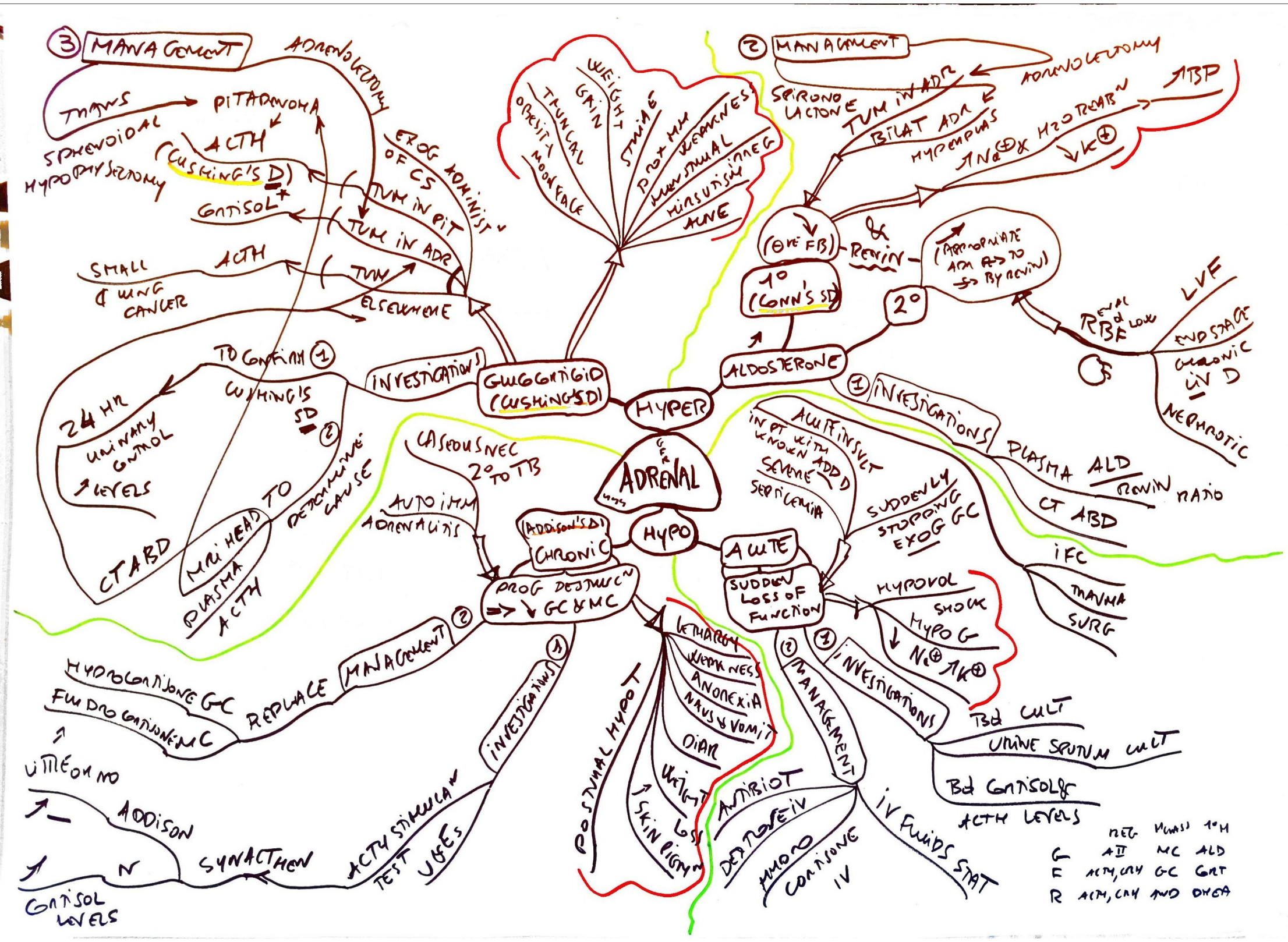
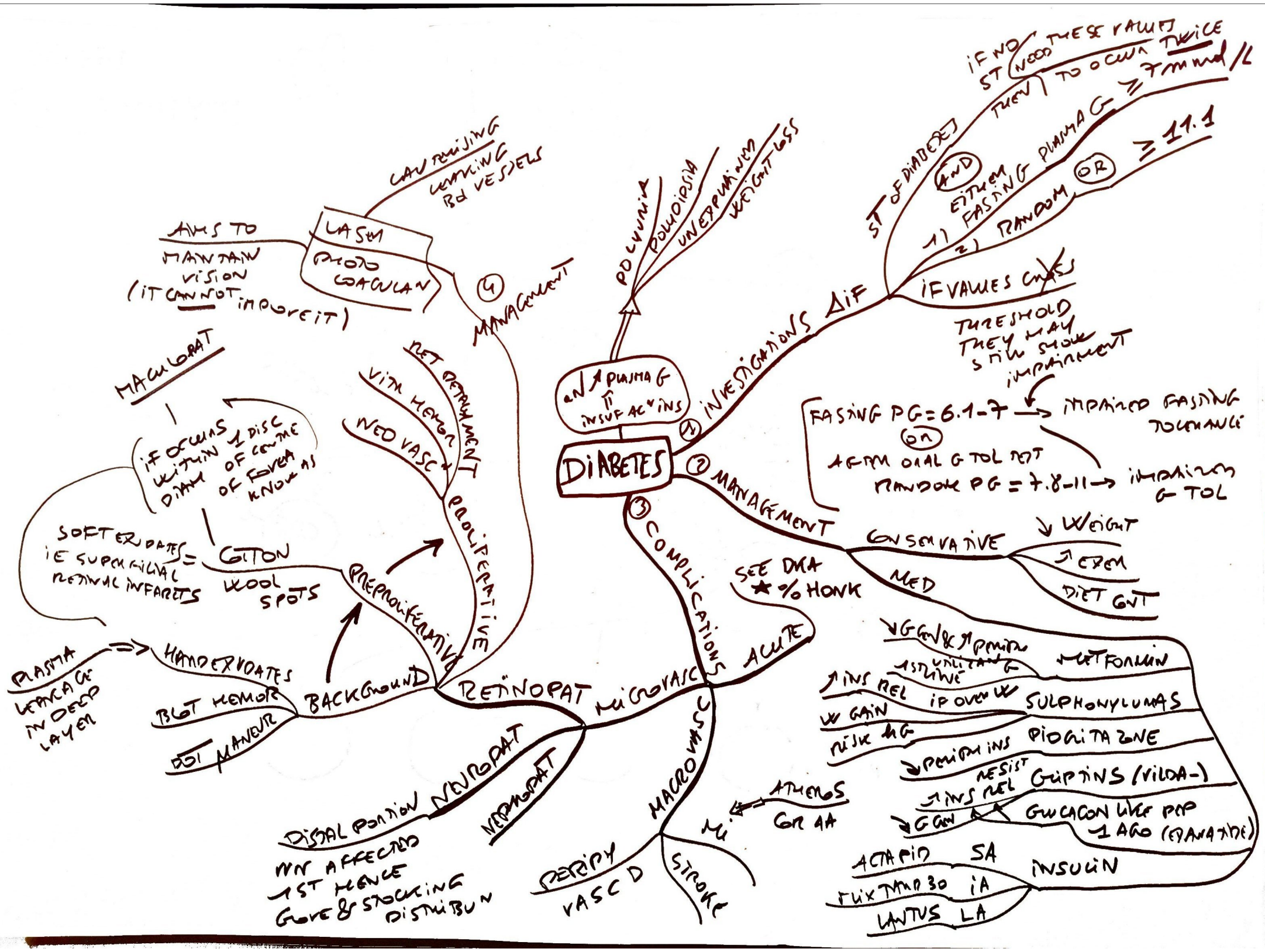
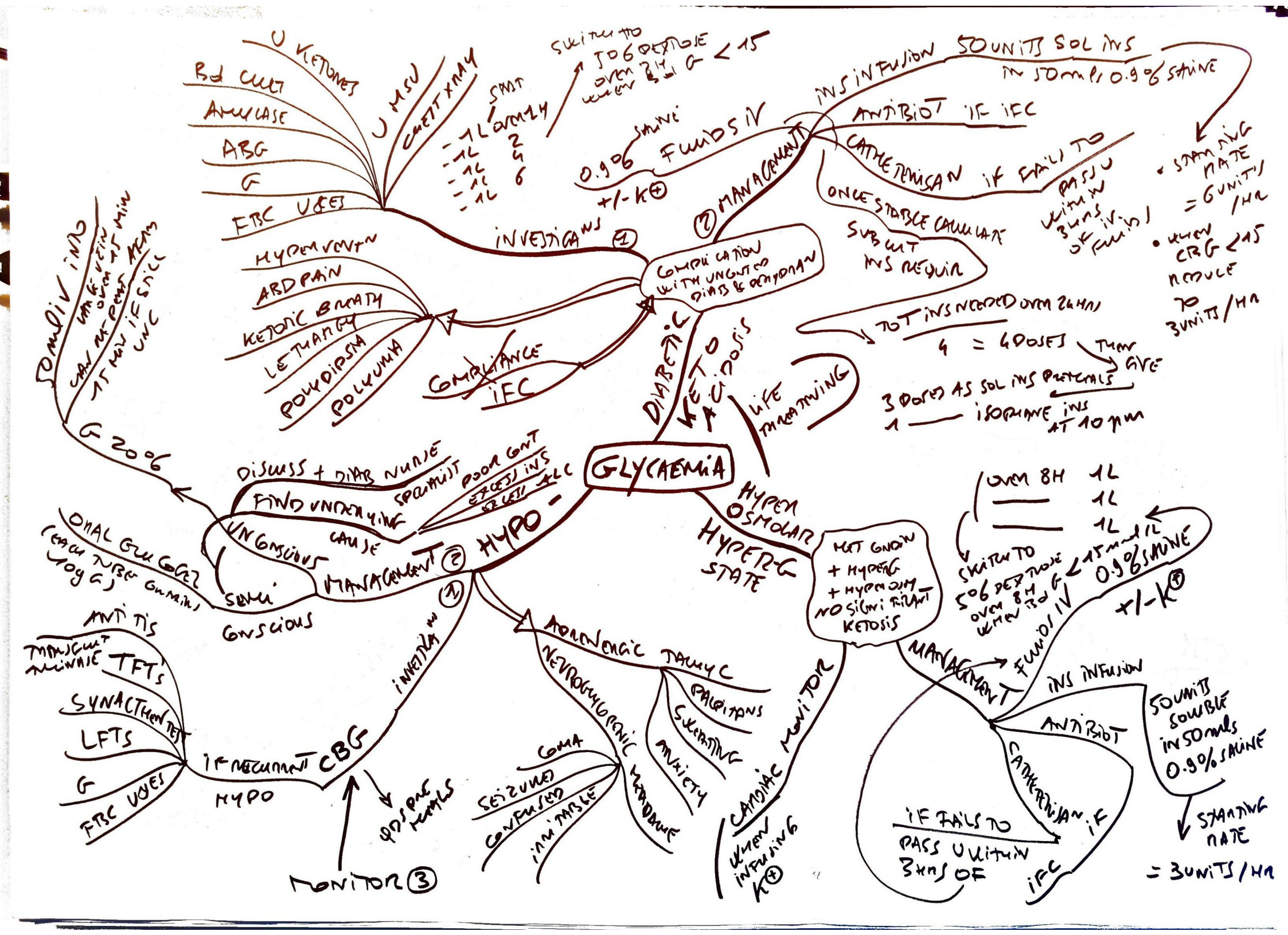
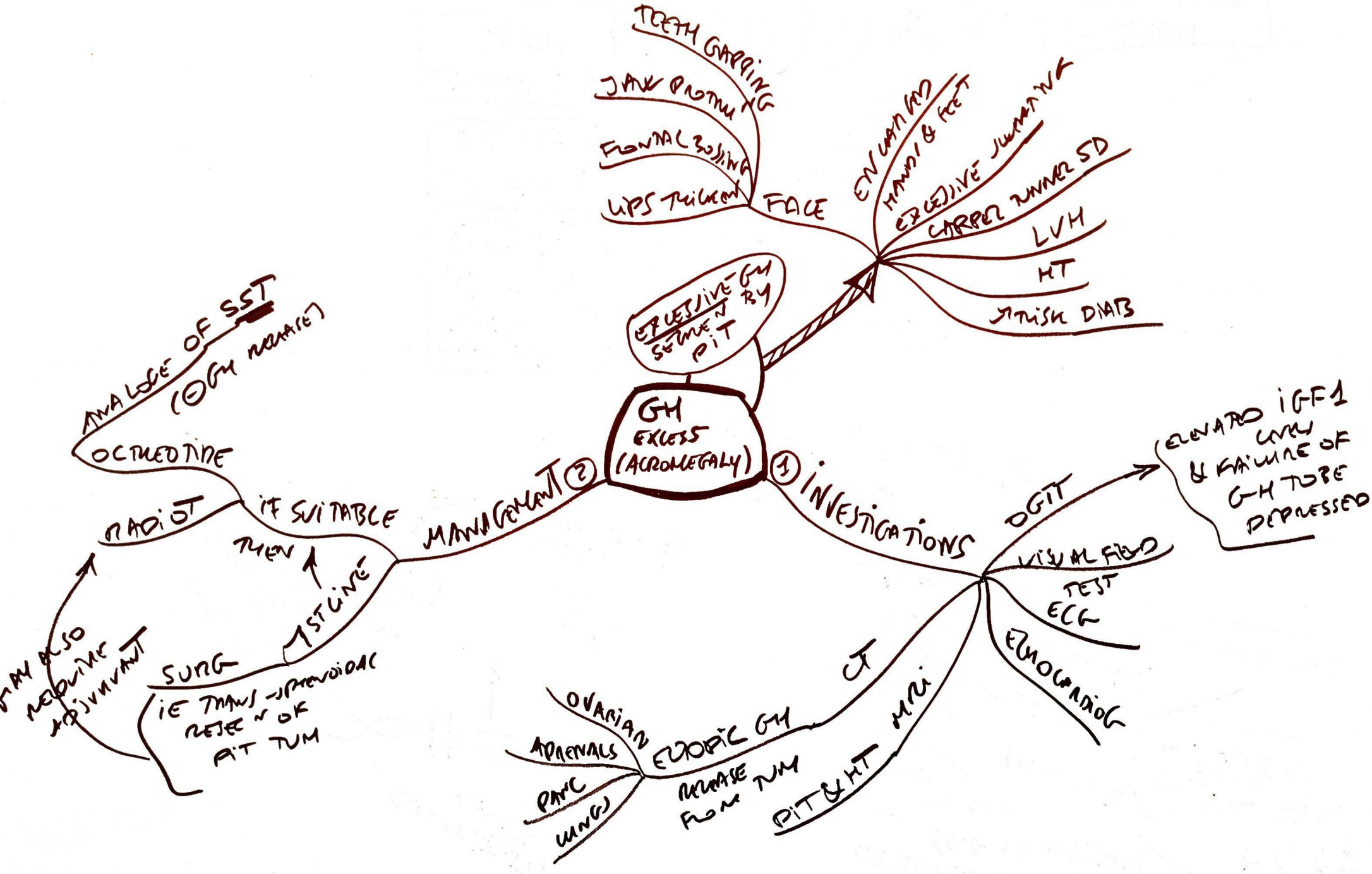
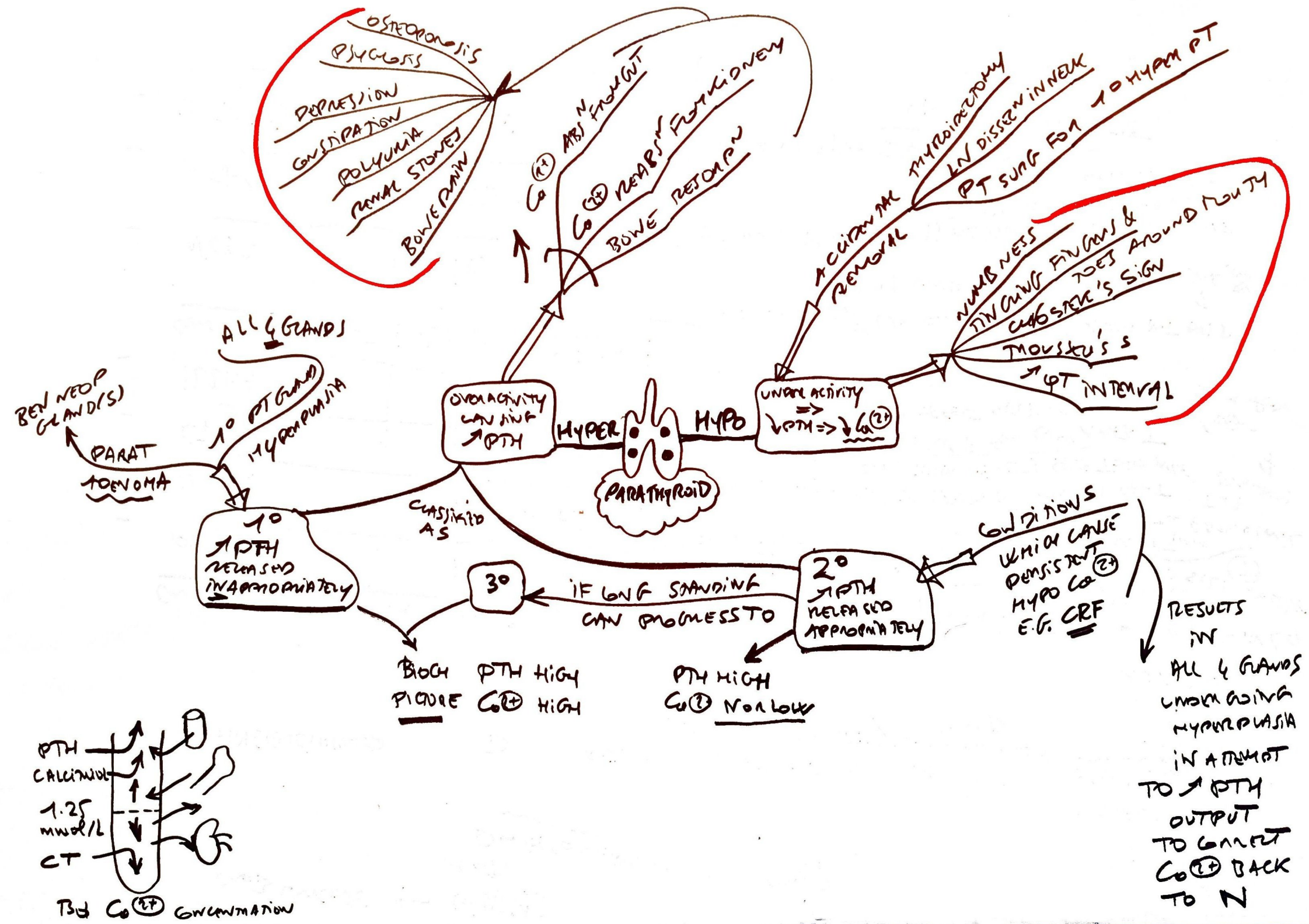

ADRENAL
HYPER
GLUCOCORTICOID (CUSHING'S D)
INVESTIGATIONS
3 MANAGEMENT
ADRENALECTOMY
THYMUS
PITADENOMA
ACTH
(CUSHING'S D)
SPHENOIDAL
HYPOPHYSECTOMY
CORTISOL
EXOG ADMINISTN OF CS
TUM IN PIT
TUM IN ADR
SMALL CELL LUNG CANCER
ACTH
TUM
ELSEWHERE
TO CONFIRM 1
24 HR URINARY CONTROL
LEVELS
CUSHING'S SD 2
CT ABD
MRI HEAD
PLASMA ACTH
TO DETERMINE CAUSE
WEIGHT GAIN
TRUNCAL OBESITY
MOONFACE
STRIAE
PROX MYOPATHY
HYPERGLYCAEMIA
HIRSUTISM
ACNE
ALDOSTERONE
1° (CONN'S SD)
2°
(⊖ FB)
RENIN
(APPROPRIATE ADR → TO → BY RENIN)
RENAL BF ↓
LVF
END STAGE
CHRONIC LIV D
NEPHROTIC
INVESTIGATIONS 1
PLASMA ALD / RENIN RATIO
CT ABD
IFC
TRAUMA
SURG
SPIRONOLACTONE
TUM IN ADR
BILAT ADR
HYPERPLAS
↑ Na⊕ & H2O REABN
↓ K⊕
↑ BP
ADRENAL
HYPO
CHRONIC
ADDISON'S D
AUTOIMM ADRENALITIS
CASEOUS NEC 2° TO TB
PROG DESTRUCTN ⇒ ↓ GC & MC
INVESTIGATIONS 1
MANAGEMENT 2
REPLACE
HYDROCORTISONE GC
FLUDROCORTISONE MC
LITTLE OR NO
↑ ACTH
ADDISON
↑ N
SYNACTHEN
ACTH STIMULATN TEST
VGES
CORTISOL LEVELS
LETHARGY
WEAKNESS
ANOREXIA
NAUS & VOMIT
DIAR
↑ SKIN PIGMTN
↓ WEIGHT LOSS
POSTURAL HYPOT
ACUTE
SUDDEN LOSS OF FUNCTION
ACUTE INSULT IN PT WITH KNOWN ADD
SEVERE SEPTICAEMIA
SUDDENLY STOPPING EXOG GC
HYPOVOL SHOCK
HYPO G
↓ Na⊕ ↑ K⊕
INVESTIGATIONS 1
Bld CULT
URINE SPUTUM CULT
Bld CORTISOL & ACTH LEVELS
MANAGEMENT 2
ANTIBIOT
DEXTROSE IV
HYDROCORTISONE IV
IV FLUIDS STAT
NEG MCASS 1°H
G AII MC ALD
F ACTH, CRH GC GRT
R ACTH, CRH AND DHEA


DIABETES
① MANAGEMENT
② MANAGEMENT
③ COMPLICATIONS
④ INVESTIGATIONS
LASER
PHOTO COAGULAN
AIMS TO MAINTAIN VISION (IT CANNOT IMPROVE IT)
CAUTERIZING SEALING BD VESSELS
MACULOPAT
IF OCCURS WITHIN 1 DISC DIAM OF CENTRE OF FOVEA KNOWN AS
SOFTER DATES IE SUPERFICIAL RETINAL INFARCTS
COTTON WOOL SPOTS
PREPROLIFERATIVE
VITR HEMOR
NEO VASC
RET DETACHMENT
PROLIFERATIVE
PLASMA LEAKAGE IN DEEP LAYER
HAMOEXUDATES
BLOT HEMOR
FOV MACULAR
BACKGROUND
RETINOPAT
MICROVASC
ACUTE
MACROVASC
DISTAL PORTION AFFECTED 1ST HENCE GLOVE & STOCKING DISTRIBUN
NEUROPAT
NEPHROPAT
PERIPH VASCD
STROKE
MI
ATHEROS
GR AA
POLYURIA
POLYDIPSIA
UNEXPLAINED WEIGHT LOSS
and ↑ PLASMA G ⇑ INSUFACTVINS
ST OF DIABETES
THEN
1) EITHER FASTING PLASMA G ≥ 7 mmol/L
AND
2) RANDOM OR ≥ 11.1
IF VALUES CROSS THRESHOLD THEY MAY STILL SHOW IMPAIRMENT
FASTING PG = 6.1-7 → IMPAIRED FASTING TOLERANCE
OR
AFTER ORAL G TOL TEST RANDOM PG = 7.8-11 → IMPAIRED G TOL
CONSERVATIVE
↓ WEIGHT
↑ EXER
DIET CNT
MED
SEE DMA ★ % HONK
↓ G GEN & ↑ PERIPH UTILISATION
METFORMIN
↑ INS REL
↓ GAIN
RISK ↓G
IP OVER WK
SULPHONYLUREAS
PIOGLITAZONE
↓ PERIPH INS RESIST
↑ INS REL
GLIPTINS (VILDA-)
↓ G GEN
GLUCAGON LIKE PEP 1 AGO (EXENATIDE)
ACTRAPID SA
FLEXTARD 30 IA
LANTUS LA
INSULIN


GLYCAEMIA
HYPO
MANAGEMENT ②
DISCUSS + DIAB NURSE SPECIALIST
FIND UNDERLYING CAUSE
POOR CONT
EXCESS INS
SEVERE ALC
UNCONSCIOUS
ORAL GLUCOSE (EACH TUBE CONTAINS 10g G)
SEMI CONSCIOUS
G 20%
SOMOLIV INJ
MANAGEMENT ①
INVESTIGATIONS
CBG
IF RECURRENT HYPO
TFTs
SYNACTHEN TEST
LFTs
G
FBC U&Es
ANTI TBs
MONITOR ③
HYPERG
HYPERG STATE
HYPER OSMOLAR
MET CONDN + HYPERG + HYPOKALAEMIA NO SIGNIFICANT KETOSIS
SKIN TENTING
SLOW CAPILLARY REFILL
DIABETIC KETOACIDOSIS
LIFE THREATENING
COMPLICATION WITH UNCONTROLLED DIABETES MELLITUS
COMPLIANCE
IFC
HYPERVENTN
ARD PAIN
KETONIC BREATH
LETHARGY
POLYDIPSIA
POLYURIA
U KETONES
U GLUCOSE
U MSU
CHEST X RAY
ABG
G
FBC U&Es
SKIN TENTING
- 1L 2
- 1L 4
- 1L 6
- 1L
0.9% SALINE
FLUIDS IV
+/- K+
MANAGEMENT ②
INS INFUSION
50 UNITS SOL INS IN 50ml 0.9% SALINE
ANTIBIOT IF IFC
CATHETERISAN IF FAILS TO PASS U WITHIN 3 HRS OF FLUIDS
ONCE STABLE CANULAT
SUBCUT INS REQUIR
TOT INS NEEDED OVER 24 HRS
4 = 4 DOSES
THEN GIVE
3 DOSES AS SOL INS PRECEDES
1 — ISOPHANE INS AT 10 pm
STARTING RATE = 6 UNITS/HR
WHEN CBG < 15 REDUCE TO 3 UNITS/HR
SKIN TENTING TO 5% DEXTROSE OVER 24 HR WHEN BGL < 15
OVER 8H 1L
— 1L
— 1L
SKIN TENTING TO 5% DEXTROSE OVER 8H WHEN BGL < 15 mmol/L
0.9% SALINE
+/- K+
MANAGEMENT
FLUIDS IV
INS INFUSION
ANTIBIOT
CATHETERISAN IF
IF FAILS TO PASS U WITHIN 3 HRS OF
IFC
50 UNITS SOLUBLE INSULIN IN 50 mls 0.9% SALINE
STARTING RATE = 3 UNITS/HR
ADRENERGIC TACHYC
PALPITATIONS
SWEATING
ANXIETY
NEUROGLYCOPENIC MANIFESTATIONS
COMA
SEIZURES
CONFUSED
IRRITABLE
CARDIAC MONITOR
WHEN INFUSING K+
INVESTIGATION
QDSPAC PROTOCOLS
MONITOR ③


GH EXCESS (ACROMEGALY)
EXCESSIVE GH SEEN BY PIT
FACE
TEETH GAPPING
JAW PROTRUSION
FRONTAL BOSSING
LIPS THICKENED
ENLARGED HANDS & FEET
EXCESSIVE SWEATING
CARPEL TUNNEL SD
LVH
HT
TRUE DIAB
1 INVESTIGATIONS
OGTT
ELEVATED IGF1 CAN'T SUPPRESS & FAILURE OF GH TO BE DEPRESSED
VISUAL FIELD TEST
ECG
ECHOCARDIOGRAM
CT
MRI
PIT & HT
ECTOPIC GH
OVARIAN
ADRENALS
PANC
LUNGS
RELEASE FROM TUM
2 MANAGEMENT
IF SUITABLE
OCTREOTIDE
ANALOGUE OF SST
(↖GH RECEPTOR)
RADIOT
THEN
1ST LINE
SURG
IE TRANS-SPHENOIDAL RESECTION OF PIT TUM
MAY ALSO RECEIVE ADJUVANT


OSTEOPOROSIS
PSYCHOSIS
DEPRESSION
CONSTIPATION
POLYURIA
RENAL STONES
BONE PAIN
Ca(2+) ABS FROM GIT
Ca(2+) REABS FROM KIDNEY
BONE RESORPN
ACCIDENTAL THYROIDECTOMY
LN DISSECN IN NECK
PT SURG FOR 1° HYPER PT
ACCIDENTAL REMOVAL
NUMBNESS
TINGLING FINGERS & TOES AROUND MOUTH
CHOSTEK'S SIGN
TROUSSEU'S S
↑QT INTERVAL
ALL 4 GLANDS
BEN NEOP GLAND(S)
PARAT ADENOMA
1° PT GLAND HYPERPLASIA
OVERACTIVITY CAUSING ↑PTH
HYPER
HYPO
PARATHYROID
UNDERACTIVITY ⇒ ↓PTH ⇒ ↓Ca(2+)
CLASSIFIED AS
1° ↑PTH RELEASED INAPPROPRIATELY
3°
IF LONG STANDING CAN PROGRESS TO
2° ↑PTH RELEASED APPROPRIATELY
CONDITIONS WHICH CAUSE PERSISTENT HYPO Ca(2+) E.G. CRF
RESULTS IN ALL 4 GLANDS UNDERGOING HYPERPLASIA IN ATTEMPT TO ↑PTH OUTPUT TO CORRECT Ca(2+) BACK TO N
BLOOD PICTURE
PTH HIGH
Ca(2+) HIGH
PTH HIGH
Ca(2+) NORMAL/LOW
PTH
CALCITRIOL
1.25 mmol/L
CT
Bld Ca(2+) CONCENTRATION

THYROID
SOLITARY NODULE
SIMPLE GOITRE
HYPO
HYPER
FNAC
INVESTIGATIONS 1
MANAGEMENT 2
PALPABLE DISCRETE SWELLING IN OTHERWISE APPARENTLY
ENLARGED TOF UNKNOWN ETIO
MANAGEMENT
NIL
SURG FOR COSMETIC REASON
IF COMPRESSES TRACHEA
MULTINODULAR GOITRE
DIFFUSE PAINLESS GOITRE
POSSIBLE ALLEVIATION
AS IT USUALLY REGRESSES SPONTANEOUSLY
THORM ARE PRODUCED
⇒ ↑TSH IN AN ATTEMPT
TO BRING BACK LEVELS TO N
⇒ HYPERPLASIA GLAND
OVER MANY YEARS THIS STIMULATION CAN CAUSE NODULES TO FORM
TOTALLY OR TLOBECTOMY
PAL
SURG IF
COMPLICATIONS OF SURG
THYROTOXIC STORM
BLEEDING
LAR EDEMA
REC LAR N PAR
HYPO
UNDERACTIVITY + ↘THYR HORM RELEASE
TFTS
INV 1
↓TSH ↑T3 ↑T4
RADIOISOT SCAN
↑ CAUSES
SURG
MAN 2
RADIACTIVE IODINE MED
CARBIMAZOLE
IF NOT TOLERATED
PROPYLTHIOURACIL
TOXIC MULTINODULAR GOITRE
FUNCT FOL ADENOMA
ANOD WITHIN A MULTINODULAR GOITRE WITH A DIVOT OF ↓ IN DOMINANT NODULE
↓T HORM INDEP OF TSH
TBEN TUM T WITH A SOLITARY NODULE
PERIORBITAL EDEMA OR PLETHORA
EXOPH
OPHTHALMOP
DIFFUSE
SYMPATHETICAL GOITRE
PRET BIND MYXOEDEMA
WEIGHT LOSS
PROX MYOP
TREMOR
AF
HEAT INTOL
TACHYC
LID LAG
RETRACTION
SWEATING
MORE LIKELY IF ITIS A
INVESTIGATION 1
MANAGEMENT 2
TFTS
↑TSH
↓T4
SUCKS
THYROXINE MED
LITHIUM
AMIODARONE
POST PARTUM
IODINE III
HASHIMOTO THYROIDITIS
INITIALLY
LATER
DEJUNCTION BY LA
INFERIOR ++
LA
DIFFUSE +
FINAL GOITRE
GOITRE
AUTOIMM
↘FOL ↓ → DEJUNCTION
COMMON DIAGNOSIS
MENTAL SLOWING
DRY SKIN
PUFFY FACE
COLD INTOL
BRADYC
WEIGHT GAIN
GOITRE
DIFFUSE
SYMPATHETICAL GOITRE
PRET BIND
MYXOEDEMA
LID LAG
RETRACTION
SWEATING
HEAT INTOL
TACHYC
PROX MYOP
AF
WEIGHT LOSS
TREMOR
EXOPH
OPHTHALMOP
DYSPH
DYSPHAGIA
EXTENSIVELY INVADES
ADJ TISS IN NECK
ANAPLASTIC
MALT
CARCINOMAS
FOLLICULAR ADENOMA
FOLLICULAR 20-60
PAPILLARY 70%
MET TO REG LN CAN BE △ OR USING FNA
CANNOT ≠
NEED HISTO EXAM
VIA BLOOD STREAM
LUNG & BONE
METS
VERY POOR TT
EXTENSIVELY INVADES
ADJ TISS IN NECK
METS
ANISEN FROM PANAFOLD
MEDULLARY
4 TYPES
DYPN
→CT
EXCELLENT TT
HOWEVER SLOW GROWING
LIFE-LONG THYROXINE
THOUGH