



HTA et laboratoire

Cours Labmed 2^{ème} partie
CPLN Neuchâtel 15 mars 2010



labmed
schweiz suisse svizzera
Section romande



Ecole
Supérieure
du canton
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HTA (hypertension artérielle) et laboratoire

- Définition de l'HTA
- Conséquences de l'HTA
- Prise en charge par le médecin
- Laboratoire et HTA:
 - Bilan de base
 - Bilan étiologique: causes endocrines?

Prise en charge par le médecin

1. Mesure de la pression: HTA ?
2. Bilan de base
 1. Autres FRCV (diabète, lipides, tabac, ...)
 2. Conséquences de l'HTA (atteinte des organes cibles)
 3. Cause HTA?
3. Evaluation du risque CV global
4. Traitement



Définition HTA

- **HTA** = pression artérielle mesurée $>$ seuil
- **HTA** si pression
 - ≥ 140 mmHg systole et/ou
 - ≥ 90 mmHg diastole



Mesure de la pression artérielle



Mesure de la pression artérielle

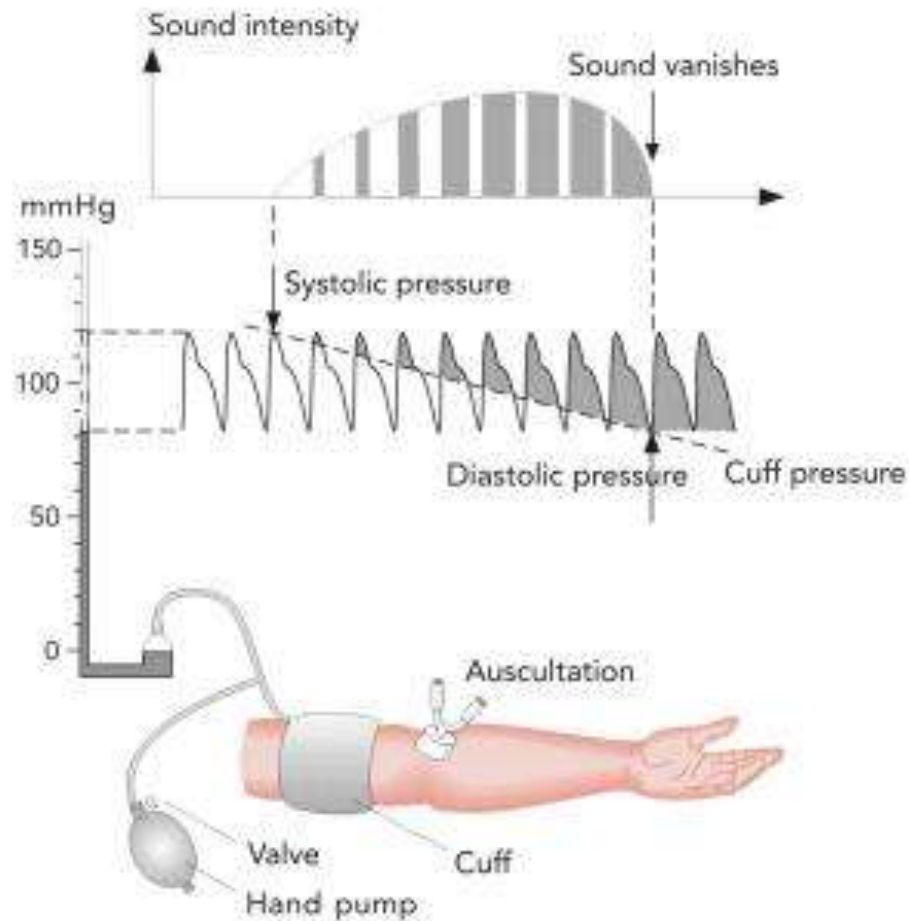


Figure 6-7 Blood pressure measurement with cuff

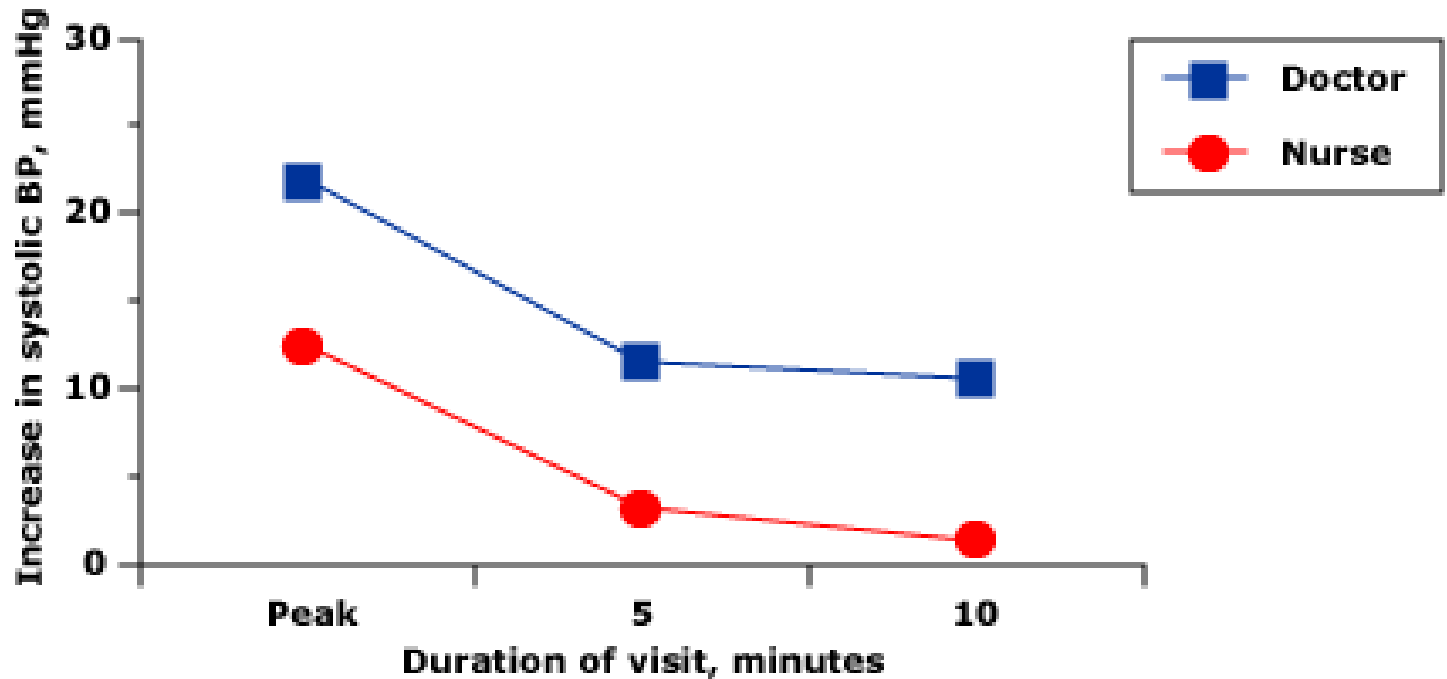
Mesure de la pression artérielle



Mesure de la pression artérielle MAPA (mesure ambulatoire de la pression artérielle ou Remler)



Mesure de la pression artérielle: effet « blouse blanche »



Mesure de la pression artérielle: effet « blouse blanche »

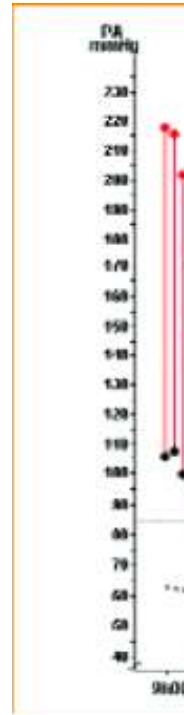


Fig. 1. Enregistrement
PAS: pression artér

Mesure de la pression artérielle: effet « blouse blanche »

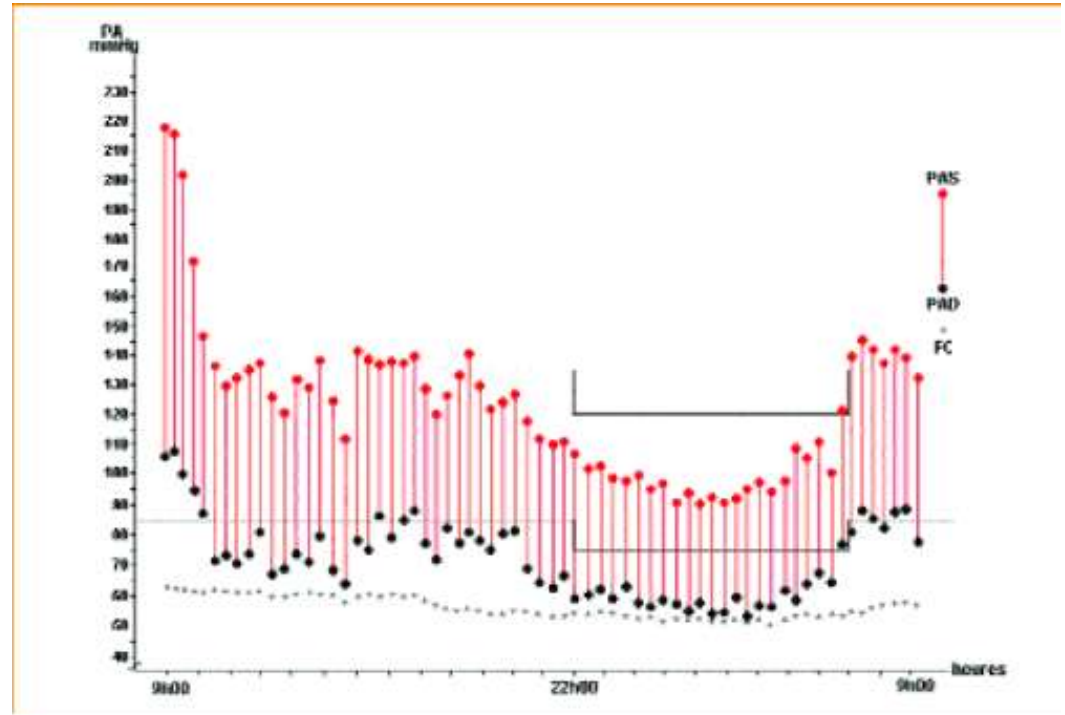


Fig. 1. Enregistrement de la pression artérielle de 24 heures.

PAS: pression artérielle systolique; PAD: pression artérielle diastolique; Fc: fréquence cardiaque.



Définitions HTA

BP Classification	Systolic BP, mm Hg*		Diastolic BP, mm Hg*
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Stage 1 hypertension	140-159	or	90-99
Stage 2 hypertension	≥160	or	≥100
Stade 3	≥180	ou	≥110

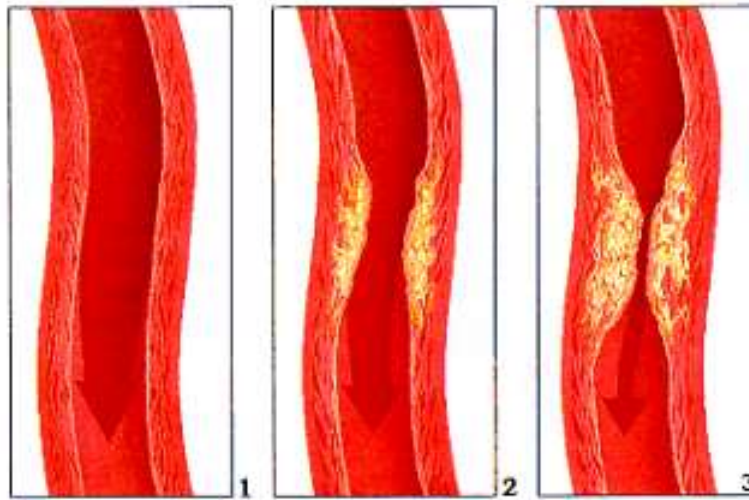
~~« La bonne pression c'est 120 + l'âge »~~

$$\del{120 + 75 \text{ ans} = 195 \text{ mmHg}}$$

Pourquoi dépister l'HTA ?

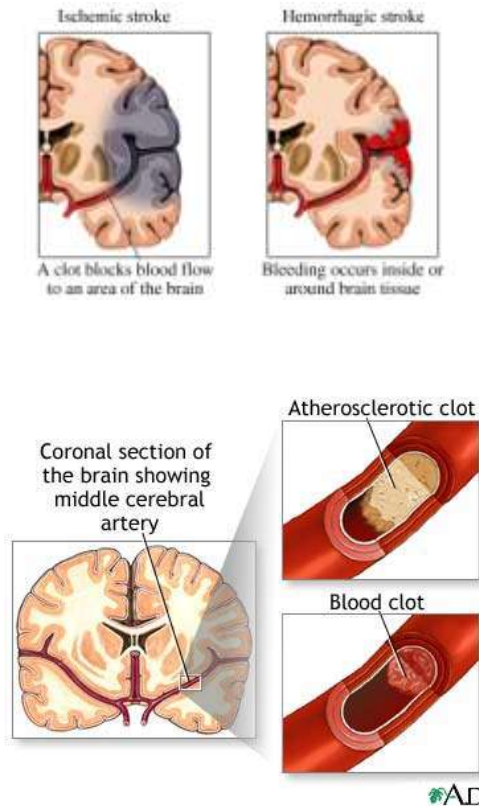
- « maladie » silencieuse !! (aucun symptômes, cf cholestérol, ...)
- C'est un **FRCV**
- Les **FRCV** → ↗ **risque** de complications cardiovasculaires

FRCV → ↗ athéromatose

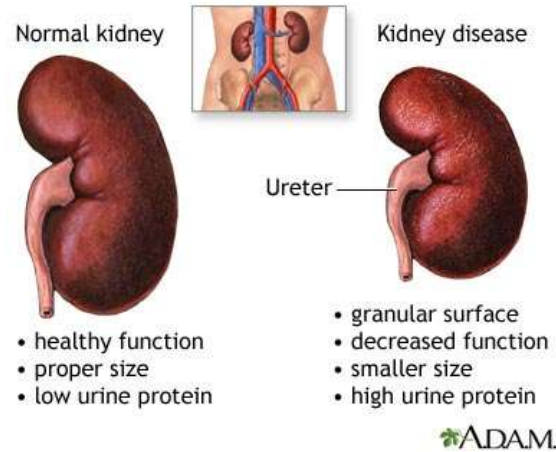


Conséquences à long terme de l'HTA

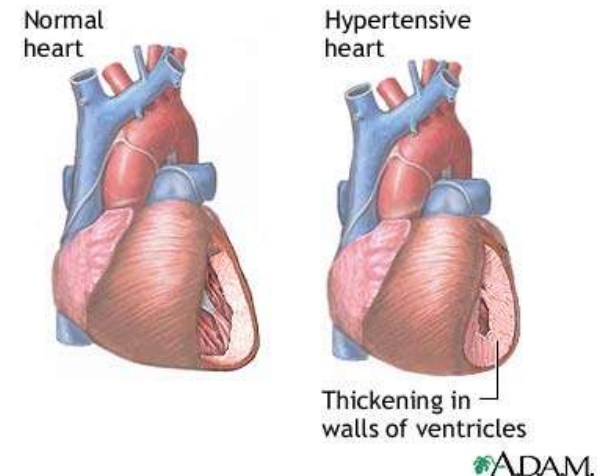
AVC



IRC



IC



FRCV: estimation du risque CV global

FRCV modifiables

- Tabac
- Diabète
- Hypercholestérolémie
- Obésité
- Sédentarité
- Stress, dépression
- Alimentation (régime méditerranéen, régime DASH)
- ...



FRCV non modifiables

- Age
- Antécédents familiaux
- Génétique

Stratification of CV risk

Blood pressure (mmHg)					
Other risk factors, OD or disease	Normal SBP 120–129 or DBP 80–84	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP $\geq$ 180 or DBP $\geq$ 110
No other risk factors	Average risk	Average risk	Low added risk	Moderate added risk	High added risk
1–2 risk factors	Low added risk	Low added risk	Moderate added risk	Moderate added risk	Very high added risk
3 or more risk factors MS, OD or diabetes	Moderate added risk	High added risk	High added risk	High added risk	Very high added risk
Established CV or renal disease	Very high added risk	Very high added risk	Very high added risk	Very high added risk	Very high added risk

Stratification of CV risk in four categories of added risk. SBP=systolic blood pressure; DBP=diastolic blood pressure; CV=cardiovascular; HT=hypertension; OD=subclinical organ damage; MS=metabolic syndrome.

Prévention primaire et prévention secondaire

Prévention primaire

- Population en bonne santé:
« check-up »

Prévention secondaire

- ECG: HVG?
- IRC?
- Protéinurie/microalb?
- AVC
- IC
- Maladie coronarienne
- IAMI (souffle fémoral)

Traitement



Exercise and maintenance of a healthy weight

Lifestyle changes and/or medication may reduce high blood pressure to healthy levels

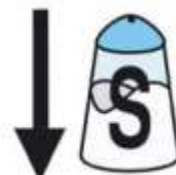


Medications such as diuretics, beta-blockers, calcium channel blockers and ACE inhibitors

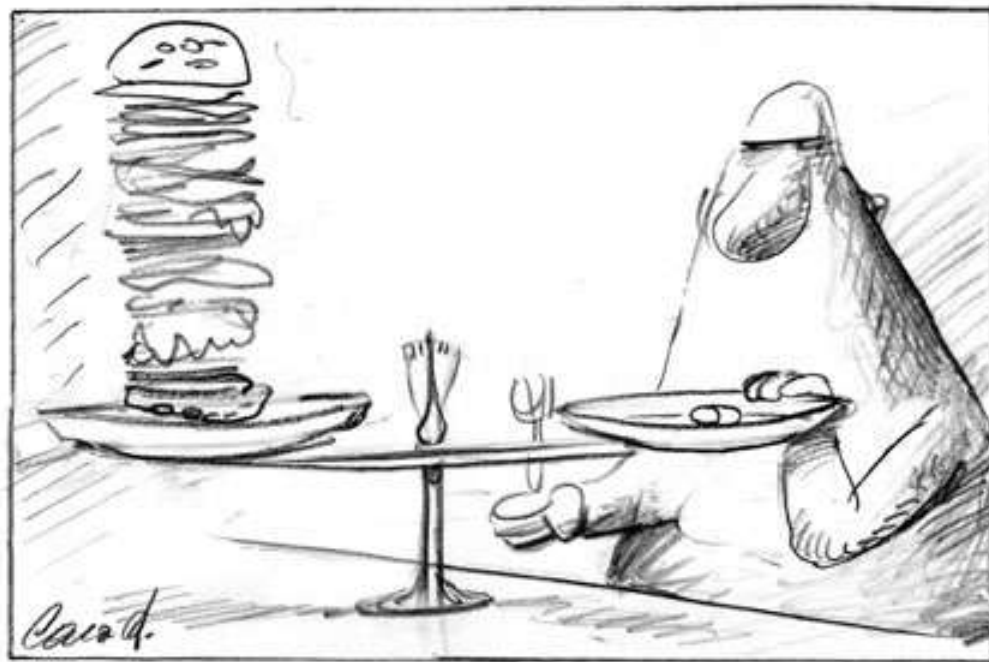
A healthy, low-salt diet rich in natural sources of vitamins and minerals



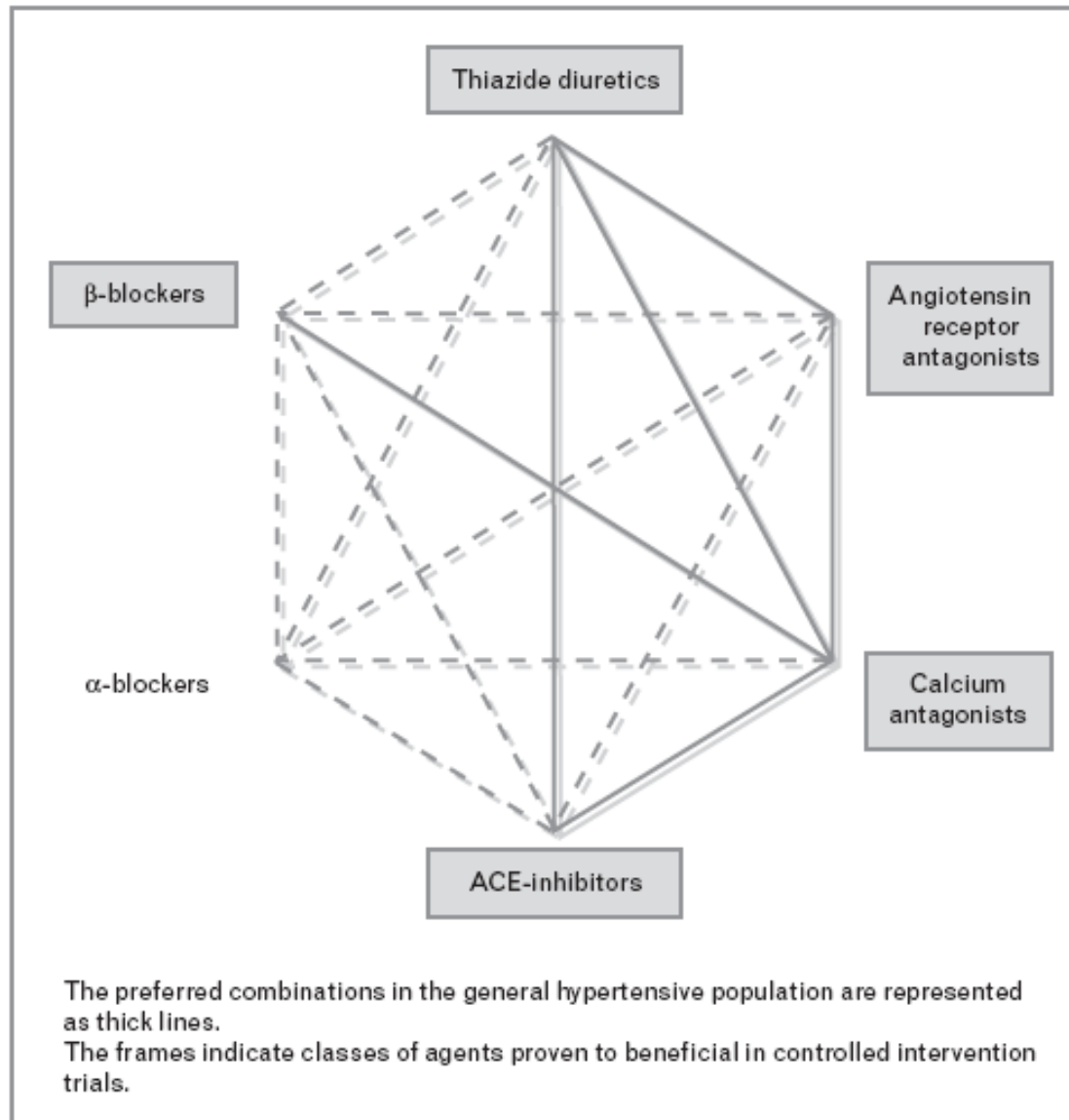
ADAM.



Eat healthy food, reduce salt and exercise regularly.



Combinaisons thérapeutiques



Traitement

- Mesures comportementales (« hygiène de vie », lifestyle)
- Médicaments
- Baisser la pression artérielle
 - < 140/90 mmHg
 - < 130/80 mmHg
 - Voire < 125/75 mmHg
- But: prévenir les complications à long terme

HTA (>140/90 mmHg) → bilan biologique

- **Sang:**

- Glycémie à jeun
- Lipides:
 - Cholestérol total
 - HDL
 - LDL
 - (trigly)
- Créatinine, K⁺, (Na⁺) : cf dia suivantes

- **Urine:** cf dia suivantes

- Stix (bandelette) +/- sédiment si stix +
 - prot/créat (si prot+ au stix)
 - alb/créat (si stix négatif)

- ECG: HVG ? Ancien infarctus ?

Kaliémie

- Hypokaliémie:
 - Effet secondaire du traitement:
 - Diurétiques
 - Hyperaldostérisme ??

Status-sédiment urinaire (SSU) + spot

- SSU:
 - Hématurie ? (glomérulaire)
 - Protéinurie ?
- Rapport albumine/créatinine (microalbuminurie) et protéine/créatinine (protéinurie franche)

Continuum

Protéinurie:

$N < 200 \text{ mg/24h}$

Protéinurie:

$> 200 \text{ mg/24h}$

Protéinurie

néphrotique:

$> 3000 \text{ mg/24h}$



Albuminurie:

$N < 30 \text{ mg/24h}$

Microalbuminurie:

$30 - 300 \text{ mg/24h}$

Macroalbuminurie:

$> 300 \text{ mg/24h}$

Concentration vs prot/créat

- Individu X avec néphropathie diabétique et hypertensive: protéinurie à **500 mg/24h**
 - Urine A: 6 heures dans la vessie, 100 ml
 - $500 \text{ mg} / 4 = 125 \text{ mg} / 100 \text{ ml} = 1.25 \text{ g} / \text{l}$
 - Urine B: 6 heures dans la vessie, 1000 ml
 - $500 \text{ mg} / 4 = 125 \text{ mg} / 1000 \text{ ml} = 0.125 \text{ g} / \text{l}$
- Intérêt du rapport prot/**créat** ou albumine/**créat** → estimation/24h !!
(remplace >95% les urines de 24h)

COMPLET	Date de prélèvement	10.08.08	12.08.08	14.03.10
Edité le	Heure prélèvement	19 h 15	15 h 15	07 h 00
14.03.10 à 17 h 20	Demande numéro	AD0832 3784	AD0833 1261	AD1010 4246
Rapport n° 1	Reçue le	10.08.08	12.08.08	14.03.10
Page 1 / 1	à (site)	19 h 16 (PO)	15 h 20 (PO)	13 h 30 (PR)
Analyses	Val. us.	Unités		
URINE				
<u>Status urinaire:</u>				
Couleur		jaune	[A]	jaune
Aspect		claire	trouble	claire
Glucose ql		négatif	négatif	négatif
Bilirubine		négatif	faible	négatif
Corps cétoniques		négatif	1.5	négatif
Poids spécifique		1020	1025	1010
Sang occulte		faible	faible	négatif
pH		6.0	6.0	5.0
Protéines	g/l	env 1.0	env 1.0	env 1.0
Urobilinogène	µmol/l	env 3.2	env 3.2	env 3.2
Nitrites		négatif	négatif	négatif
Leucocytes (qualit.)		traces	faible	faible
<u>Sédiment urinaire:</u>				
Cellules épithéliales		+	nombreuses	++
Leucocytes, /champ		1-3	12-15	4-6
Erythrocytes, /champ		0-2	7-10	rares
Cylindres		négatif	+ [B]	+ [D]
Cristaux		négatif	+++ [C]	négatif
Germes		+	+++	+
Autres éléments		--	--	--
Chimie quantitative:				
Sodium	7 - 200	mmol/l		48
Potassium	17 - 200	mmol/l		22
Chlore		mmol/l		72
Osmolalité	850 - 1350	mosmol/kg		* 313 [E]
Créatinine		µmol/l		9040
Protéines totales	< 200	mg/l		* 312
Rapport Protéine/Créatinine		mg/mmol		34.5
Protéinurie 24 h estimée		mg		345
Albumine immunologique	< 20	mg/l		* 150 [E]
Urée		mmol/l		132.0

Causes de l'HTA

>95% des HTA: HTA primaire ou essentielle (pas de cause spécifique)

Facteurs favorisants:

- SAS
- AINS
- OH
- Obésité
- Sel alimentaire
- Pillule

HTA secondaire:

- Endocrine:
 - Phéochromocytome
 - Cushing
 - Hyperaldo primaire
- HTA rénovasculaire
- IRC
- Coarctation aortique

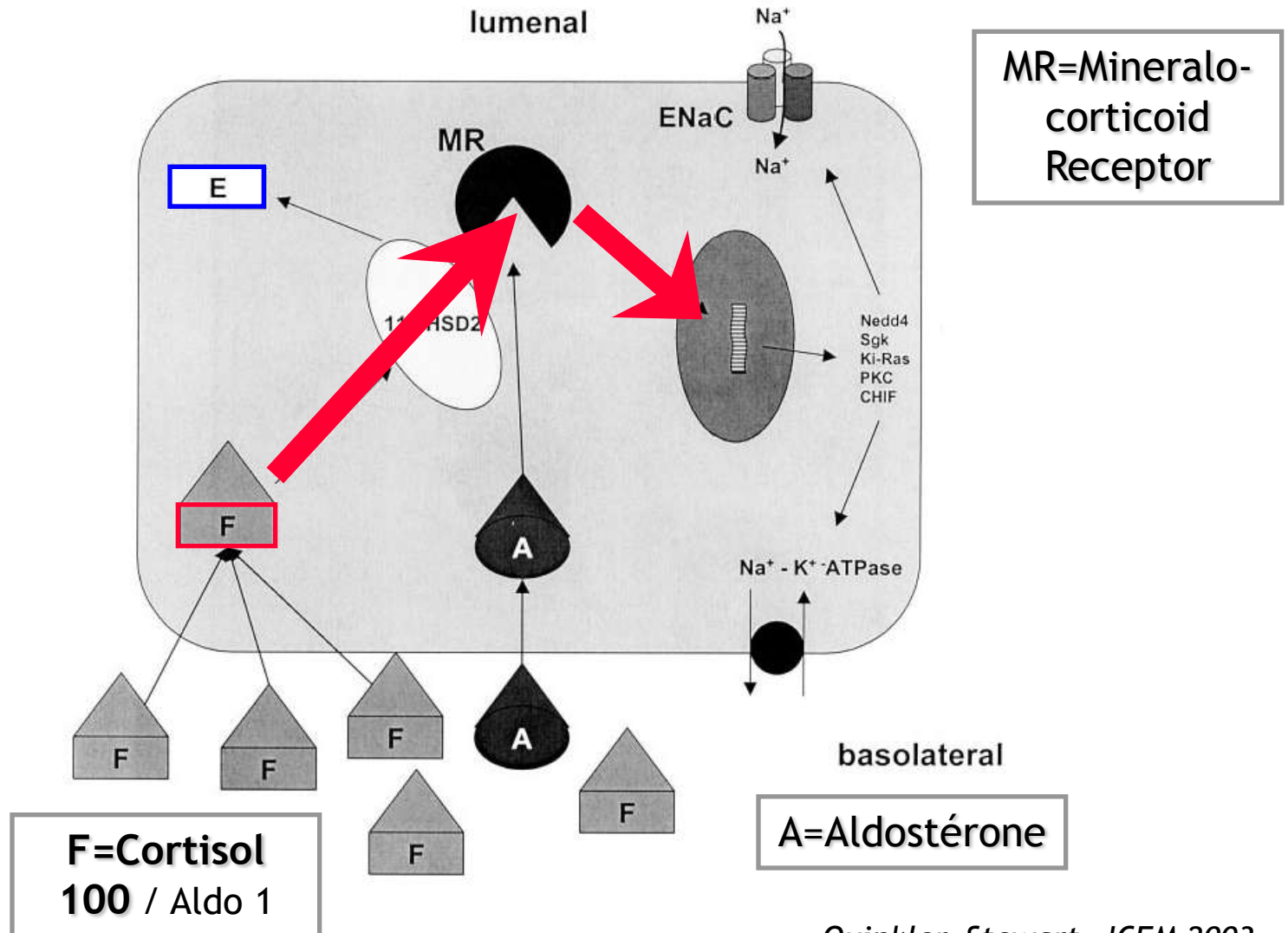
Causes endocriniennes d'HTA (expliquent rarement à elles seules une HTA):

- Thyroïdiennes:
 - Hypothyroïdie
 - Hyperthyroïdie
- Hyperparathyroïdie
- Acromégalie

Causes endocriniennes d'HTA:

- Apparent mineralocorticoid excess (AME) / 11 β -hydroxystéroïde deshydrogénase type 2
 - Héritaire
 - Mutations → Déficit enzymatique 11 β -HSD 2
 - Acquis
 - Régliste: acide glycyrrhizique (inhibition 11 β -HSD 2)
 - Cushing (saturation 11 β -HSD 2)

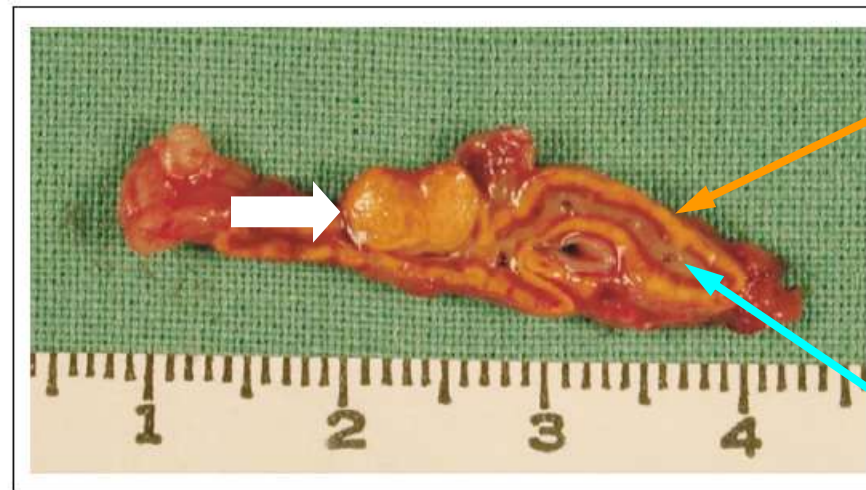
Cortisol F $\rightarrow$ 11 β -HSD 2 $\rightarrow$ Cortisone E



Causes endocriniennes d'HTA:

- **Hyperaldostéronisme primaire**
 - Le moins rare (jusqu'à 10% des HTA **réfractaires**)
- Phéochromocytome
- Syndrome de Cushing

Hyperaldostéronisme primaire

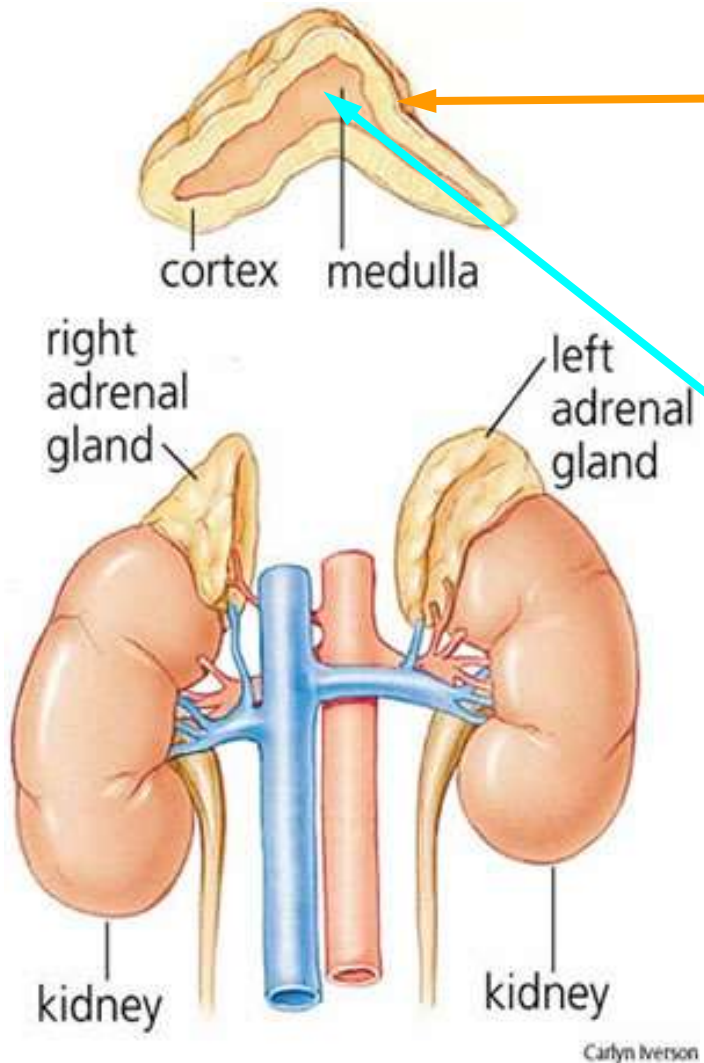


Cortico-
surrénale

Médullo-
surrénale

Fig. 1. Aldosterone-producing adenoma missed by computer-aided tomography (CAT), but identified by adrenal venous sampling. Left adrenal gland containing a 7-mm aldosterone-producing adenoma removed from a 37-year-old normokalaemic, hypertensive man who was requiring three antihypertensive agents to maintain hypertension control, and who had a raised aldosterone:renin ratio and primary aldosteronism (PAL) confirmed by fludrocortisone suppression testing (FST). Although CAT scanning failed to detect an adrenal mass lesion, adrenal venous sampling lateralized aldosterone production to the left adrenal. Laparoscopic left adrenalectomy led to cure of hypertension and biochemical cure of PAL (repeat FST).

Hormones et surrénale



Cortex:

- Cortisol
- Aldostérone
- Androgènes

Medulla:

- Catécholamines
 - Noradrénaline
 - Adrénaline
 - (dopamine)

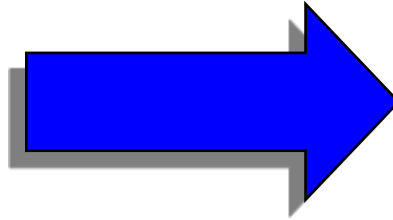
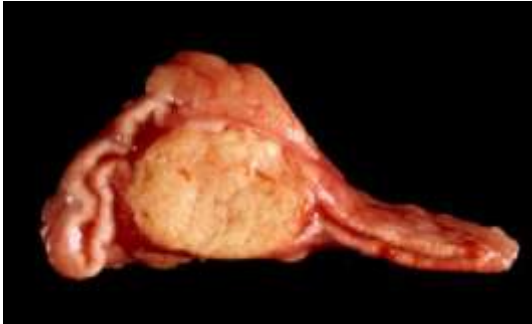
TABLE I. M. W., FEMALE, 34 YEARS OF AGE. ADMITTED TO METABOLIC RESEARCH LABORATORY, APRIL 27, 1954.

Chronic complaint	Intermittent spasms, weakness and paralysis of muscles for 7 years.
Patient interview	October, 1947, "paralyzed" from hips down for 2 days. Several similar attacks since but less severe and shorter. Frequent attacks of great muscular weakness. Other attacks of typical tetany involving upper and sometimes lower extremities. Well between attacks. Blood pressure known to be 180 to 190 for 4 years. Trace of urinary albumin repeatedly. Polyuria and nocturia for years. No vomiting, diarrhea, or drugs
Physical examination	Well-developed musculature. Positive Chvostek and Trousseau signs. Blood pressure 176/104. Heart size normal. No edema. Reflexes hyperactive

TABLE II. INITIAL LABORATORY VALUES, M. W.

BLOOD VALUES		URINARY AND RENAL VALUES	
1. Counts normal, Hb	14.4 Gm.	1. Urine protein	O to Tr., Sed. neg.
2. Eosin	198/c. mm.	2. pH	7.17-7.54
3. Serum creatinine	0.54-1.02	3. End. Creat. Clearance	125-170 L./day
4. NPN	33-38 mg. %	4. PSP 50% in 15 min., 80% in 2 hr.	
5. Sugar	84 mg. %	5. 18 h conc. test	sp. gr. 1.011
6. Serum Protein	6.9%, alb. 4.9%	18th h 10 mg. pitressin. 20th h	sp. gr. 1.011
7. Serum Ca	10.2 mg. %	6. Ca 122 mg./day with intake	137 mg./day
8. Serum Inorg. P.	2.4 mg. %	7. 17-ketosteroids	5.7 mg./day
9. Serum Cl	101-103 meq./L.	8. 17-hydroxysteroids	5.0 mg./day
10. Serum Na	146-151	9. K-U/P 13 at serum K-2.5 meq.	
11. Serum K	1.6-2.5	Metabolic alkalosis with hypokalemia and tetany	
12. Serum pH	7.62		
13. CO ₂ comb. power	82 vol. %	<i>Possibilities</i>	
		1. Excessive activity of aldosterone	
		2. Specific renal tubular lesion with K loss	
Program: To have complete balance study with assays for aldosterone			

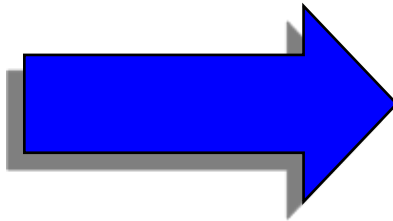
Hyperaldo primaire



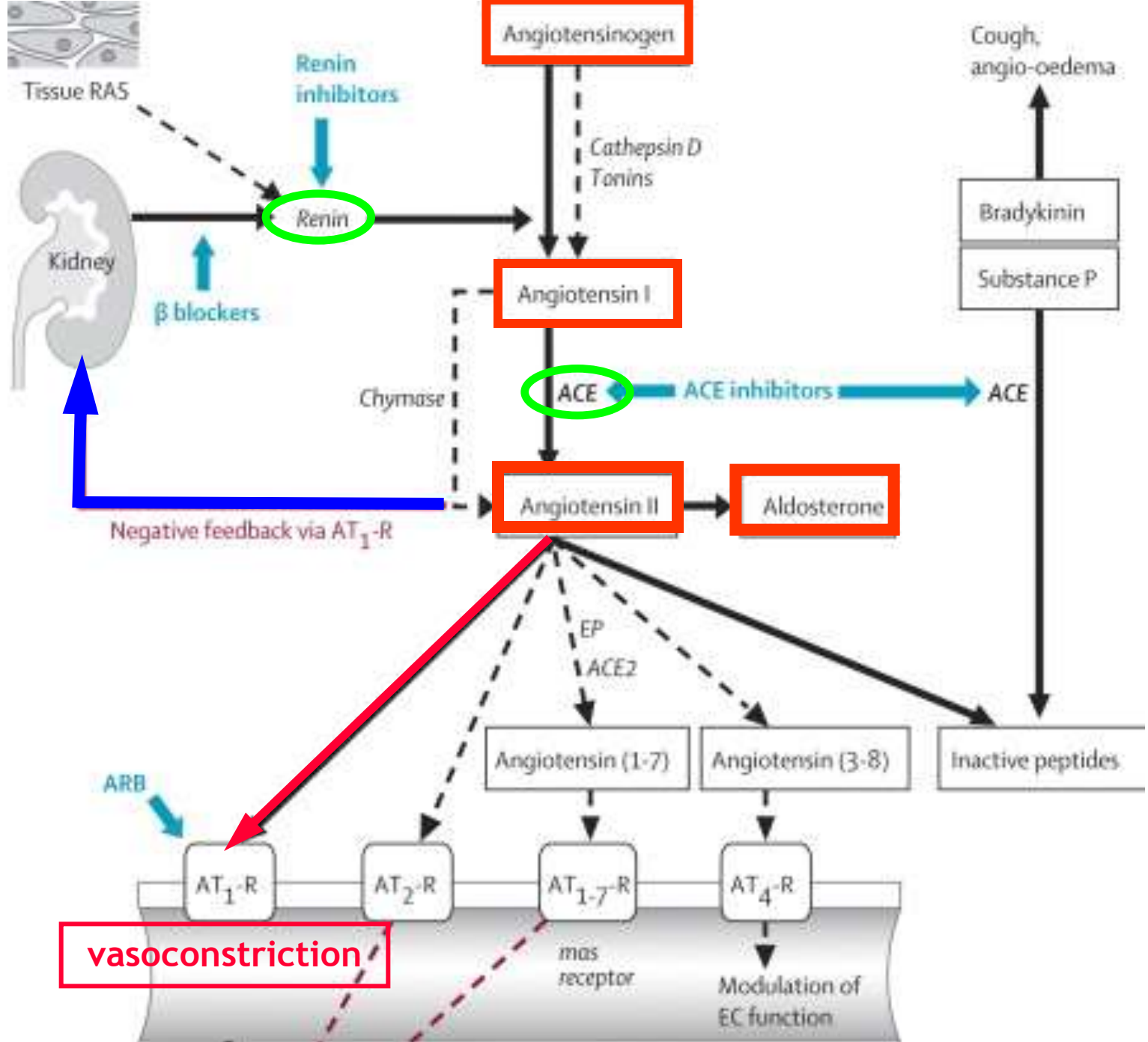
Hyperplasie
bilatérale des
surrénales

=

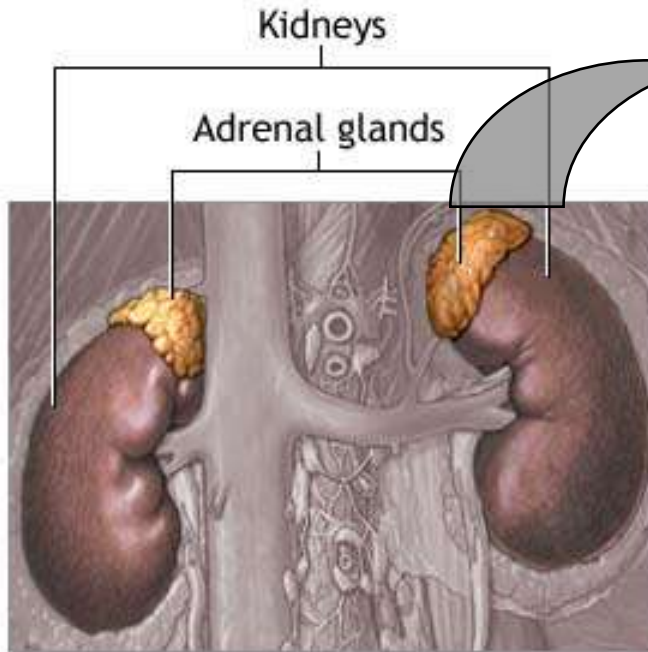
Hyperaldo
primaire
idiopathique



Spironolactone
(amiloride, eplerenone)



Glandes surrénales



ADAM.

Aldostérone

➤ Réabsorption du Na^+ ➔

➤ Pression artérielle

➤ Excrétion du K^+ ➔

hypokaliémie

➤ Excrétion de H^+ ➔
alcalose métabolique
(augmentation des
bicarbonates, HCO_3^-)

Qui dépister?

- ~~Tous les hypertendus?~~
- Patients hypertendus sélectionnés:
 - Hypokaliémie (<3.0-3.5 mmol/l)
 - Jeune (< 40-45 ans?)
 - HTA réfractaire
 - HTA secondaire

Comment dépister? Aldo/Rénine

- Normaliser la kaliémie (>3.5 mmol/l)
- Si possible sans médicaments, si nécessaire:
 - Physiotens® (moxonidine)
 - ($+\alpha_2$ central) Catapressan® (clonidine)
 - Anticalciques (diltiazem, nifedipine, amlodipine)
 - (α bloquant: $-\alpha_1$ périph) prazosine
- Prise de sang:
 - **PAC (aldo) et PRA (renine) pour rapport Aldo/Rénine**
 - Déambulation, PS assis (stimulation rénine)
 - Matin avant 9h00 (aldo max)

Comment dépister? Aldo/Rénine

Situation	Rénine	Aldostérone
Hyperaldo secondaire (hypovolémie, IC, ...)		
Hyperaldo primaire		
HTA essentielle à rénine basse		

Interprétation rapport (unités)

PAC/PRA >0.6-0.9 (nmol/l)/(ng/ml/h)

ET

Aldo PAC >0,5-0,6 nmol/l



Couché

Aldo 0.03-0.4 nmol/l

Rénine 0.2-1.6 ng/ml/h



Debout-Assis

Aldo 0.1-0.9 nmol/l

Rénine 0.8-3.7 ng/ml/h

Dépistage → Confirmation

Table 2. Test protocols of confirmatory tests used in primary aldosteronism.

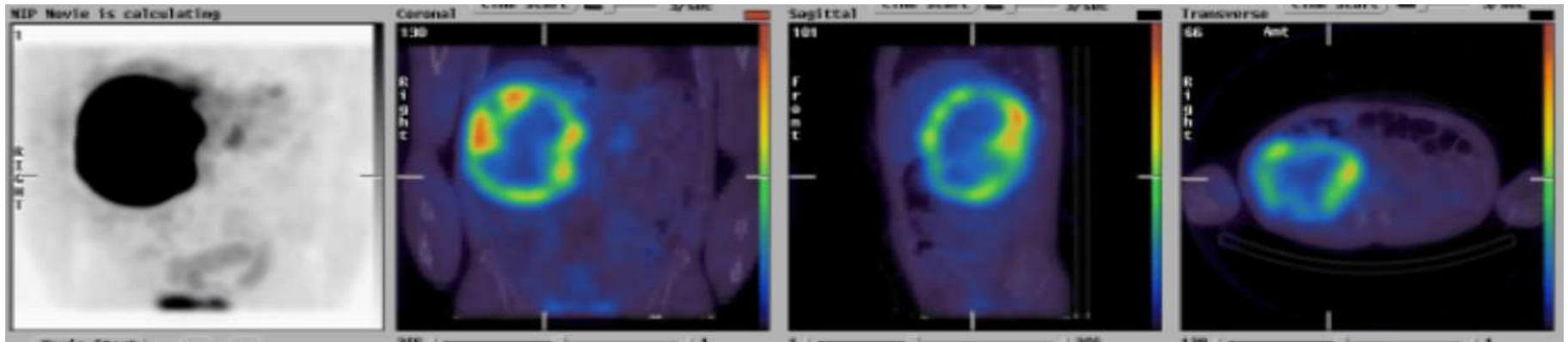
Test	Protocol	Threshold value for primary aldosteronism	Reference
Fludrocortisone suppression test	0.1 mg fludrocortisone/6 hourly and sodium supplementation (slow release natrium 30 mmol/day) over 4 days	Upright plasma aldosterone at day 5 at 10am >6 ng/dl	Stowasser et al 2001 ⁷¹
Saline infusion test	2000 ml 0.9% saline i.v. over 4 hours (while recumbent); beginning between 8am and 10am	Plasma aldosterone after saline infusion >5 ng/dl	Kem et al 1984 ⁷² , Holland et al 1984 ⁷³
Oral sodium loading	3 days of oral sodium loading (urinary sodium >200 mEq/day), collection of 24 hours urine	Urinary aldosterone after sodium loading >12 µg/day	Young 2002 ⁷⁷
Captopril test	25 mg Captopril p.o., patients in seated position	Plasma aldosterone after 2 hours >15 ng/dl	Lyons et al 1983 ⁹⁹
Urinary aldosterone metabolites	Tetrahydroaldosterone in a 24 hours urine sample	Tetrahydroaldosterone >66 µg/day	Abdelhamid et al 2003 ⁸¹
Losartan suppression test	50 mg oral losartan, patients in upright position	Decrease of plasma aldosterone after 4 hours <40%	Mantero et al 2003 ⁷⁰

i.v., intra-venous; p.o., per os/orally.

« En quatre lettres: souvent recherché, jamais trouvé »



PHÉO



Phéo = tumeur de la médulla des surrénales

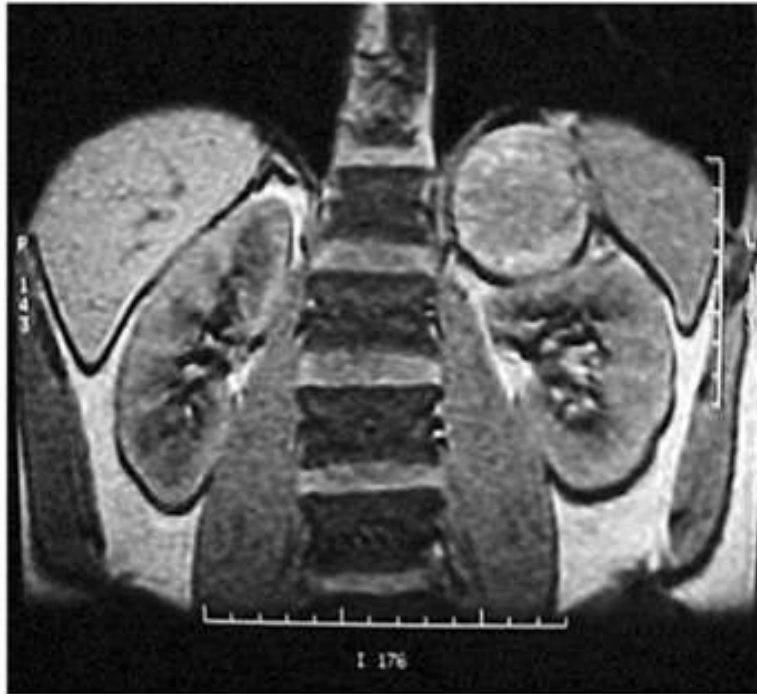
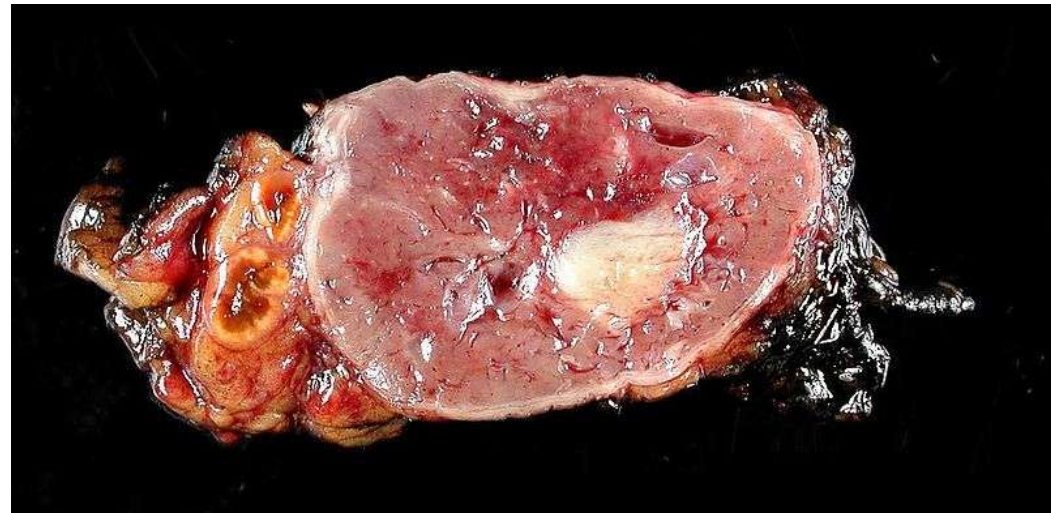
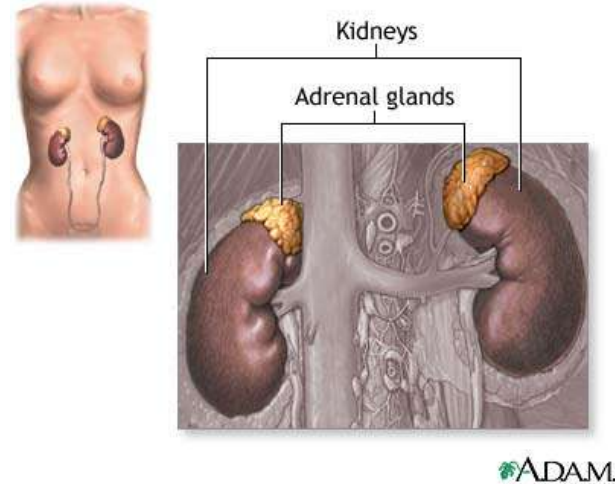
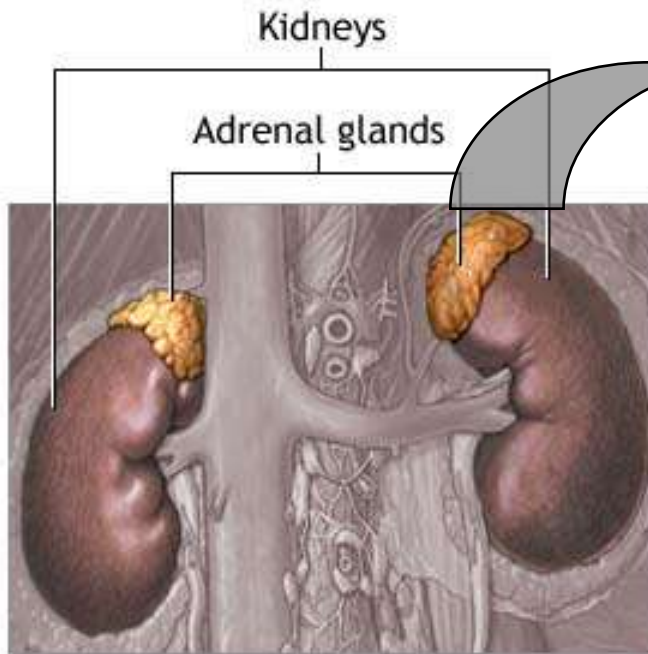


Figure 2 – MRI showing large adrenal mass - hypersignal in T2.



Phéo = tumeur de la médulla des surrénales



Catécholamines

Adrénaline (=épinéphrine),
Noradrénaline
(=norépinéphrine), dopamine



Vasoconstriction,
palpitations, HTA, sudations,
céphalées, pâleur

Quand rechercher un Phéochromocytome?

Triade
paroxystique

	Frequency
Headache	60–90%
Palpitations	50–70%
Sweating	55–75%
Pallor	40–45%
Nausea	20–40%
Flushing	10–20%
Weight loss	20–40%
Tiredness	25–40%
Psychological symptoms (anxiety, panic)	20–40%
!! Sustained hypertension	50–60%
Paroxysmal hypertension	30%
!! Orthostatic hypotension	10–50%
Hyperglycaemia	40%

Table adapted from references 17, 20, and 21. *Frequency in patients tested because of signs and symptoms.

Table 1: Frequency of signs and symptoms (%) of phaeochromocytoma*

Comment le rechercher?

Biochemical Diagnosis of Pheochromocytoma Which Test Is Best?

Jacques W. M. Lenders, MD, PhD

Karel Pacak, MD, PhD

McClellan M. Walther, MD

W. Marston Linchan, MD

Massimo Mannelli, MD

Peter Friberg, MD, PhD

Harry R. Keiser, MD

David S. Goldstein, MD, PhD

Graeme Eisenhofer, PhD

Context Diagnosis of pheochromocytoma depends on biochemical evidence of catecholamine production by the tumor. However, the best test to establish the diagnosis has not been determined.

Objective To determine the biochemical test or combination of tests that provides the best method for diagnosis of pheochromocytoma.

Design, Setting, and Participants Multicenter cohort study of patients tested for pheochromocytoma at 4 referral centers between 1994 and 2001. The analysis included 214 patients in whom the diagnosis of pheochromocytoma was confirmed and 644 patients who were determined to not have the tumor.

Main Outcome Measures Test sensitivity and specificity, receiver operating characteristic curves, and positive and negative predictive values at different pretest prevalences using plasma free metanephrines, plasma catecholamines, urinary catechol-

Conclusion Plasma free metanephrines provide the best test for excluding or confirming pheochromocytoma and should be the test of first choice for diagnosis of the tumor.

JAMA. 2002;287:1427-1434

www.jama.com

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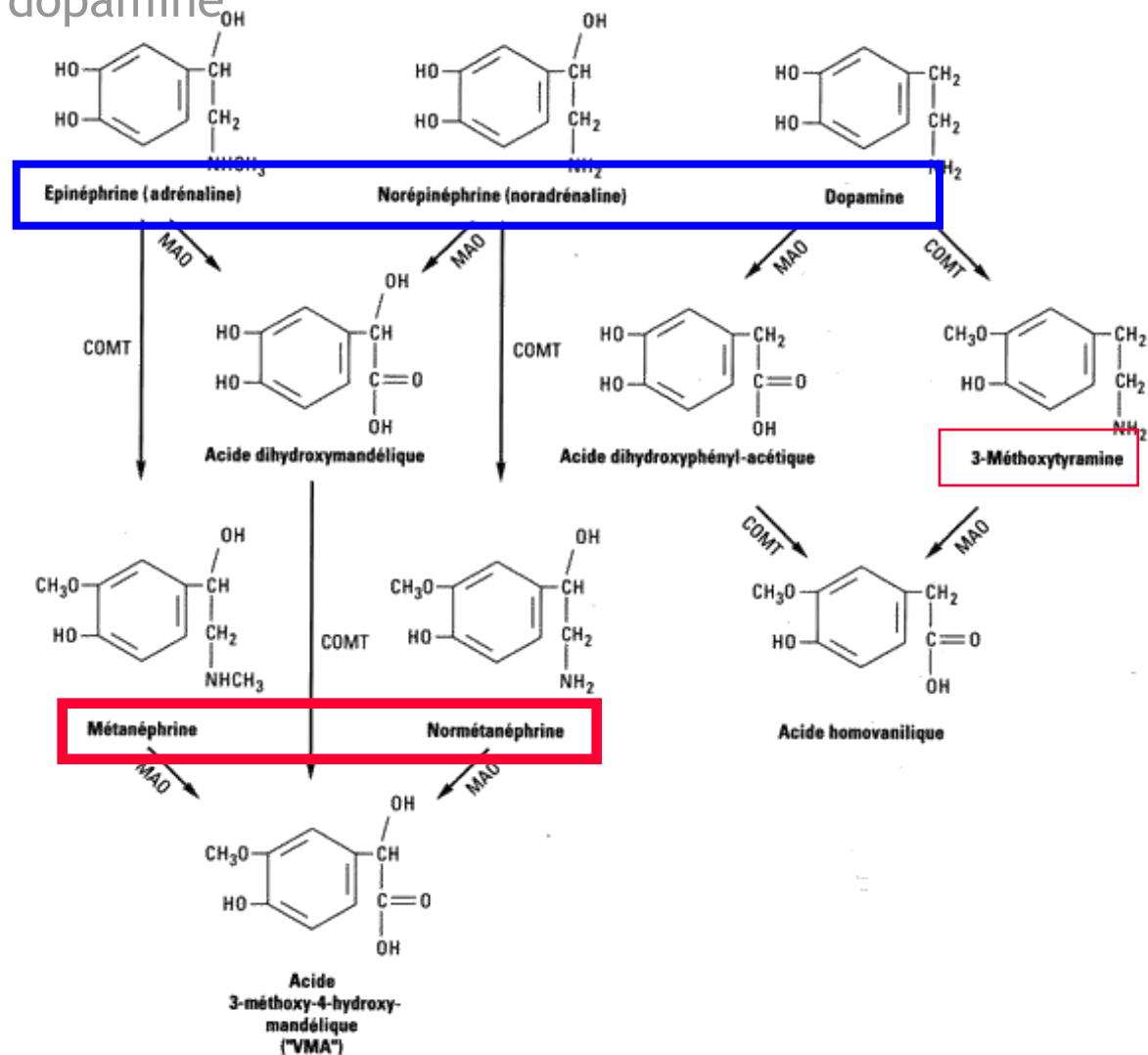
and Blood Institute (Dr Keiser), and Clinical Neurocardiology Section, National Institute of Neurological Disorders and Stroke (Drs Goldstein and Eisenhofer), National Institutes of Health, Bethesda, Md.

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Toward Optimal Laboratory Use Section Editor: David H. Mark, MD, MPH, Contributing Editor.

Catécholamines → Métanéphrines

- La Normétanéphrine ← noradrénaline
- La Métanéphrine ← adrénaline
- La 3-méthoxytyramine ← dopamine



Tests biochimiques proposés

- Métanéphrines libres plasmatiques (CHUV)
 - Normétanéphrine
 - Métanéphrine
 - 3-méthoxytyramine
- Abandonner:
 - catécholamines plasmatiques
 - VMA urinaire
 - Récoltes urinaires de 24h pour catécholamines ou métanéphrines

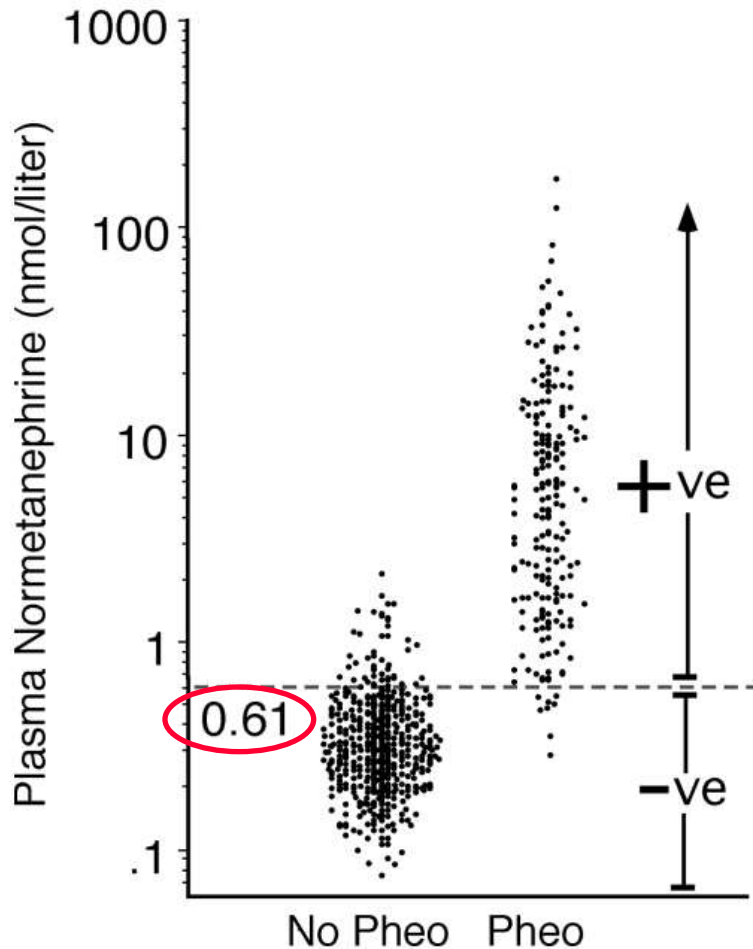
Interprétation des résultats

	Presence of phaeochromocytoma		
	Unlikely*	Possible	Likely†
Urine tests			
Catecholamines (HPLC)			
Norepinephrine (nmol/24 h)	<500	500–1180	>1180
Epinephrine (nmol/24 h)	<100	100–170	>170
Fractionated metanephrines (HPLC)			
Normetanephrine (nmol/24 h)	<3000	3000–6550	>6550
Metanephrine (nmol/24 h)	<1000	1000–2880	>2880
Total metanephrines (spectrophotometry)			
Total of normetanephrine and metanephrine (μmol/24 h)	<6	6–12.7	>12.7
VMA (spectrophotometry)			
VMA (μmol/24 h)	<40	40–55	>55
Blood tests			
Catecholamines (HPLC)			
Noradrenaline (nmol/L)	<3.00	3.00–7.70	>7.70
Adrenaline (nmol/L)	<0.45	0.45–1.20	>1.20
Free metanephrines (HPLC)			
Normetanephrine (nmol/L)	<0.60	0.60–1.40	>1.40
Metanephrine (nmol/L)	<0.30	0.30–0.42	>0.42

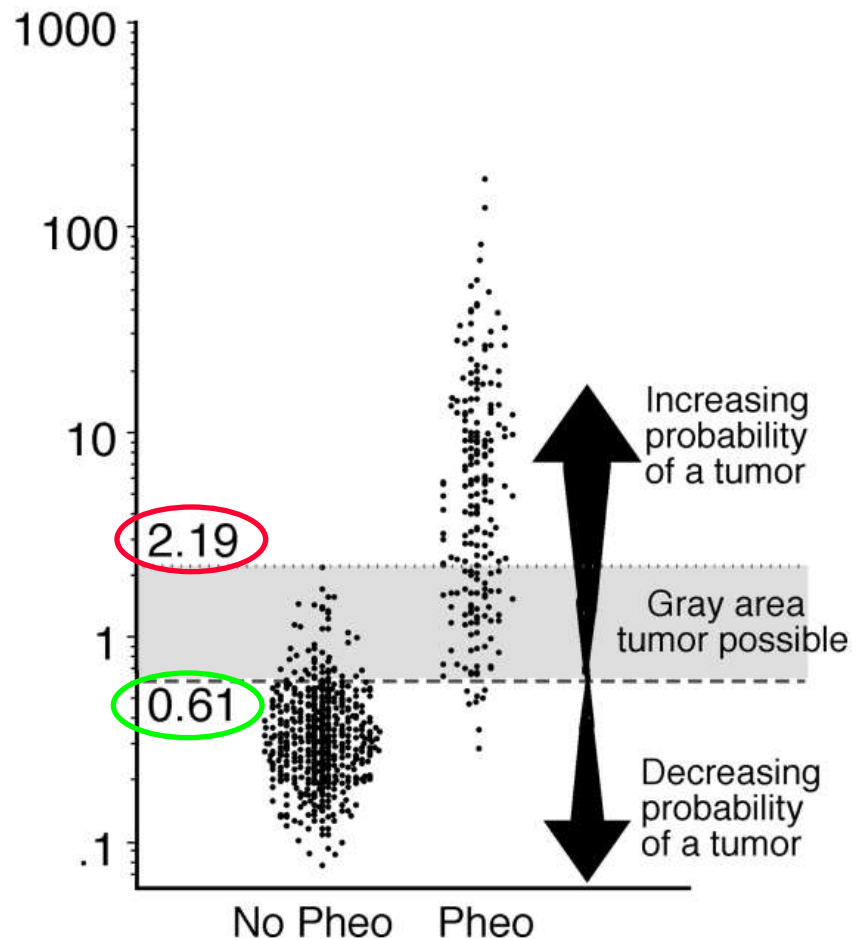
HPLC=high-pressure liquid chromatography. Unlikely=number of true-negative results>>number of false-negative results; possible=false-positive results>true-positive results; likely=true-positive results>>false-positive results. *Cut-off points represent upper reference limits used for estimation of sensitivity and specificity in table 3; therefore, the negative predictive value (ie, likelihood that phaeochromocytoma is not present) varies for every test, depending on differences in the test characteristics in table 3. †Cut-off points are calculated from 99th percentile in a reference group of 644 patients who had no phaeochromocytoma. Table adapted from reference 64.

Interprétation des résultats

Binary Approach



Continuous Approach



Pièges diagnostiques

- Médicaments !

SSRI = Selective Serotonin Reuptake Inhibitors

SNaRI = Selective Noradrenergic Reuptake Inhibitors

NaSSA = Noradrenergic and Specific Serotonergic Antidepressant

NaRI = Noradrenergic Reuptake Inhibitor

Nature of interference	
Analytical methods	
Coffee (including decaffeinated coffee)	HPLC assays: plasma catecholamines
Labetalol	Spectrophotometric and fluorometric assays: urinary catecholamines and metanephrines;
Sotalol	HPLC assays: plasma catecholamines
Buspirone	HPLC assays: urinary metanephrines
Paracetamol	HPLC assays: plasma-free metanephrines
Levodopa	HPLC assays: catecholamines and metabolites
α -methyldopa	HPLC assays: catecholamines
Sympathomimetics (eg, amfetamines, ephedrine)	Spectrophotometric and fluorometric assays: plasma and urinary catecholamines
Pharmacodynamic or pharmacokinetic interference	
Tricyclic antidepressants	Blocks norepinephrine reuptake, causing rises in plasma and urinary norepinephrine, normetanephrine, and VMA
SSRI, SNaRI, NaSSA NaRI	
Phenoxybenzamine	Blocks presynaptic α_2 adrenoceptors, causing increases in plasma and urinary norepinephrine, normetanephrine, and VMA
Monoamine oxidase inhibitors	Blocks deamination, causing up to five-fold increases in plasma and urinary metanephrines
Levodopa	Metabolised by enzymes that also convert catecholamines
α -methyldopa	Metabolised by enzymes that also convert catecholamines
Stimulants (eg, caffeine, nicotine)	Increased plasma and urinary catecholamines
Sympathomimetics (eg, amfetamines, ephedrine)	Increased plasma and urinary catecholamines
Calcium-channel blockers (dihydropyridines)	Increased plasma catecholamines due to sympathetic activation

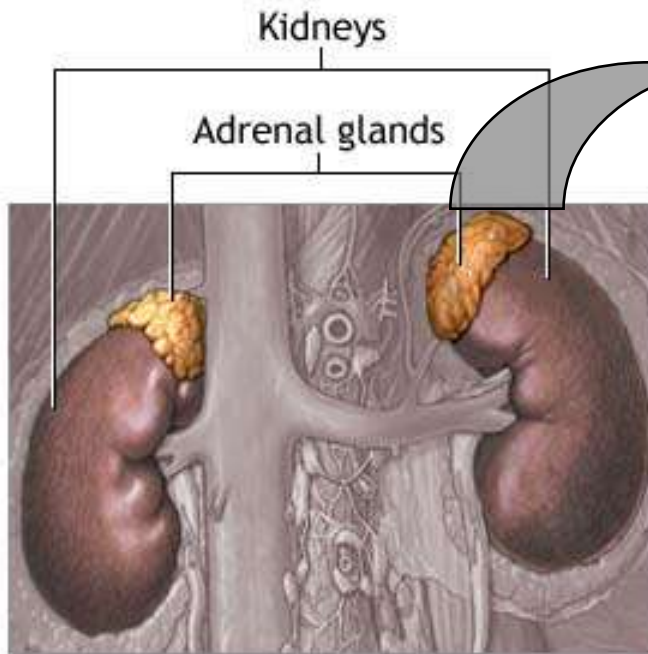
HPLC=high-pressure liquid chromatography.

- Situations de stress (Soins intensifs)
- Apnées du sommeil obstructives !!

HTA et Cushing



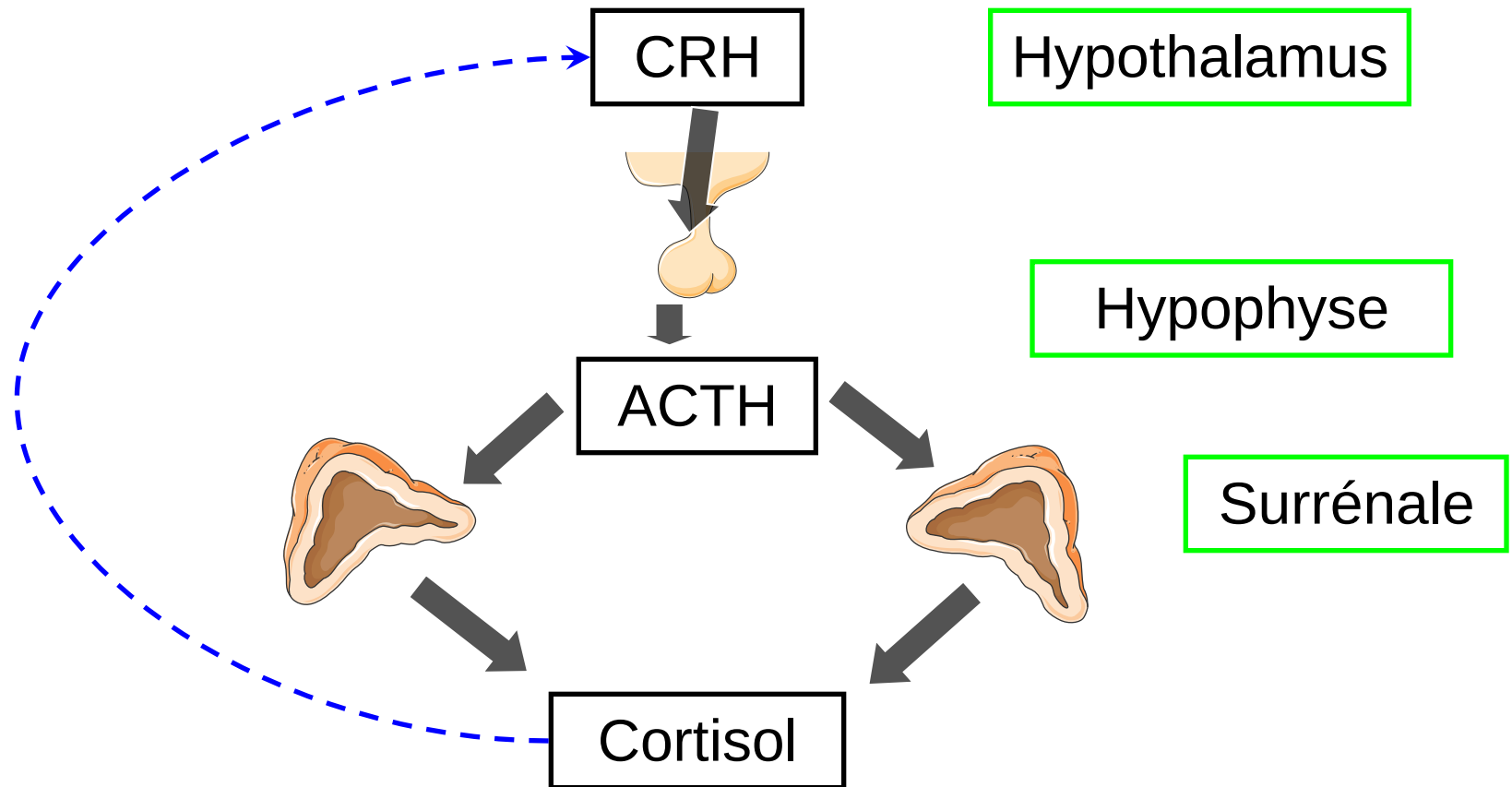
Cushing: production par la surrénale d'un excès de cortisol (ACTH dép ou ACTH indép)



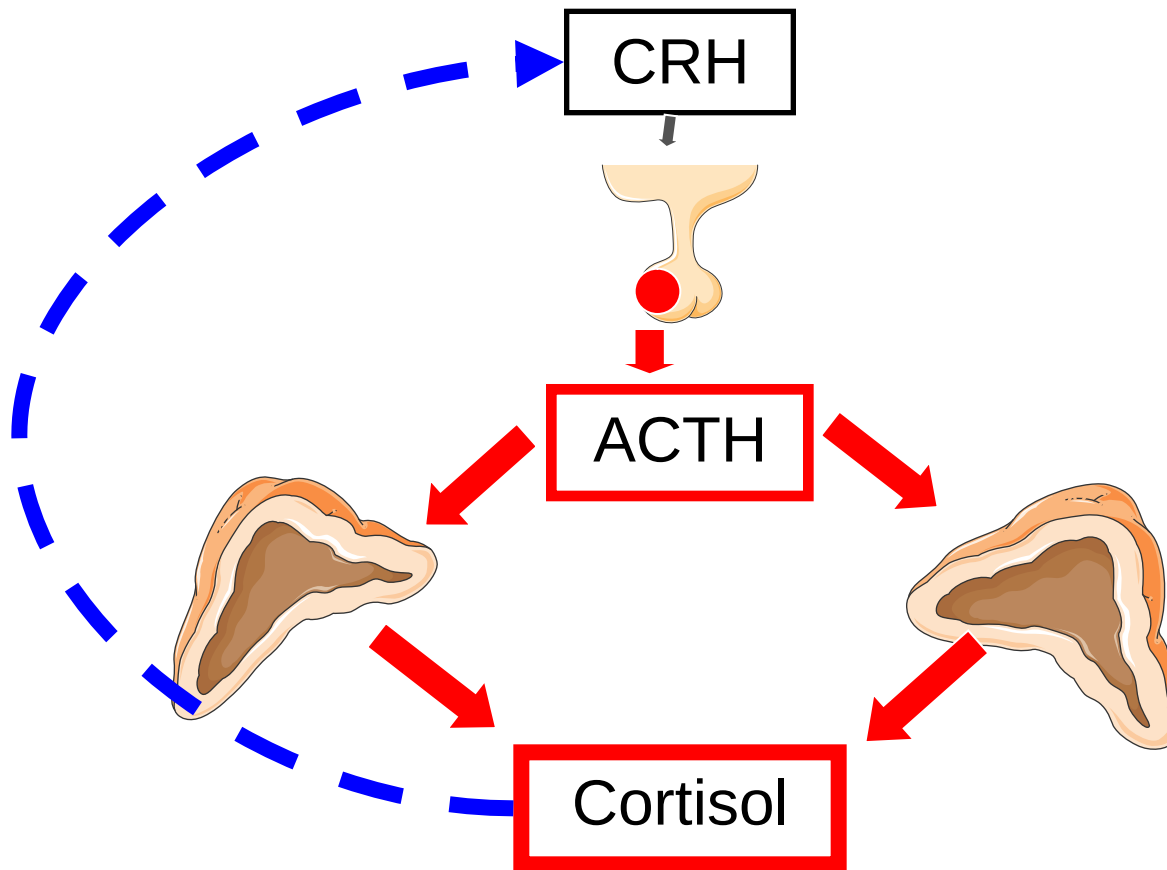
Cortisol

Antiinflammatoire, stress,
immunité

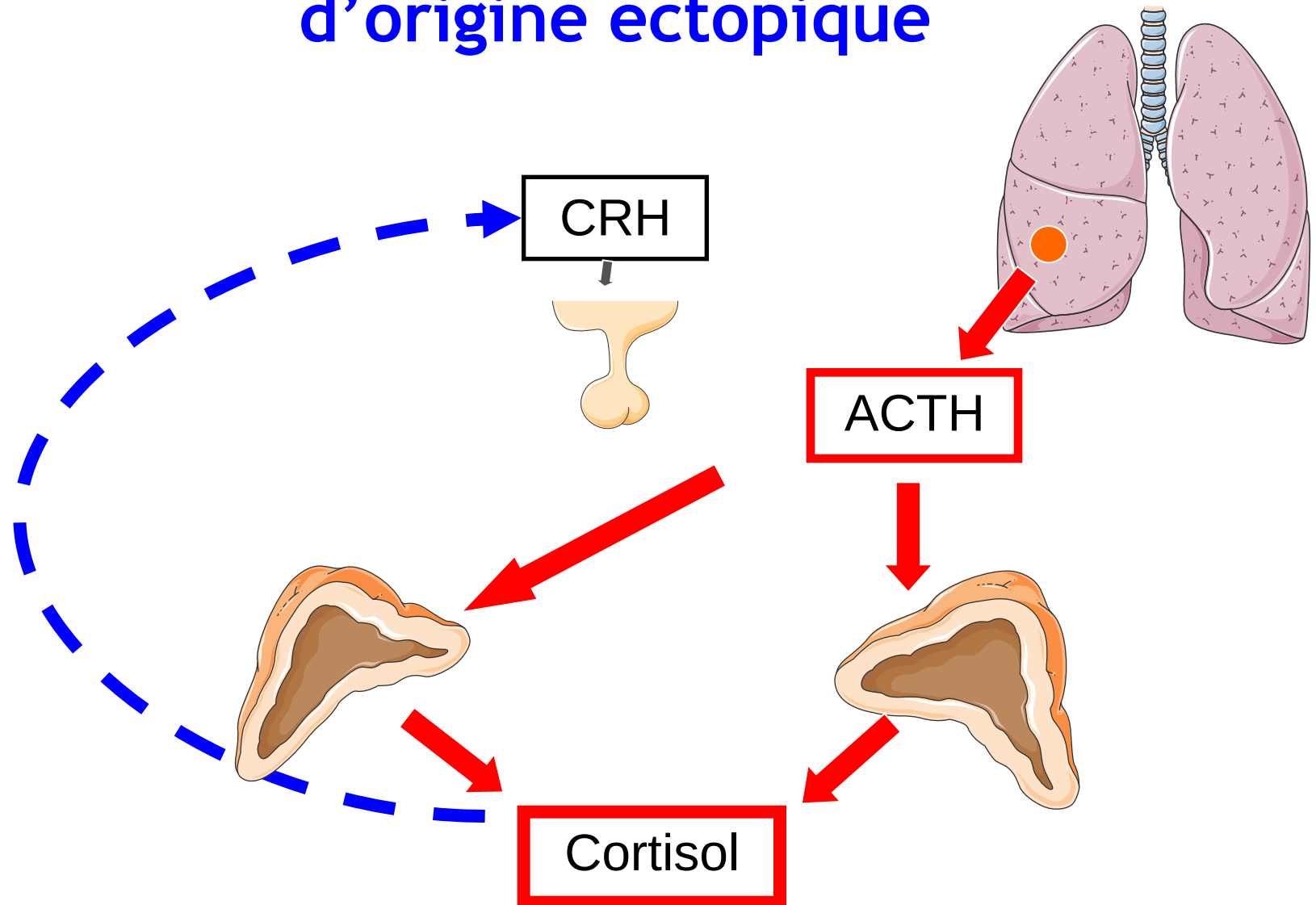
Physiologie normale de l'axe hypothalamo-hypophysaire-surrénalien (HHS)



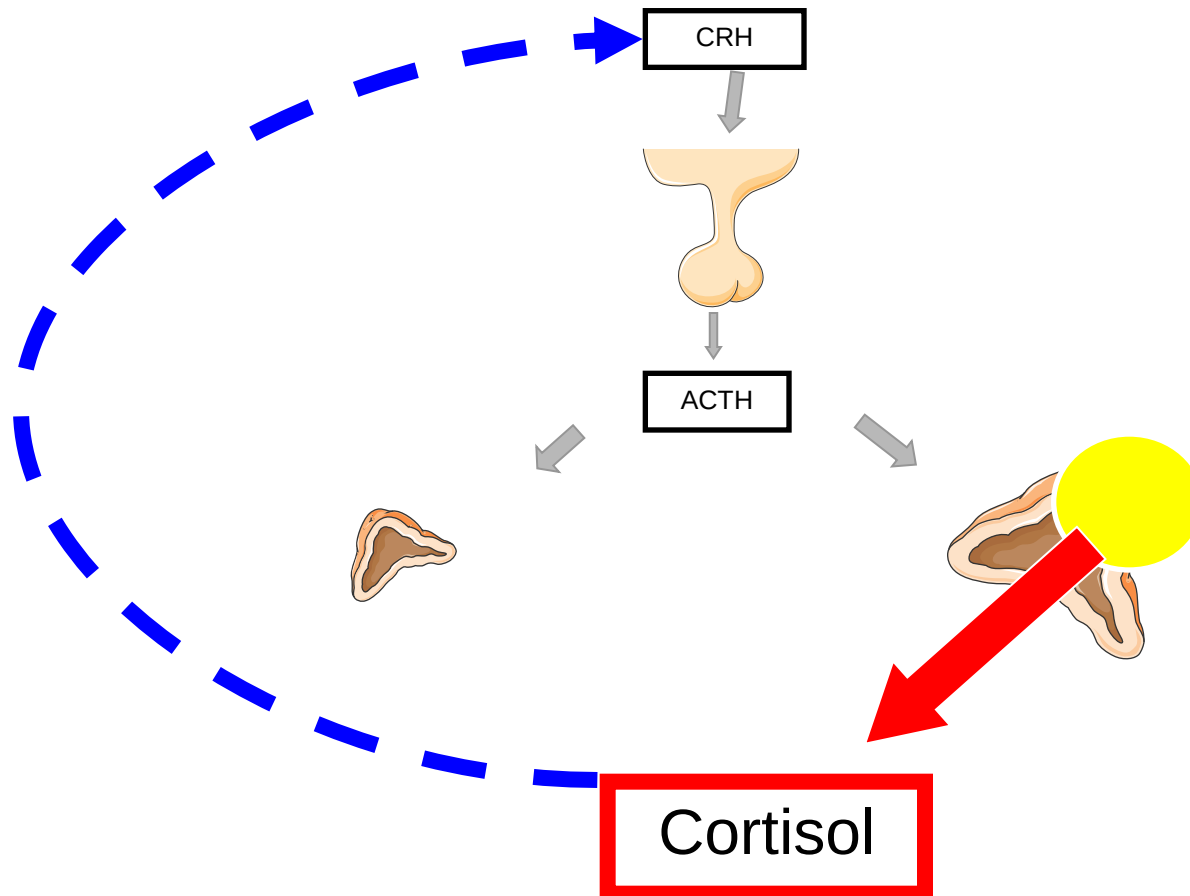
Maladie de Cushing (Adénome hypophysaire à ACTH)



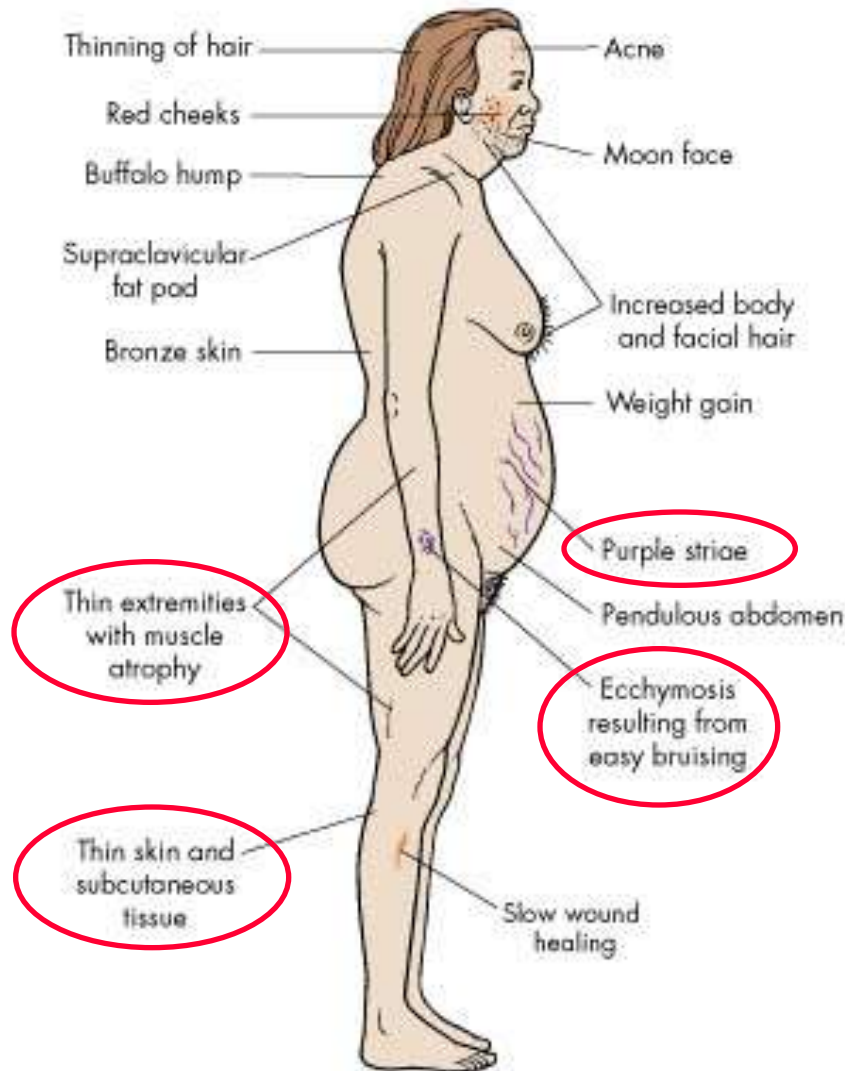
Syndrome de Cushing d'origine ectopique



Cushing d'origine surrénalienne (tumeur surrénalienne unilatérale)



Quand rechercher un Cushing?



Dynamique
(voir anciennes
photos!)

Troubles psy
Ostéoporose
HTA
DM
Infections

Cushing?



Figure 14-14. Minnie G., Cushing's index patient, at age 23 years. (From Cushing H. The basophil adenomas of the pituitary body and their clinical manifestations [pituitary basophilism]. Bull Johns Hopkins Hosp 1932;50:137-195.)

(From Cushing H. The basophil adenomas of the pituitary body and their clinical manifestations [pituitary basophilism]. Bull Johns Hopkins Hosp 1932;50:137-195.)

Cushing?



Cushing?



Comment le rechercher?

1. Estimer probabilité clinique
2. Etablir l'hypersécrétion autonome de cortisol
 - Test de suppression court 1mg Dex (overnight)
 - 23h00 1mg Dexaméthasone
 - 8h00 Cortisol plasmatique (<50 nmol/l = freination adéquate)
 - Cortisolurie de 24h (2x) (avec la créat)
 - Cortisol salivaire 23h00
 - Choix du laboratoire (HUG)
3. ACTH dépendant/indépendant
4. Recherche cause (IRM hypophysaire/CT surrénales)



View from Tate Modern gallery, London Jan 2006