH R
THYR HORM PO
IgG
TSI
THYROID STIMULATING IMMUNOGLOBIN
SIMILAR REACO
WITH
TGI
THYROID GROWTH IMMUNOGLOBULIN
⊕
STIMULATES
HYPERPLASIA
ENLARGEMENT
NL
IN ADDIO
AF
OTH AUTO
ANTI MICROSOMAL
Ab
EXOG ADMIN O
THYR HORM
STIMULATE
OVARII
PITHYPERF
DYNAMIC TEST
EXCESS PO TSH
NODULAR HYPERPLASIA
ADENOMAS
NEOP AREAS
CAN BE "HOT" NODULES
PLUMMER D
COMB O
MOD GOITER
∅
TSI
TGI
TBII
THYR TISSUE
HYPERT
THYROID FUNCTIONAL
CAN BE

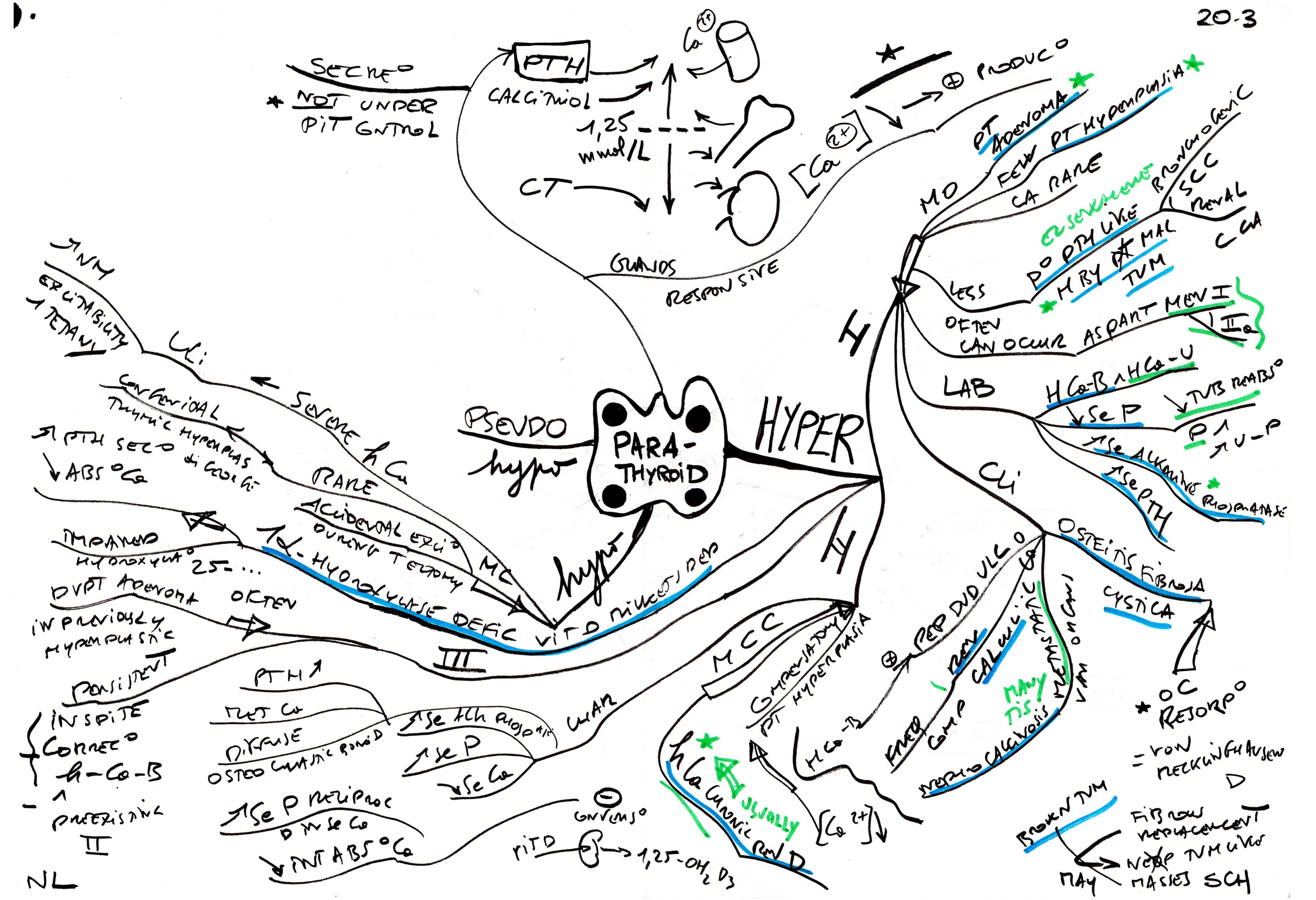
SGH

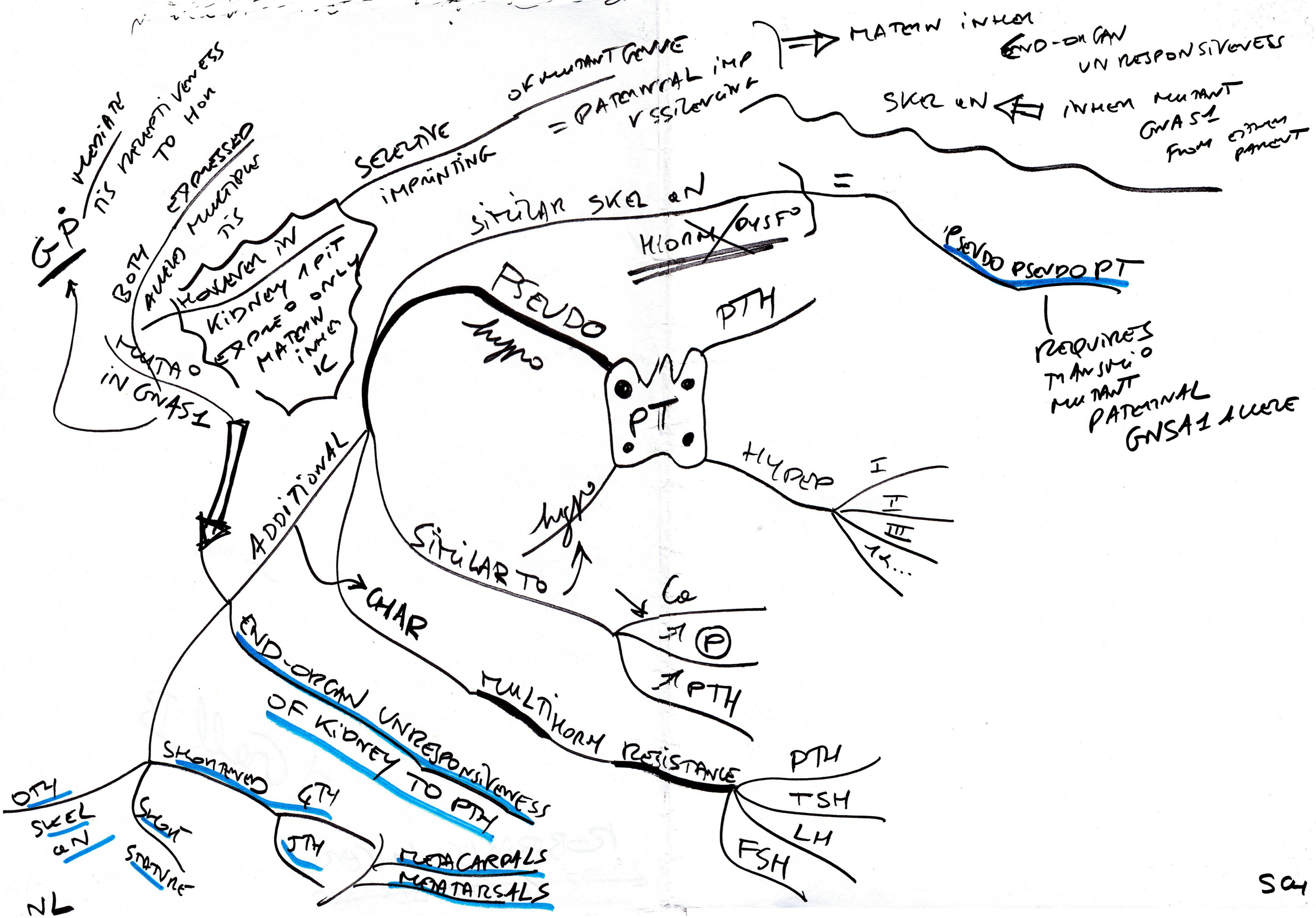

HASHIMOTO THYROIDITIS
AUTOIMMUNE
♀>♂
CLI
OFTEN INAPPARENT
SLOW COURSE
THYROID
NON TENDER
MODERATELY ENLARGED
MAY OCCASIONALY HAVE AN EARLY TRANSIENT HYPER PHASE
A MOST COMMON
MOST OFTEN EUTHYROID AT FIRST
BUT TRANSIENT HYPER MAY OCCUR
DEVELOPS LATE
KIDNEY GLANDS SHRUNKEN & SCARRED
HISTO
MASSIVE INFILTRATES + GERMINAL CENTER FORMAT°
LC
FOLLICLES ATROPHIC
HÜRTHLE C
= EPIT C WITH EOSINOPHILIC GRANULAR CYTOP
PROMINENT
MAY ALSO ASSOCIATED
↑ INCIDENCE OTH AUTOIMM
PERNICIOUS ANEMIA
DM
SJÖGREN SD
⊕ VE HLA DR5
HLA B5
VARIOUS AUTO ≥ 1
MOST PROMIN ANTI
THYROGLOBULIN – T
PEROXIDASE – TSHR
– IODINE R
♀>♂
SUBACUTE (DE QUERVAIN, GRANULOMATOUS)
FOC DESTRUCT THYR TIS
GRANULOMATOUS IFM
CHAR
MAY INCUDE
VIRAL IFC MUMPS
COXSACKIEVIRUS
HYPER
SOMET TRANSIENT
SELF LIMITED COURSE
SEVERAL WEEKS
FLU LIKE ILLNESS + PAIN TENDERNESS THYR
RIEDEL
ORIGIN ?
REPLACEMENT BY FIBROUS TISSUE
MIMIC CARCINOMA
CAN CLINICALLY

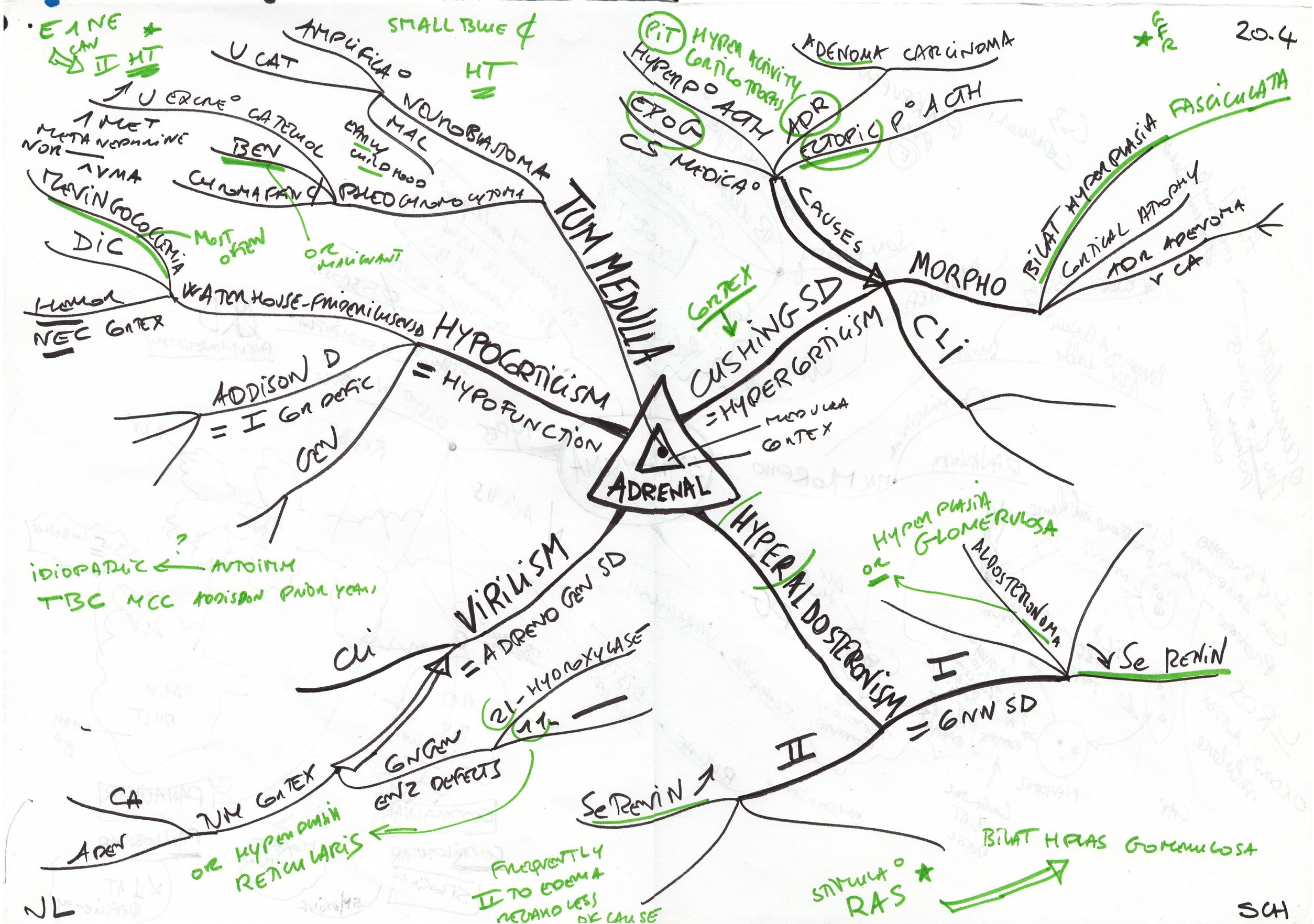
NL

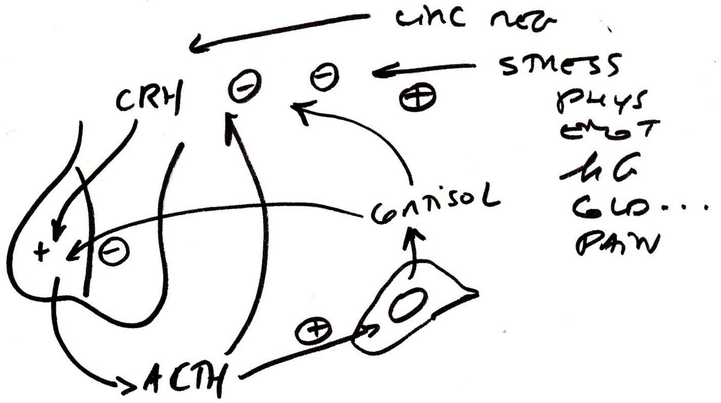
SCM


RET
MUTA°
FAM
FOLLICULAR ADENOMAS
IN ALL OTH ASPECTS INDISTINGUISHABLE FROM
SPORADIC
MEN II
TYPE III
20% CASES
ret-PTC
ADDITIONAL
NOVEL FUSION GENES
RET
ANALOGOUS
bcr-abl
FUSION IN CHRONIC MYELOID LEUKEMIA
ALSO ⇒ ALSO CONSTITUTIVE
TYROSINE KINASE DOMAIN
TYROSINE KINASE DOMAIN OF
ACTIVATE
BENIGN ADENOMAS
MOST OFTEN SOLITARY
NODULES
CAN VARIETY HISTO PATTERNS
OCCUR
CLI
HÜRTHLE
FOLLICULAR
MOST OFTEN
NON FUNCTIONAL
CAN OCCASIONNALLY
CAUSE HYPER
OLDER
VERY POOR PROGNOSIS
MALIGNANT TUMORS
CARCINOMAS
PAPILLARY
MOST COMMON THYR CANCER
HISTO
PAPILLARY PROJECO
INTO GLANDULAR SPACES
TUM &
"GROUND-GLASS"
"ORPHAN ANNIE" NUCLEI
CALLIFIED SPHERES
=PSAMMOMA BODIES
MAY BE PRESENT
EVEN WHEN ADJACENT LN ARE PRESENT
BETTER PROGNOSIS
THAN OTHER FORMS
SOME INSTANCES
CAN BE
CHANGES K10
CHROMOSOME
RECIPROCAL TRANSLOCO
BETWEEN 10/17
PARACENTRIC INVERSION
MUTOIN
CAN
LT
TYPE
AS KINASE
BRAF
CONSTITUTIVELY
ALSO GROW
NECK
PROGNOSIS PRODUCTIVE
BEHAVIOR
PRIOR RADIOTHERAPY
RELATIVELY UNIFORM
POORER
PROGNOSIS THAN
FOLLICULAR
FOLLICLES
IDENTIFICA°
DIAGNOSIS
CAPSULAR
LYMPHOVASC INVASION
BECAUSE
IN ALL OTH ASPECTS INDISTINGUISHABLE FROM
SHEETS NIDUS & ALTHOUGH MAJORITY
+ AMYLOID STROMA
CASES
Ca 2+
HORMONE
PRODUCES
HISTO
MEDULLARY
ANAPLASTIC
NL
SCM

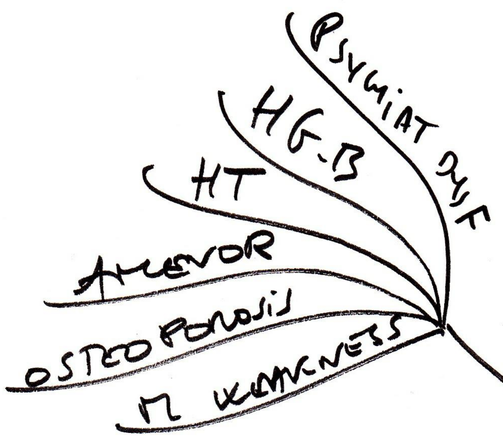

PARA-THYROID
PTH
SECRET°
* NOT UNDER PITUITARY CONTROL
CALCITRIOL
1,25 mmol/L
CT
Ca 2+
GLANDS RESPONSIVE
[Ca 2+]
PRODUC°
HYPER
PT ADENOMA
FEW PT HYPERPLASIA
MO
CA RARE
BRONCHOGENIC
SCC
RENAL
C CA
LESS OFTEN CAN OCCUR
PO PTH LIVE
HBP MAL TUM
ASPART MEN I
LAB
HCa-B ↑ HCa-U
↓Se P
↓TUB REABS°
P ↑
↑U-P
↑Se ALKALINE PHOSPHATASE
↑Se PTH
CLI
OSTEITIS FIBROSA CYSTICA
PEP DUD ULC°
RENAL COMP
CALCULI
MANY TIS.
METASTATIC CALCIFICATION
OC REJORP°
= VON RECKLINGHAUSEN D
BROWN TUM
FIBROUS REPLACEMENT
NEOP TUM LIVER
MASSES SCH
MAY
PSEUDO
hypo
hypo
hypo
SECRET°
* NOT UNDER PITUITARY CONTROL
↑NM EXCITABILITY
↑TETANY
Li
SEVERE h Ca
CONGENITAL THYMIC HYPOPLASIA
RARE
ACQUIRED ERR° DURING THYROID
MC
↑PTH SECR° di GOLGI
↓ABS° Ca
IMPAIRED HYDROXYLAT° 25-...
↓α-HYDROXYLASE DEFIC
VIT D NUTRIT DEP
DUPT ADENOMA OFTEN
IN PREVIOUSLY HYPERPLASTIC
III
PERSISTENT
IN SPITE CORRECT° h-Ca-B
- ↑ PREEXISTING
II
PTH ↑
HET Ca
DIFFUSE OSTEO CHANGES BONES
↑Se Alk PHOSPHAT
↑Se P
↓Se Ca
CHAR
↑Se P RECIPROC D IN Se Ca
↓INT ABS° Ca
NL
GOVERNS°
PTD → 1,25-OH2 D3
MCC
COMPENSATORY PT HYPERPLASIA
h Ca CHRONIC REN D
USUALLY
[Ca 2+]
H Ca-B
H
H

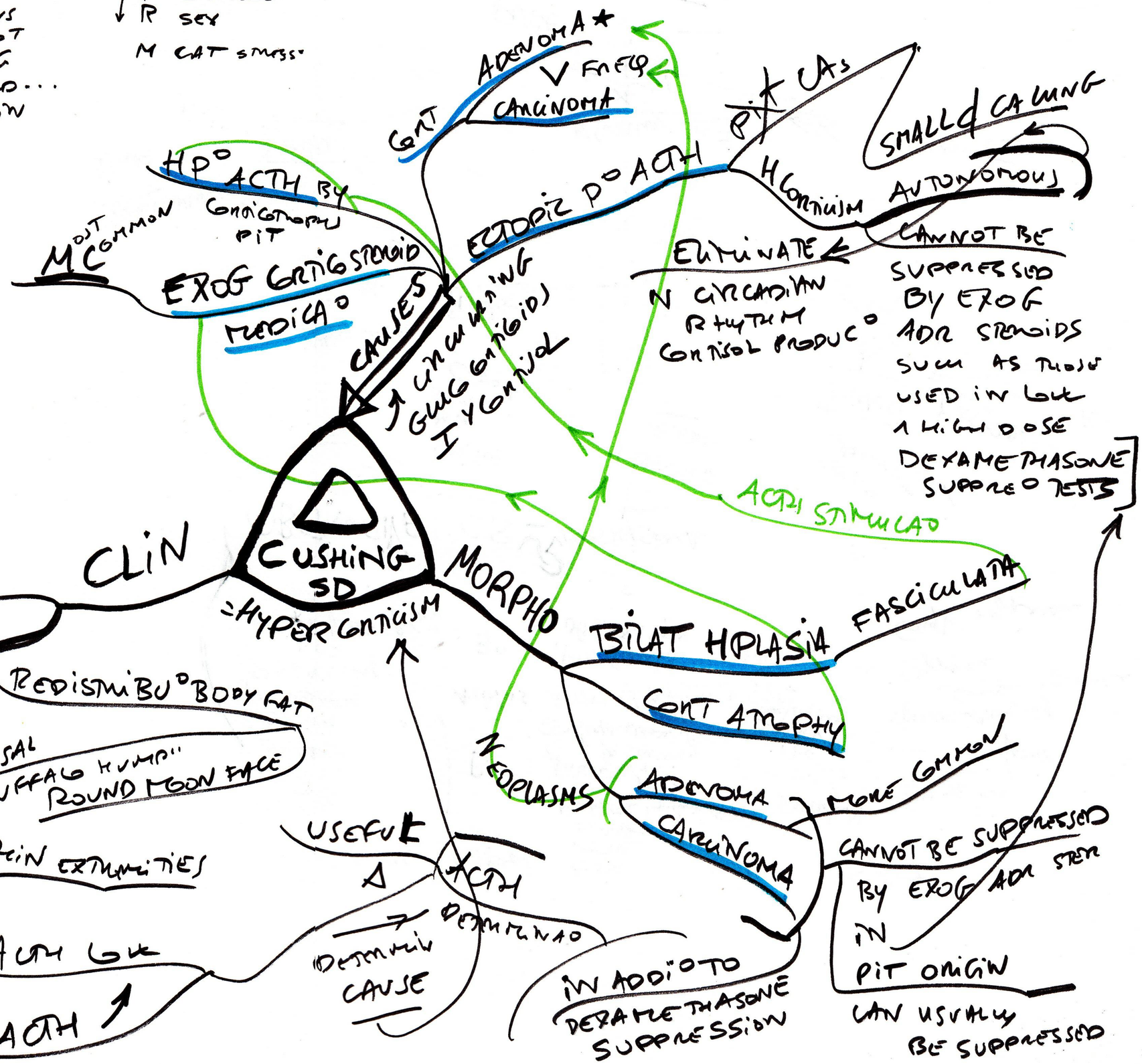

GP
MEDIATE
TIS INSENSITIVENESS TO HORM
BOTH EXPRESSED
ALLELES MUTATIONS
TIS
HOWEVER IN KIDNEY 1 PIT EXPRESS ONLY MATERNAL INHER IC
IMPRINTING
IN GNAS1
SELECTIVE
OF MUTANT GENE
= PATERNAL IMP VS SILENCING
MATERN INHER
END-ORGAN UNRESPONSIVENESS
SKEL QN
INHER MUTANT GNAS1 FROM OTHER PARENT
SIMILAR SKEL QN
=
HORM/DYSF
PSEUDO PSEUDO PT
REQUIRES TRANSMISS MUTANT PATERNAL GNSA1 ALLELE
PSEUDO
hypo
PTH
PT
HYPERP
I
II
III
IV...
hypo
SIMILAR TO
Ca
P
PTH
CHAR
END-ORGAN UNRESPONSIVENESS OF KIDNEY TO PTH
MULTIHORM RESISTANCE
PTH
TSH
LH
FSH
SHORTENED
4TH
JTH
METACARPALS
METATARSALS
OTH
SKEL QN
SHORT
STATURE
NL
SCH


20.4
ADRENAL
TUM MEDULLA
NEUROBLASTOMA
AMPULLA°
SMALL BLUE ¢
HT
U CAT
↑ U ERCNE° CATECHOL
↑ MET
METANEPHRINE
NOR
↑VMA
BEN
CHROMAFFIN ¢
PHEOCHROMOCYTOMA
MAL
FAMILY
CHILDHOOD
OR MALIGNANT
MENINGOCOCCEMIA
MOST OFTEN
DIC
WATERHOUSE-FRIDERICHSEN
HEMOR
NEC CORTEX
HYPOCORTICISM
= HYPOFUNCTION
ADDISON D
= I CORTIC
GEN
IDIOPATHIC ← AUTOIMM
TBC MCC ADDISON PRIOR YEARS
CUSHING SD
= HYPERCORTICISM
CORTEX
MEDULLA
CORTEX
CAUSES
PIT HYPER ACTIVITY CORTICOTROPHS
HYPERP° ACTH
ADR
EXOG
CS MEDICA°
ECTOPIC P° ACTH
ADENOMA CARCINOMA
MORPHO
BILAT HYPERPLASIA FASCICULATA
CORTICAL ATROPHY
ADR ADENOMA
V CA
CLI
HYPERALDOSTERONISM
I
= GNN SD
↓ Se RENIN
HYPER PLASIA GLOMERULOSA
OR
ALDOSTERONOMA
II
Se RENIN ↑
FREQUENTLY
II TO EDEMA
REGARDLESS
OF CAUSE
STIMULA°
RAS ★
BILAT HYPERPLAS GLOMERULOSA
VIRILISM
= ADRENO GEN SD
CLI
21-HYDROXYLASE
11-
GNGEN
ENZ DEFECTS
NM CORTEX
CA
ADEN
OR HYPERPLASIA
RETICULARIS
NL
SCH



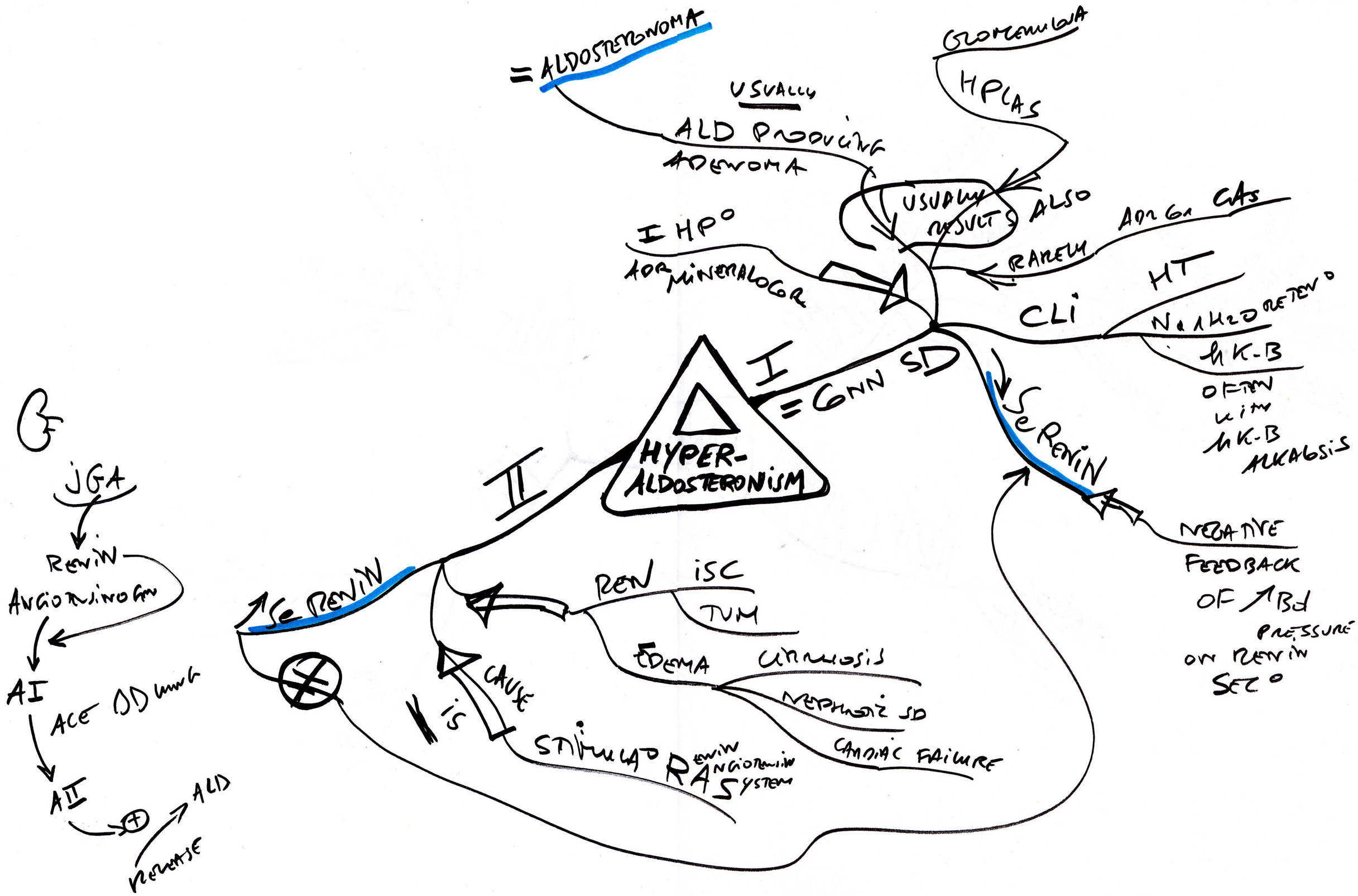
G aldosteron
F cortisol
R sex
M cat stress

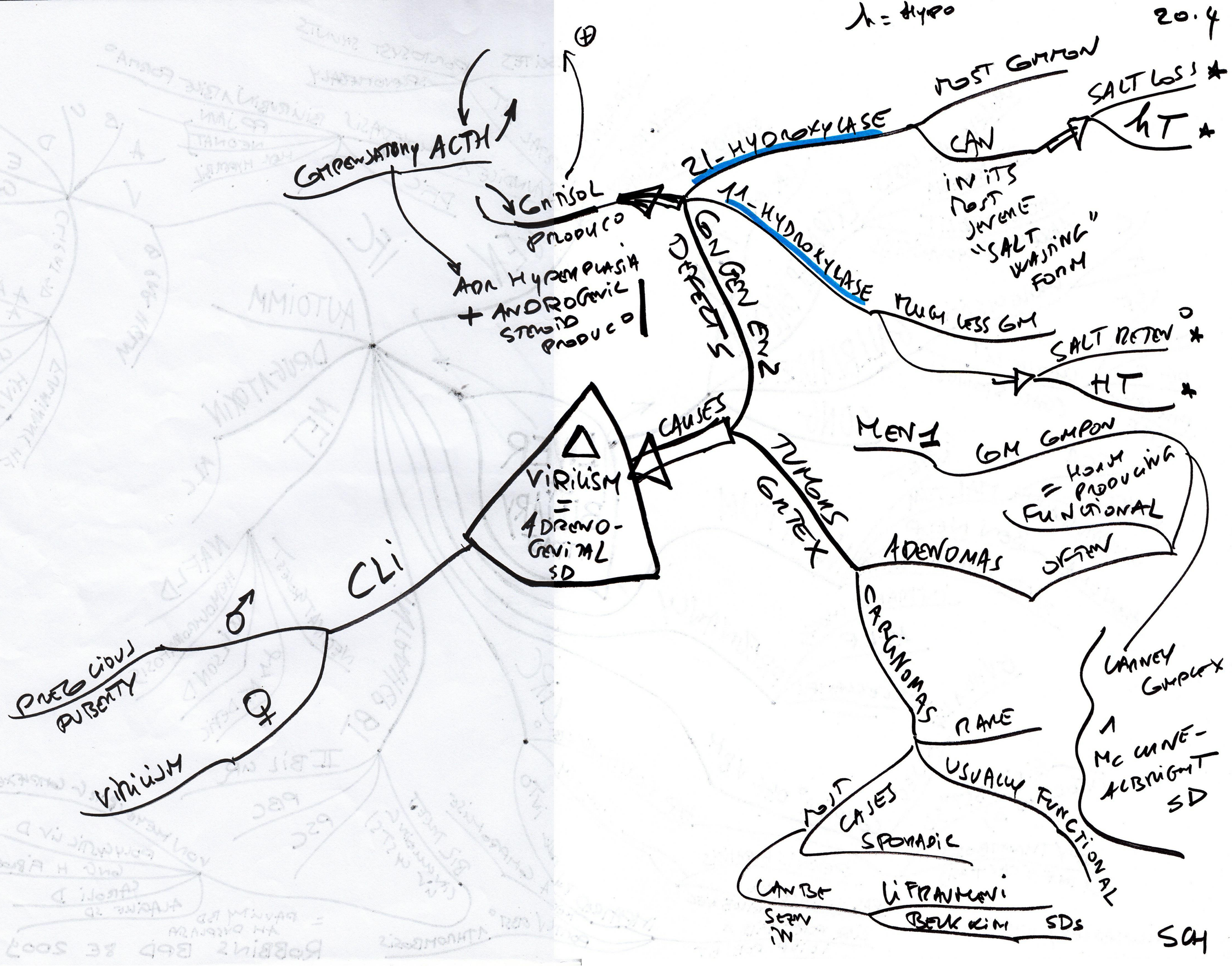




NL

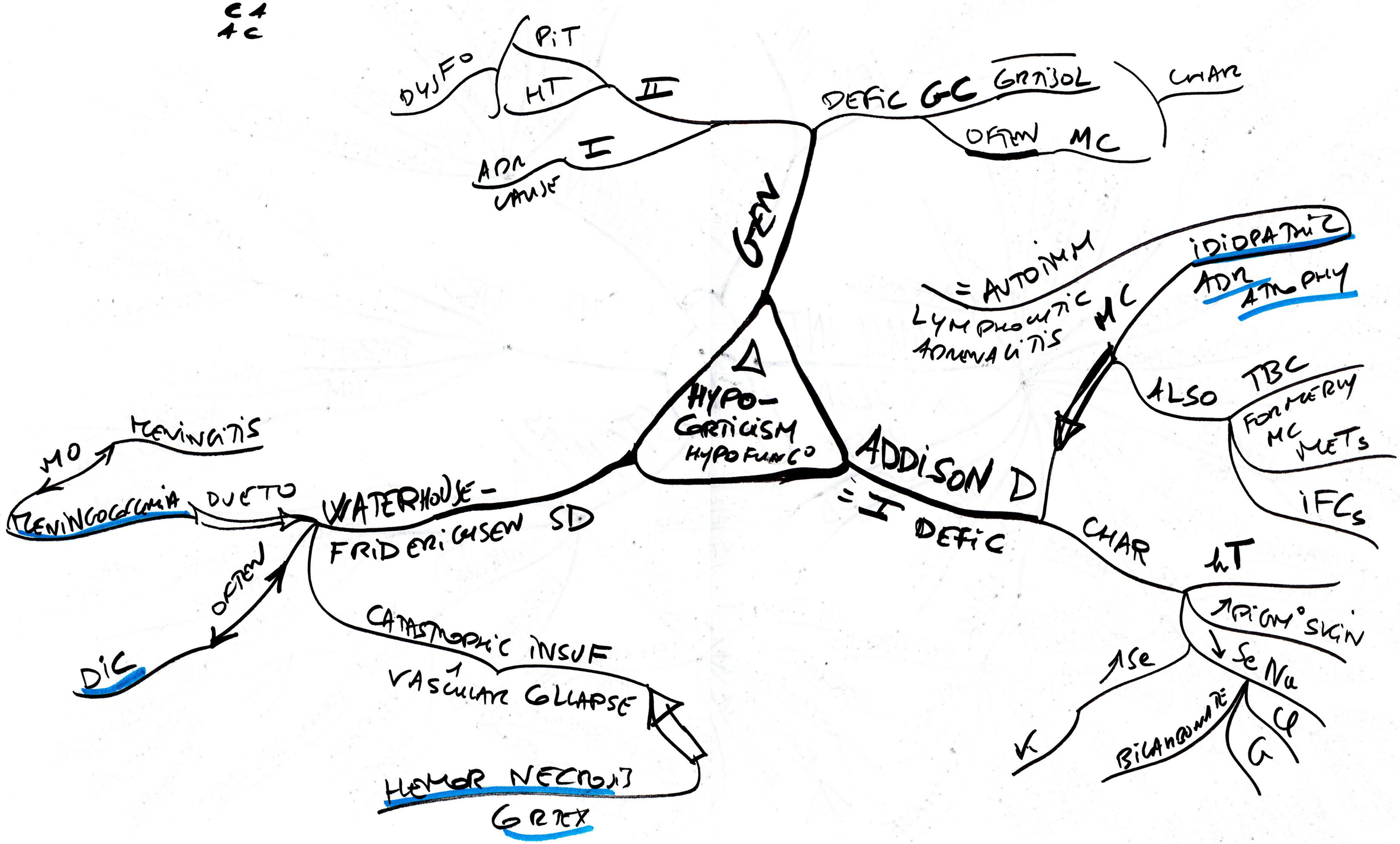
SCH


= ALDOSTERONOMA
USUALLY
ALD PRODUCING
ADENOMA
GLOMERULOSA
HPLAS
USUALLY RESULTS ALSO
RARELY
ADRGL CAS
I HPO
ADR MINERALOCOR
HT
CLI
NaH2O RETENTO
hK-B
OFTEN with hK-B ALKALOSIS
HYPER-ALDOSTERONISM
I
= GNN SD
II
SeRENIN
NEGATIVE FEEDBACK OF ↑Bd PRESSURE ON RENIN SEC°
SeRENIN
REN
iSC
TUM
EDEMA
CIRRHOSIS
NEPHROTIC SD
CARDIAC FAILURE
CAUSE
is
STIMULATO RAS
RENIN ANGIOTENSIN SYSTEM
JGA
RENIN
ANGIOTENSINOGEN
AI
ACE
AII
ALD
RENATSE
NL
SCH


h = Hypo
20.4
COMPENSATORY ACTH
⊕
CORTISOL PRODUCED
ADR HYPERPLASIA + ANDROGENIC STEROID PRODUCED
21-HYDROXYLASE
11-HYDROXYLASE
MOST COMMON
CAN
SALT LOSS *
HT *
IN ITS MOST SEVERE "SALT WASTING" FORM
MUCH LESS COM
SALT RETEN *
HT *
DIRECTLY GENERATE
CAUSES
VIRILISM = ADRENO-GENITAL SD
TUMORS CORTEX
MEN 1
COM COMPON
= HORM PRODUCING
FUNCTIONAL
ADENOMAS
OFTEN
CARCINOMAS
RARE
USUALLY FUNCTIONAL
CARNEY COMPLEX
1
MC CUNE-ALBRIGHT SD
MOST CASES SPORADIC
CAN BE SEEN IN
LI FRAUMENI
BECKWITH SDs
CLI
♂
PRECOCIOUS PUBERTY
♀
VIRILISM
NL
SCH

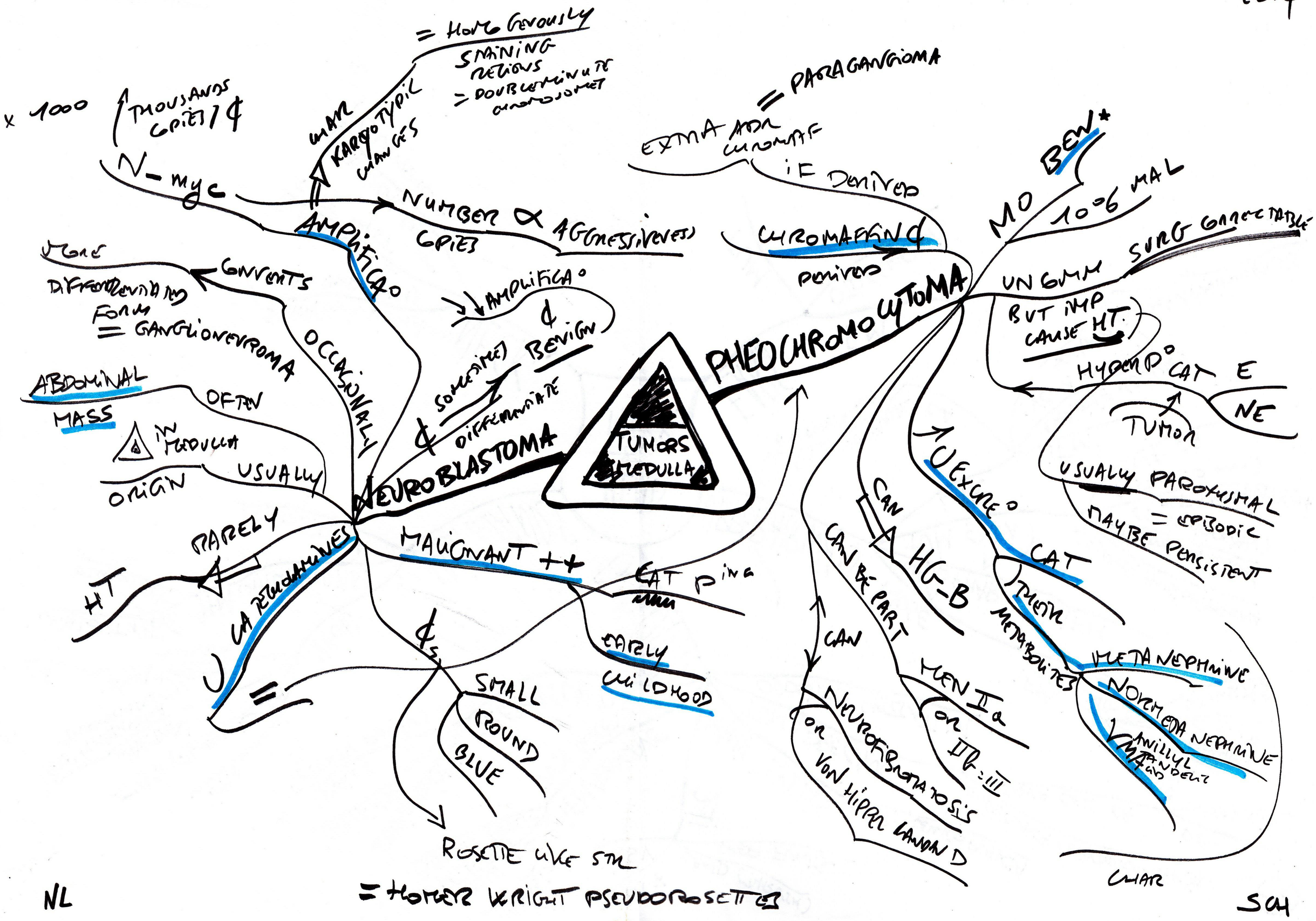
CUSHING — CORTISOL
GNN — ALDOSTERONE — HYPER
ADDISON — CORTISOL — HYPO

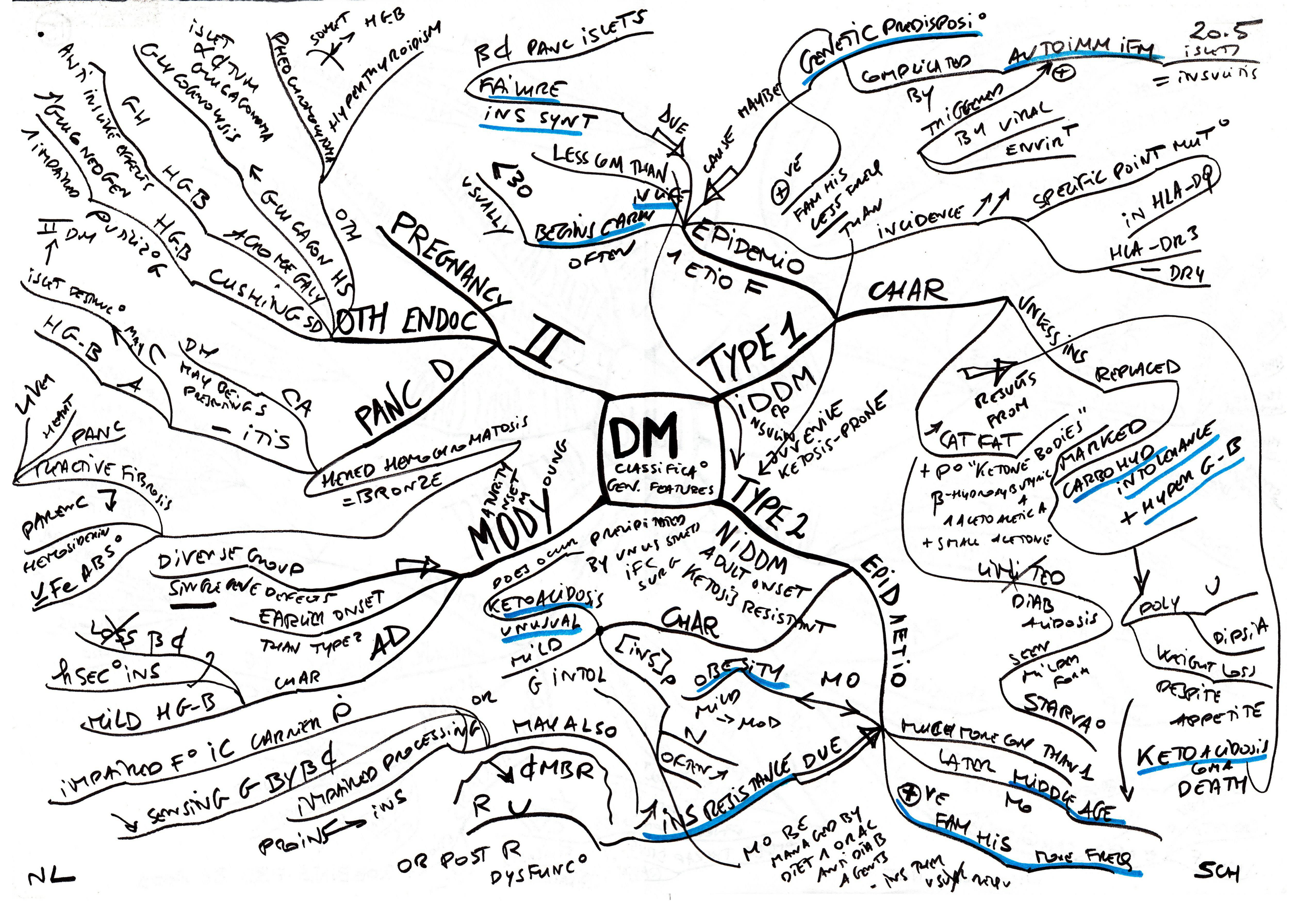
CC
C4
AC

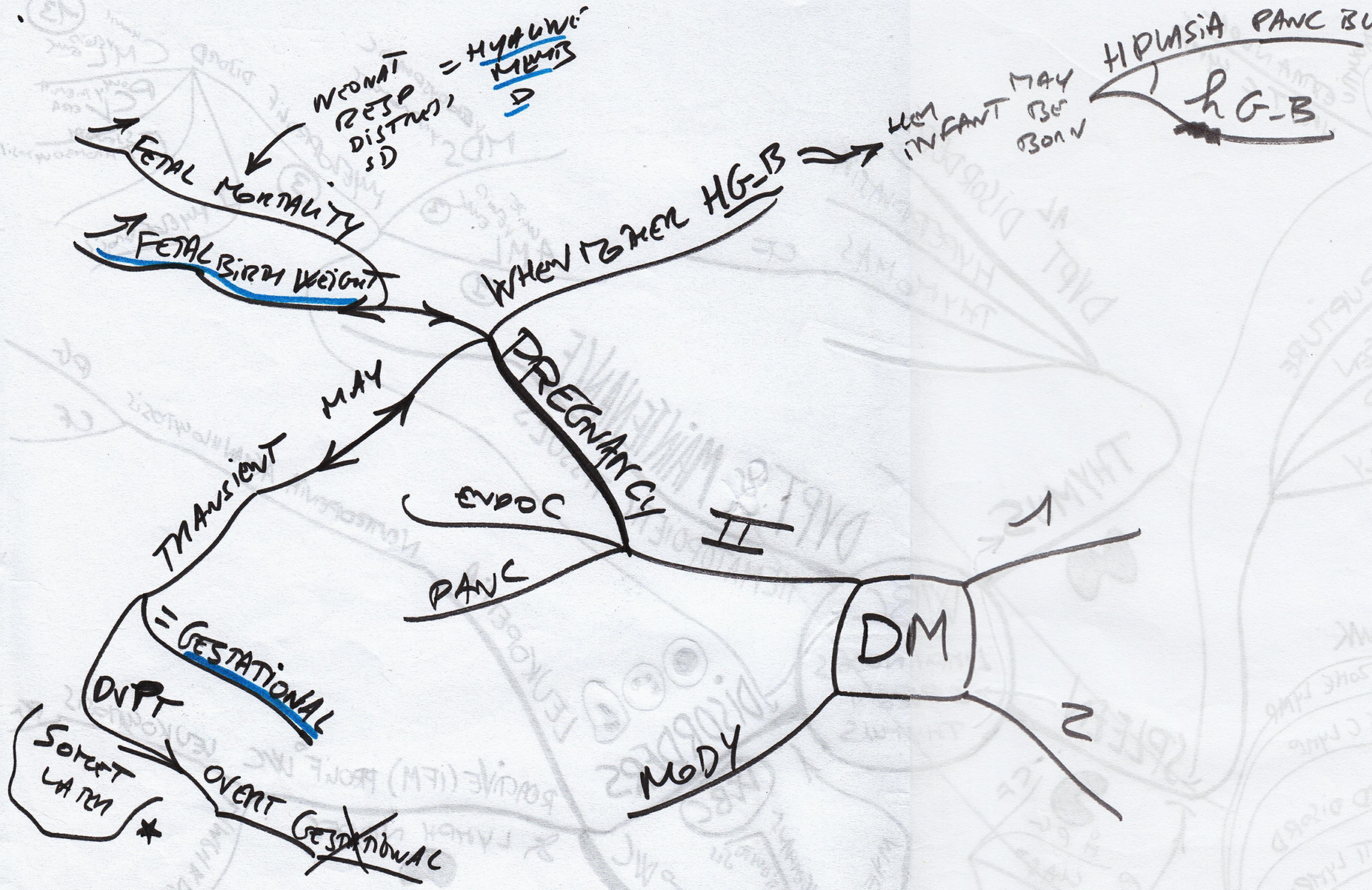


NL

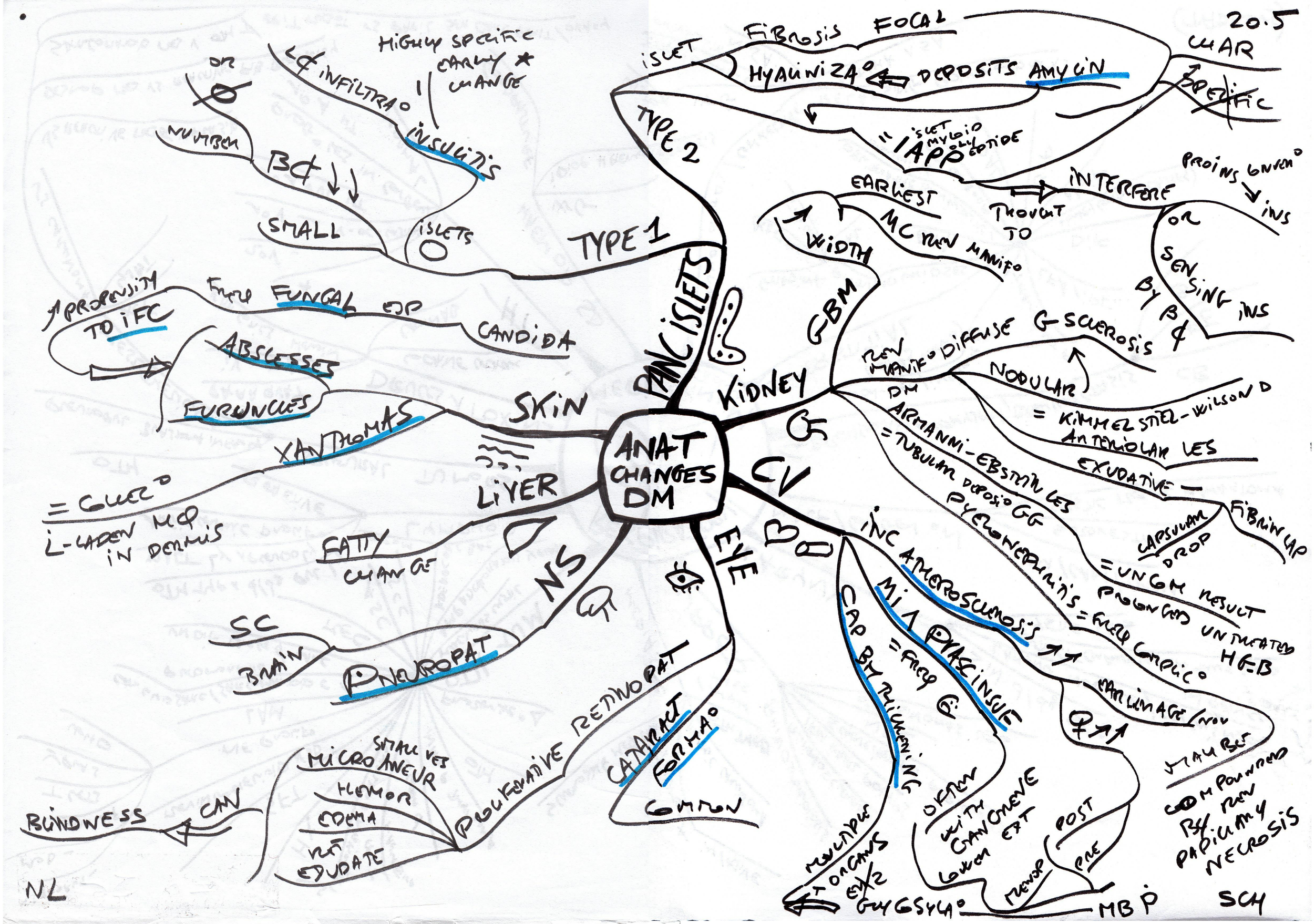
SCM

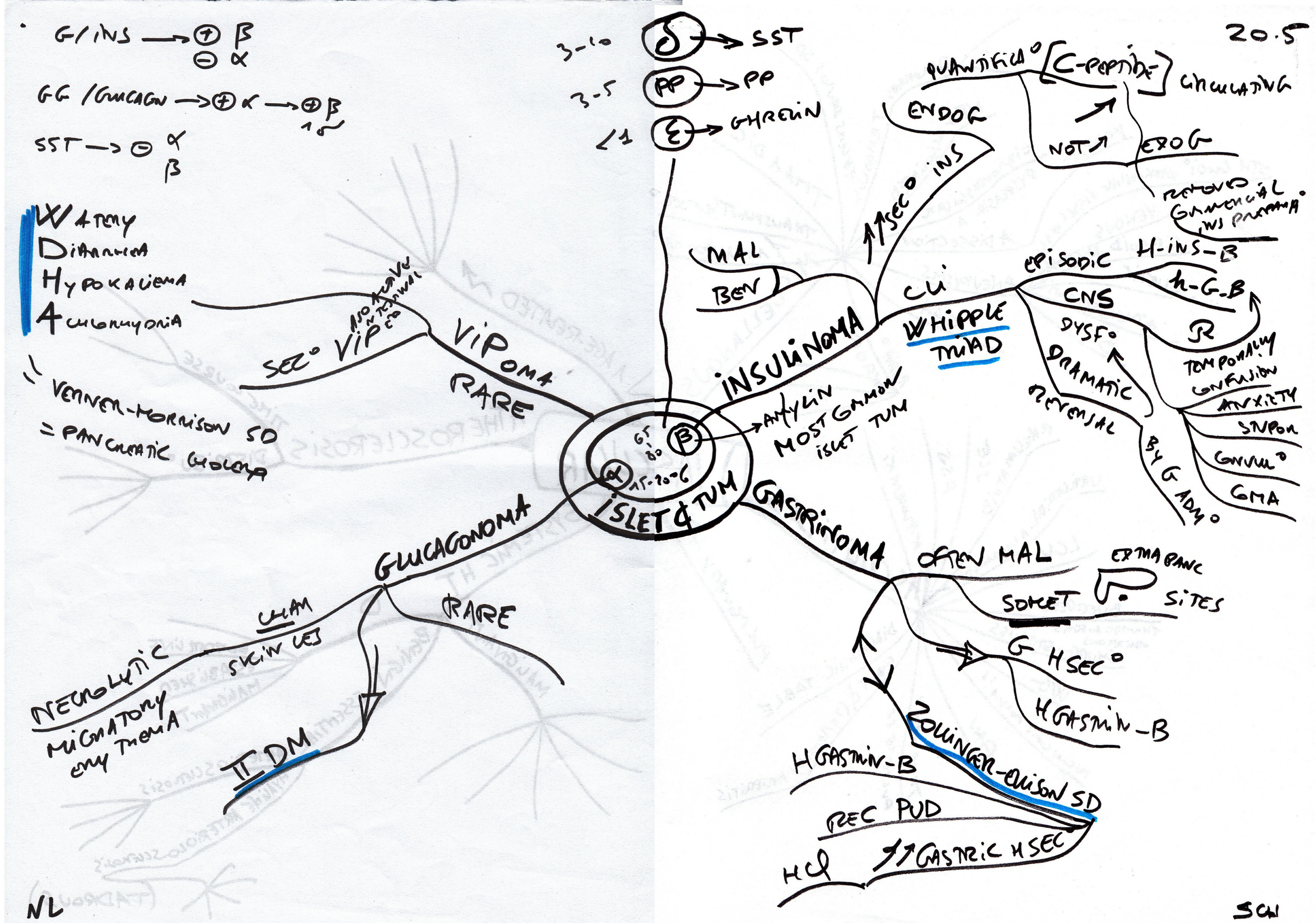

x 1000
THOUSANDS COPIES/¢
N-myc
MAR
KARYOTYPIC CHANGES
= HOMOGENOUSLY STAINING REGIONS
= DOUBLE MINUTE CHROMOSOMES
AMPLIFICATION
NUMBER ∝ AGGRESSIVENESS
COPIES
EXTRA ADR CHROMAFF
= PARAGANGLIOMA
IF DERIVED
CHROMAFFIN ¢
DERIVED
MO BEN*
10^6 MAL
SURG CONNECTABLE
UNCOMM
BUT IMP CAUSE HT.
HYPERD° CATE
NE
TUMOR
USUALLY PAROXYSMAL
= EPISODIC
MAYBE PERSISTENT
PHEOCHROMOCYTOMA
CAT
METAN METABOLITES
METANEPHRINE
NORMETANEPHRINE
VANILLYL MANDELIC
CHAR
EXCRE°
CAN
HG-B
CAN BE PART
MEN IIa
or IIb = III
CAN
NEUROFIBROMATOSIS
or
VON HIPPEL LANDAU
CONVERTS
DIFFERENTIATED FORM
= GANGLIONEUROMA
OCCASIONALLY
AMPLIFICA°
¢ BENIGN
SOMETIMES
¢ DIFFERENTIATE
NEUROBLASTOMA
TUMORS MEDULLA
ABDOMINAL MASS
OFTEN
IN MEDULLA
ORIGIN
USUALLY
RARELY
HT
CATECHOLAMINES
MALIGNANT ++
CAT P^ina
EARLY CHILDHOOD
¢s
SMALL
ROUND
BLUE
ROSETTE LIKE STR
= HOMER WRIGHT PSEUDOROSETTES
NL
SCH

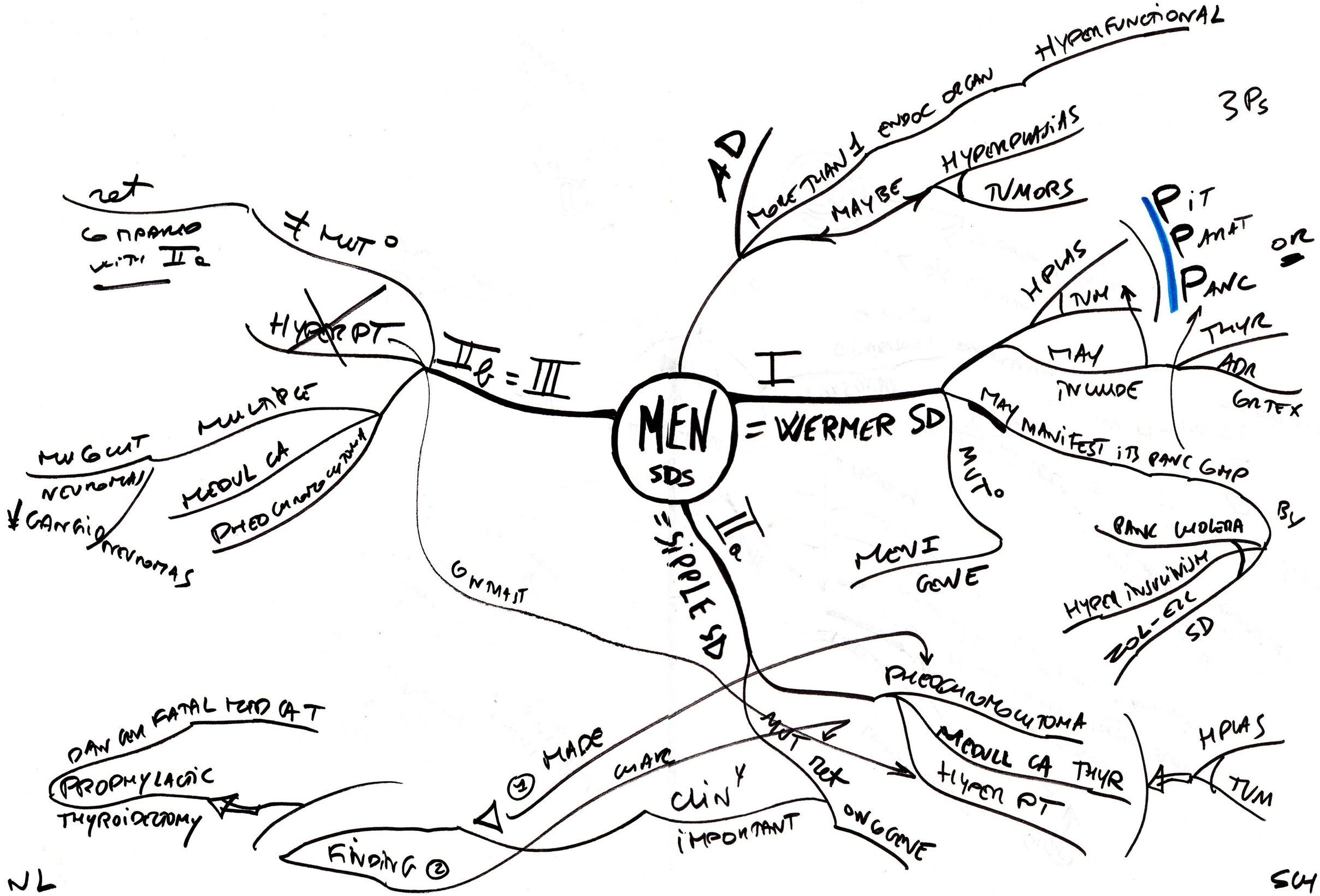

DM
CLASSIFICA° GEN. FEATURES
TYPE 1
iDDM
EP
INSULIN
JUVENILE KETOSIS-PRONE
EPIDEMIO
ETIO F
GENETIC PREDISPOSI°
COMPLICATED BY
AUTOIMM IFM
20.5
ISLETS
= INSULITIS
TRIGGERED BY VIRAL ENVIRN
CAUSE MAYBE
DUE
BCD PANC ISLETS
FAILURE
INS SYNT
LESS GM THAN
INVOLVE
BEGINS CHRON
OFTEN
USUALLY
<30
VE
FAM HIS LESS FREQ THAN
INCIDENCE ↑↑
SPECIFIC POINT MUT°
IN HLA-DQ
HLA-DR3
— DR4
CHAR
UNLESS INS REPLACED
RESULTS FROM
CAT FAT
+ PO "KETONE BODIES"
β-HYDROXYBUTYRIC
+
ACETOACETIC A
+ SMALL ACETONE
MARKED
CARBOHYD INTOLERANCE
+ HYPER G-B
UNTREATED DIAB
KETOACIDOSIS
SEEN MILDER FORM
STARVA°
POLY U
DIPSIA
WEIGHT LOSS
DESPITE APPETITE
KETOACIDOSIS
COMA
DEATH
TYPE 2
NIDDM
ADULT ONSET
INSULIN RESISTANT
EPID
ETIO
MO
MUCH MORE COM THAN 1
LATER MIDDLE AGE
VE
FAM HIS MORE FREQ
MO BE MANAGED BY DIET ORAL ANTI DIAB AGENTS
- INS THEN USUAL THERA
INS RESISTANCE DUE
OBESITY
MO
MILD → MOD
N
OFTEN
[INS]P
CHAR
DOES OCCUR PRECIPITATED BY VIRUS STRESS
IFC
SURG
KETOSIS RESISTANT
KETOACIDOSIS
UNUSUAL
MILD
G INTOL
MAY ALSO
OR
CMBR
R U
OR POST R DYSFUNC°
PROINS → INS
IMPAIRED PROCESSING
IMPAIRED F° IC CARRIER P
SENSING G BY BCD
MILD HG-B
LOSS BCD
hSEC° INS
EARLIER ONSET THAN TYPE 2
AD
CHAR
MODY
MATURITY ONSET YOUNG
DIVERSE GROUP
SINGLE GENE DEFECTS
ΣFe ABS°
HEMOSIDERIN
PANC ↓
REACTIVE FIBROSIS
PANC
LIVER
HG-B
A
C
MAY BE PRESENTING
DM
CA
— ITIS
HEMED HEMOCHROMATOSIS
= BRONZE
ISLET DESTRUC°
IMPAIRED
PUBERTY
II DM
IMPAIRED
GLUCAGON
HYPER
CUSHING SD
ACROMEGALY
GLUCAGON HS
OTH ENDOC
ISLET & DUCT GLUCAGONOMA
GLUCAGONOSIS
PHEOCHROMOCYTOMA
SOMAT → HGB
HYPERTHYROIDISM
OTH
GH
HGB
ANTI INSULIN EFFECTS
GLUCAGON
IMPAIRED
PANC D
PREGNANCY
II
NL
SCH


NEONAT RESP DISTRESS SD = HYALINE MEMB D
FETAL MORTALITY
FETAL BIRTH WEIGHT
WHEN TO ASSER HG-B
NEW INFANT MAY BE BORN
HYPOPLASIA PANC BUDD
h G-B
PREGNANCY
MAY
TRANSIENT
= GESTATIONAL
DMPT
SOMEST LATER *
OVERT GESTATIONAL
ENDOC
PANC
II
1
DM
2
MODY

SCH


ANAT CHANGES DM
PANC ISLETS
TYPE 1
TYPE 2
ISLET
FIBROSIS FOCAL
HYALINIZA°
DEPOSITS AMYLIN
= ISLET AMYLOID POLYPEPTIDE
IAPP
CHAR
SPECIFIC
INTERFERE
THOUGHT TO
PROINS CONVER° INS
OR
SENSING INS
BY BC
EARLIEST
MC NEW MANIF°
WIDTH
GBM
KIDNEY
NEW MANIF° DIFFUSE
G SCLEROSIS
NODULAR
= KIMMELSTIEL-WILSON D
ARMANNI-EBSTEIN LES
= TUBULAR DEPOSIT° GG
EXUDATIVE
FIBRIN CAP
CAPSULAR DROP
= UNCOM RESULT PROLONGED UNTREATED HGB
PYELONEPHRITIS = FREQ COMPLIC°
INC ATHEROSCLEROSIS
MI
PYAE INSULF
= FREQ GI
CAP BY THICKENING
MULTIPLE ORGANS
EYE
GLYCSYLA°
OFTEN WITH GANGRENE LOWER EXT
MENOP
POST
PNE
MBP
SCH
COMPOUNDED BY NEW PAPILLARY NECROSIS
EXAM LIMAGE NOV
VITAL BLF
EYE
PROLIFERATIVE RETINOPAT
CATARACT FORMA°
COMMON
MICROANEUR
SMALL VES
HEMOR
EDEMA
NEW EXUDATE
BLINDNESS
CAN
NS
PNEUROPAT
BRAIN
SC
LIVER
FATTY CHANGE
SKIN
XANTHOMAS
= GLUC°
L-LADEN MΦ IN DERMIS
FUNGAL EOP
CANDIDA
ABSCESSES
FURUNCLES
PROPENSITY TO IFC
NL
CV
GI
GU
BC
INFILTRA°
HIGHLY SPECIFIC EARLY CHANGE
INSULITIS
NUMBER
BC
SMALL
ISLETS
OR


G/INS → ⊕ β
⊖ α
GG / GLUCAGN → ⊕ α → ⊕ β
1°
SST → ⊖ α
β
3-10 δ → SST
3-5 PP → PP
<1 ε → GHRELIN
ISLET TUM
β
α
INSULINOMA
ANTYLIN
MOST GAMMON ISLET TUM
MAL
BEN
↑SEC° INS
QUANTIFICA° [C-PEPTIDE]
CIRCULATING
ENDOG
NOT ↗
EXOG
REMOVES GAMMOPATHY INS PROGRAMA
CLI
WHIPPLE TRIAD
EPISODIC
H-INS-B
CNS
h-G-B
DYSF°
SR
DRAMATIC
REVERSAL
TEMPORALLY CONFUSION
ANXIETY
STUPOR
GRAVIL°
GMA
BY G ADM°
VIPOMA
RARE
SEC° VIP
ALSO ACTIVE INTESTINAL
WATERY DIARRHEA
HYPOKALIEMA
ACHLORHYDRIA
= VERNER-MORRISON SD
= PANCREATIC CHOLERA
GLUCAGONOMA
RARE
CLIN
SKIN LES
NECROLYTIC MIGRATORY ERYTHEMA
II DM
GASTRINOMA
OFTEN MAL
EXTRAPANC
SOMET
SITES
G
H SEC°
H GASTRIN-B
ZOLLINGER-ELLISON SD
H GASTRIN-B
REC PUD
HCl
↑↑ GASTRIC H SEC
NL
SCH


MEN
SDS
I
= WERMER SD
AD
MORE THAN 1 ENDOC ORGAN
HYPERFUNCTIONAL
MAYBE
HYPERPLASIAS
TUMORS
3 Ps
PIT
PARAT
PANC
OR
HPLAS
TUM
MAY
INCLUDE
THYR
ADR
CORTEX
MAY MANIFEST ITS PANC GMP
MUT.
BL
PANC CHOLERA
HYPERINSULINISM
NON-ENC
SD
MENI
GENE
PHEOCHROMOCYTOMA
MEDULL CA THYR
HYPER PT
HPLAS
TUM
MUT
NEX
ONCOGENE
CLIN
IMPORTANT
MADE
CARE
FINDING
PROPHYLACTIC
THYROIDECTOMY
DAN CAN FATAL MED CAT
II = III
HYPER PT
≠ MUT
ret
COMPARED WITH IIa
IIa
= SIPPLE SD
GNMAST
MULTIPLE
MUCOCUT
NEUROMAS
MEDUL CA
PHEOCHROMOCYTOMA
GANGIO
NEUROMAS
NL
SCH