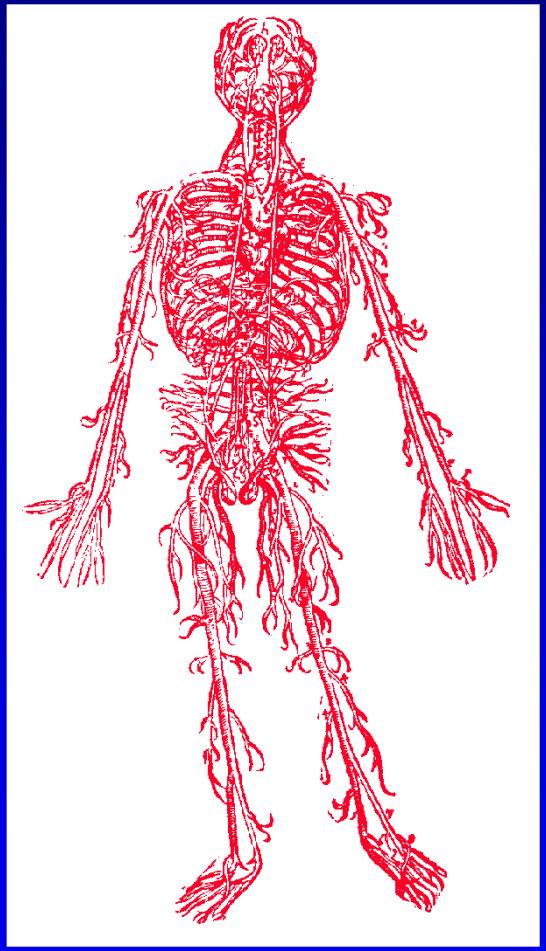
Les gros vaisseaux chez l'hypertendu

Pr Daniel Herpin

Diapos des

Professeurs Asmar et Boutouyrie

HETEROGENEITE ENTRE LES ARTERES



- Anatomie
- Histologie
- Physiologie
- Pathologie

CLASSIFICATION ANATOMIQUE

Selon le diamètre interne de l'artère :

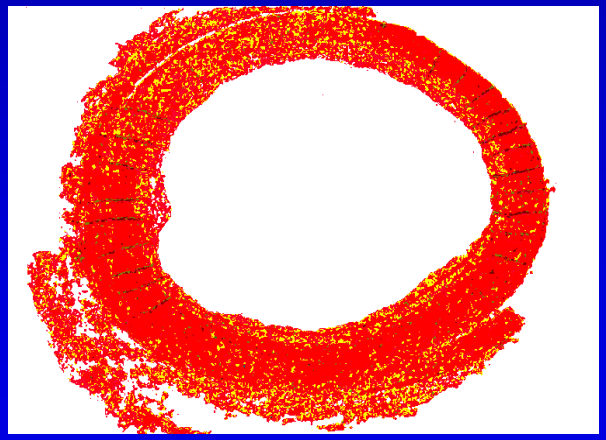
- Le système proximal : artères de grand et moyen calibre (> 2 mm)
- Le système distal : petites artères et artérioles (< 2 mm)
- La microcirculation : vaisseaux capillaires (< 10 µm)

CLASSIFICATION HISTOLOGIQUE

Selon la composante prédominante de la media

Artère élastique

(grande)



Artère musculaire

(petite)

CLASSIFICATION PHYSIOLOGIQUE

- Système Proximal
  - Fonctions conduit et amortissement
- Système Distal
  - Fonction de distribution
- Microcirculation
  - Fonctions nutritive et/ou métabolique

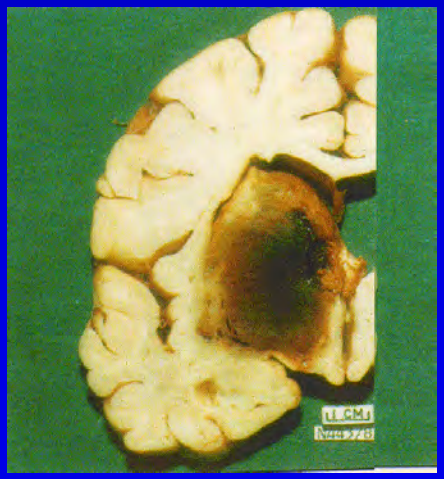
RELATION ENTRE STRUCTURE ET FONCTION

ANATOMIE	HISTOLOGIE	PHYSIOLOGIE	EVALUATION HEMODYNAMIQUE
Grande	Elastique	Conduit & Amortissement	Compliance & Distensibilité
Petite	Musculaire	Distribution	Résistance

HETEROGENEITE PATHOLOGIQUE

- La localisation des lésions peut varier selon le facteur de risque.
- Les lésions peuvent varier selon leur sites:
  - **Carotide ⇒ sténose**
  - **Aorte ⇒ anévrisme**
- Sur un même site, les lésions peuvent varier selon la structure endommagée :
  - Artères cérébrales : lacune, sténoses - thrombose, hémorragie

Hémorragie



Anévrysme aortique

HYPERTENSION

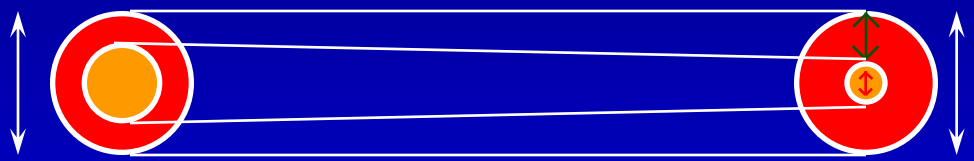
&

SYSTEME ARTERIEL

SYSTEME ARTERIEL ET HYPERTENSION

Remodelage artériel

- ***Petites Artères***

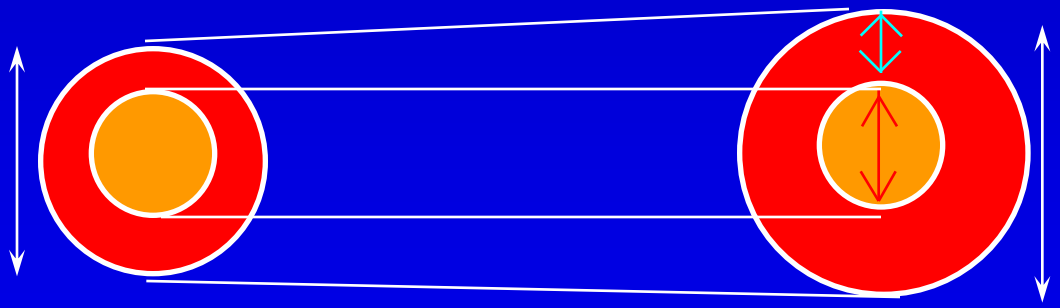


Epaisseur ↗
Diamètre intérieur ↘

Paroi / Lumière ↗

Surface cross-sectionelle ≈ (or ↗)

- ***Grandes Artères***



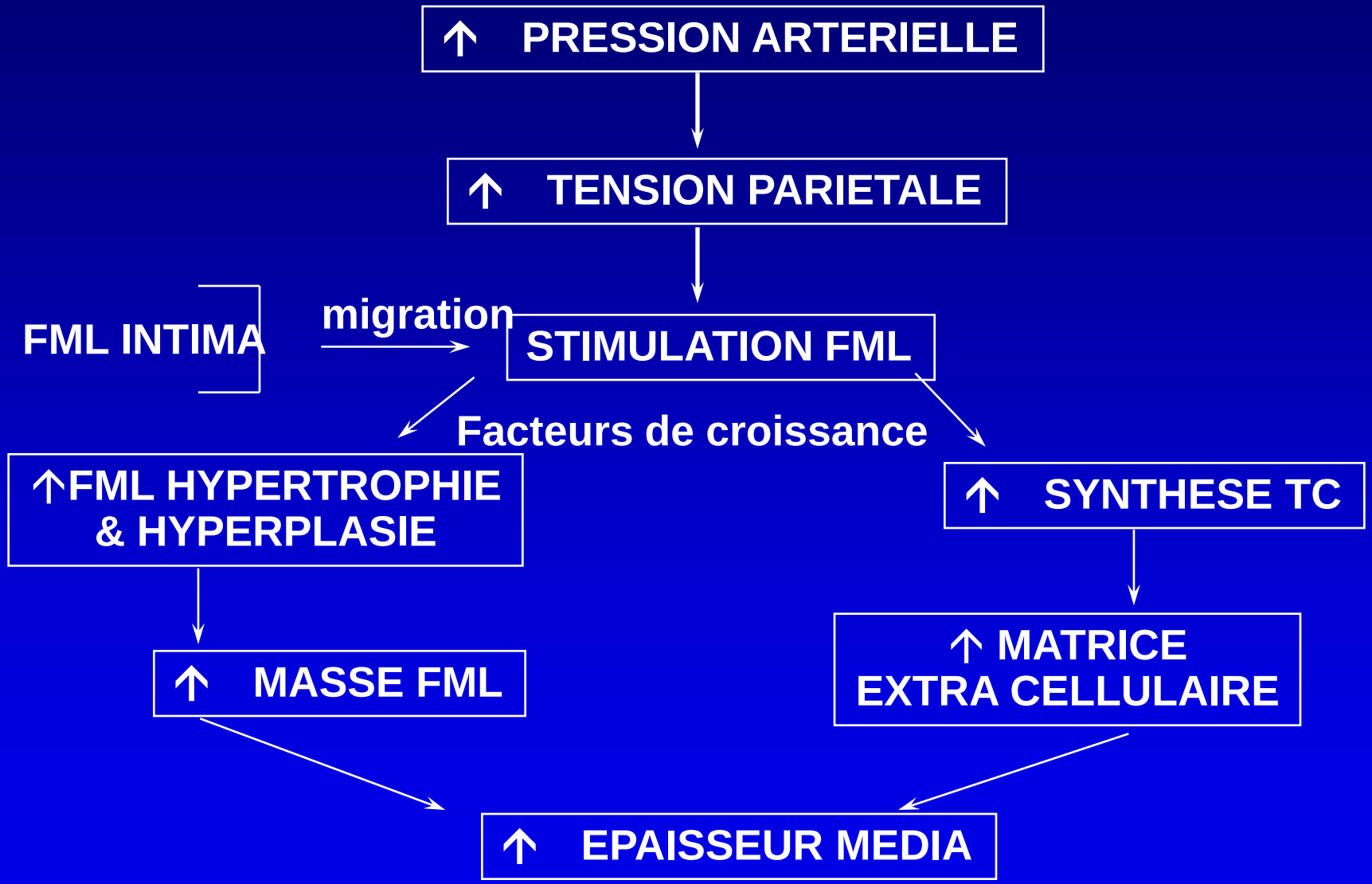
Epaisseur ↗
Diamètre intérieur ≈ (or ↗)

Paroi / Lumière ↗

Surface cross-sectionelle ↗

SYSTEME ARTERIEL ET HYPERTENSION

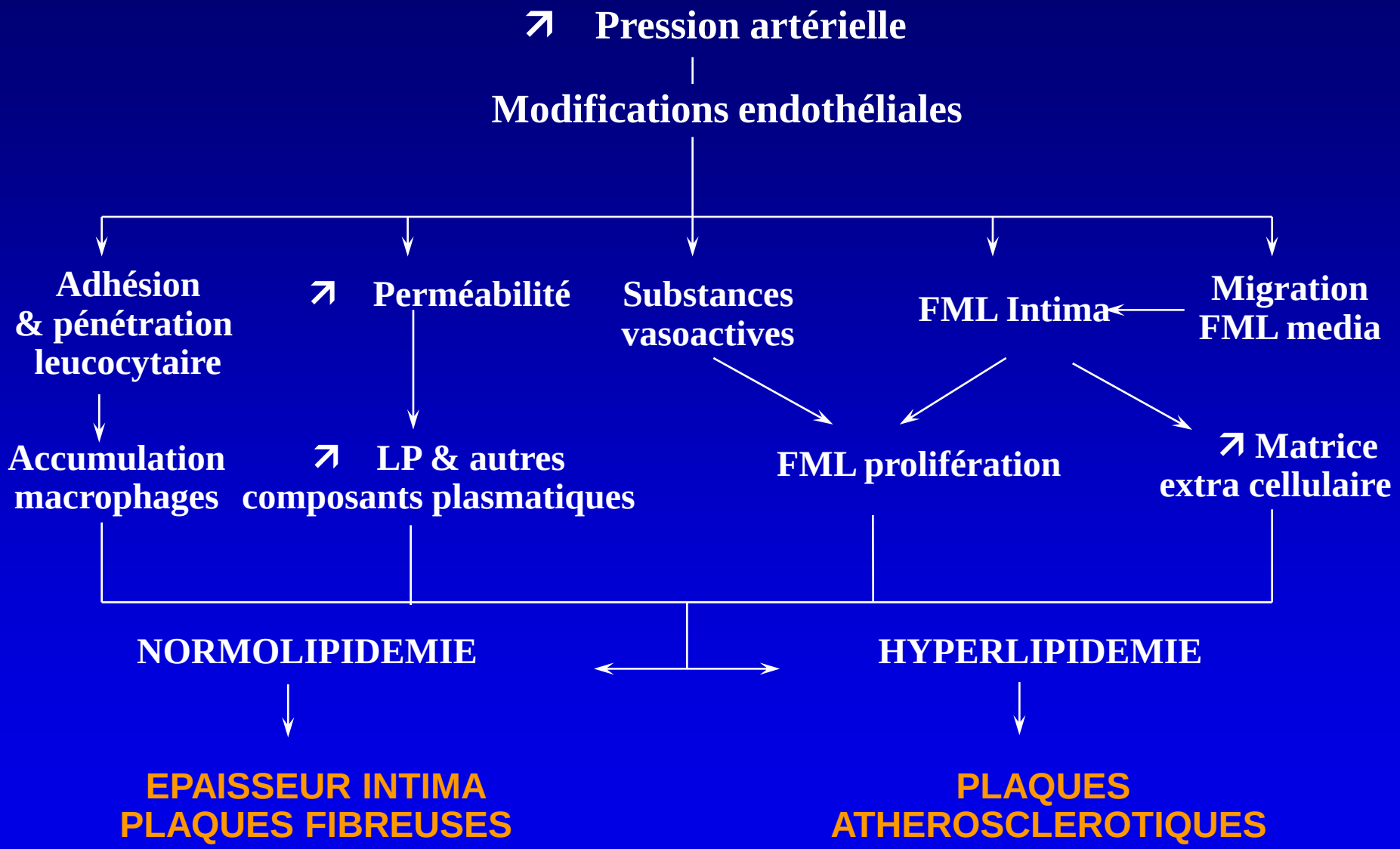
Effets de la pression artérielle sur la media



Chobanian et al. Hypertens, 1990

SYSTEME ARTERIEL ET HYPERTENSION

Effets sur l'Intima

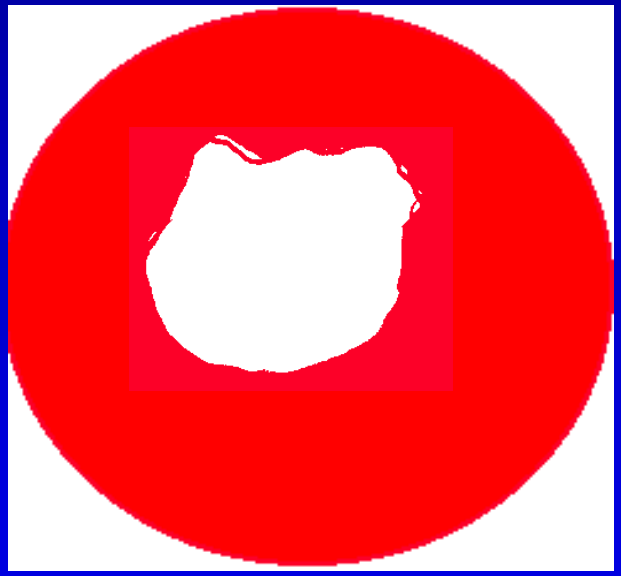


Chobanian et al. Hypertens, 1990

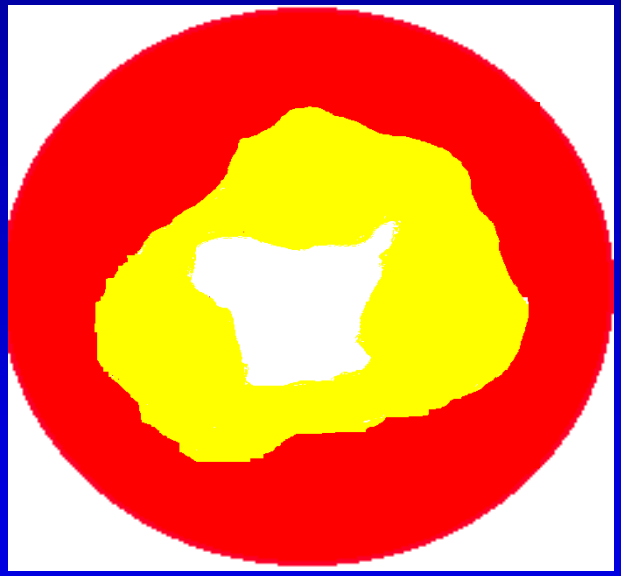
SYSTEME ARTERIEL ET HYPERTENSION

Les altérations artérielles facilitent la progression de :

Artériosclérose

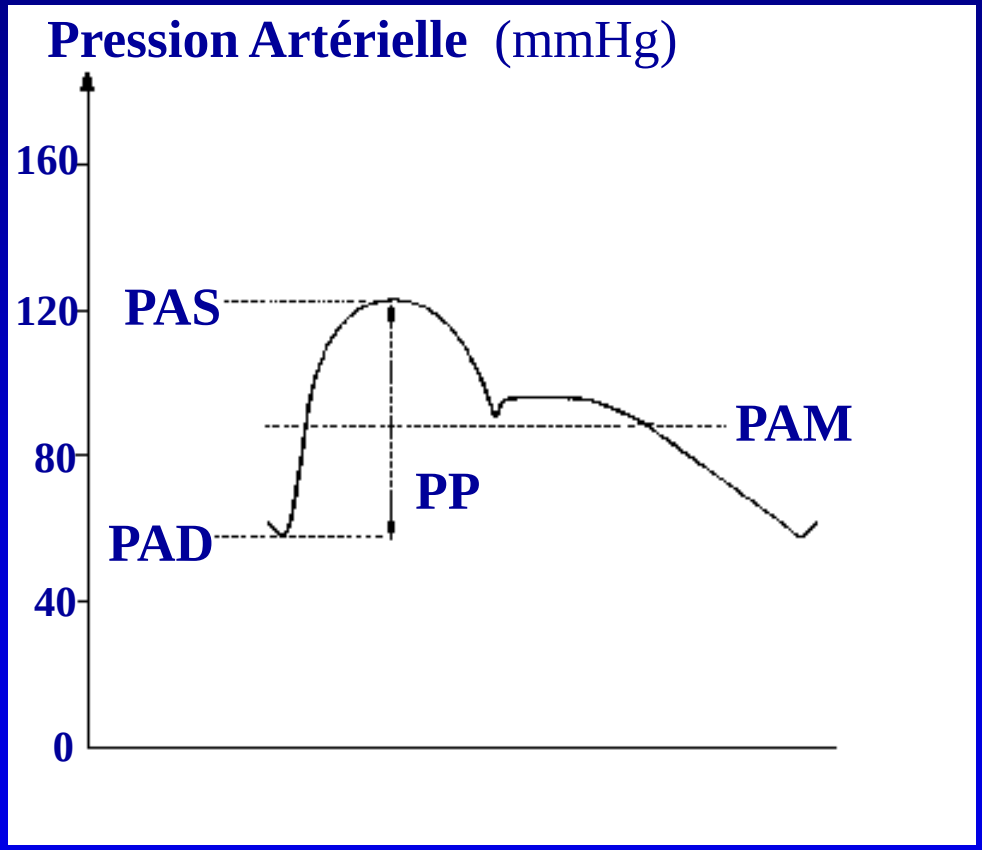


Athérosclérose



EVALUATION ARTERIELLE

Onde de pression



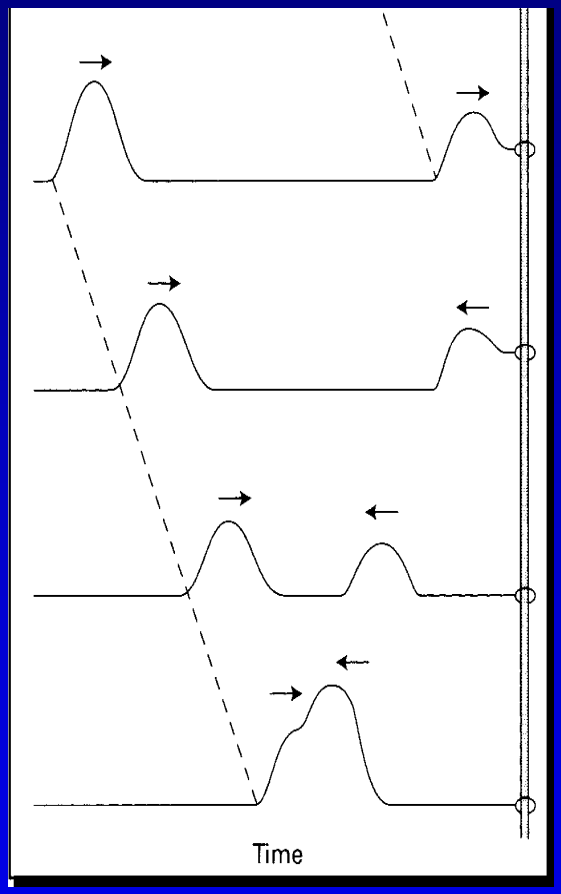
Déterminants de la PA

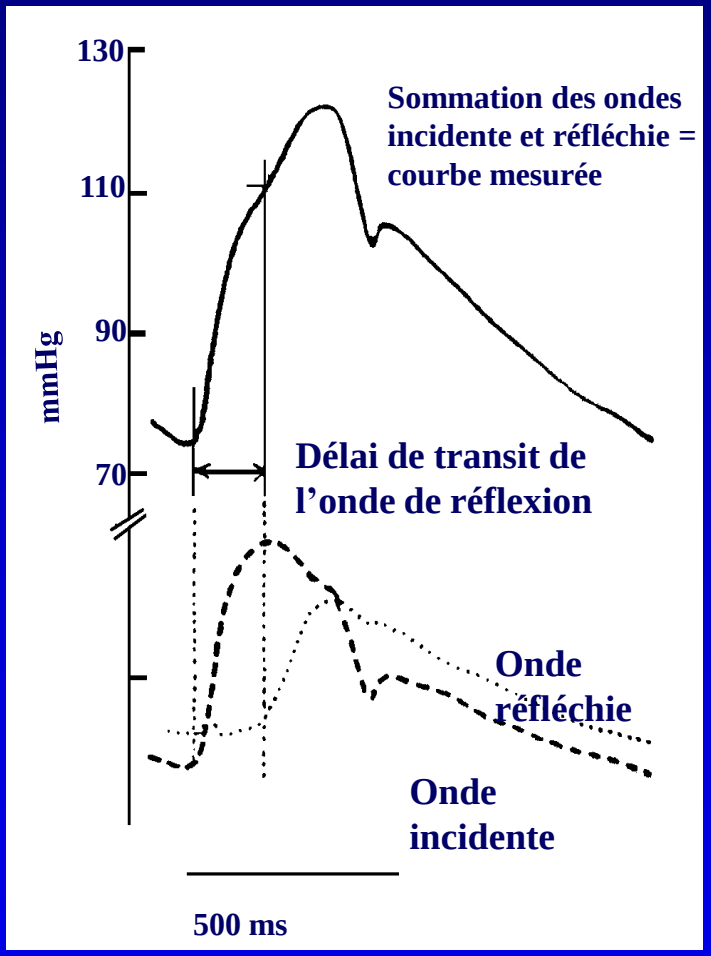
PA systolique	PA diastolique
• Éjection ventricule gauche ++ • **Compliance des gros troncs artériels** +++ • Ondes de réflexion ++ • Résistances périphériques +	■ **Résistances périphériques des petites artères** +++ ■ **Rigidité de la paroi des gros troncs artériels** +

Déterminants de la PA

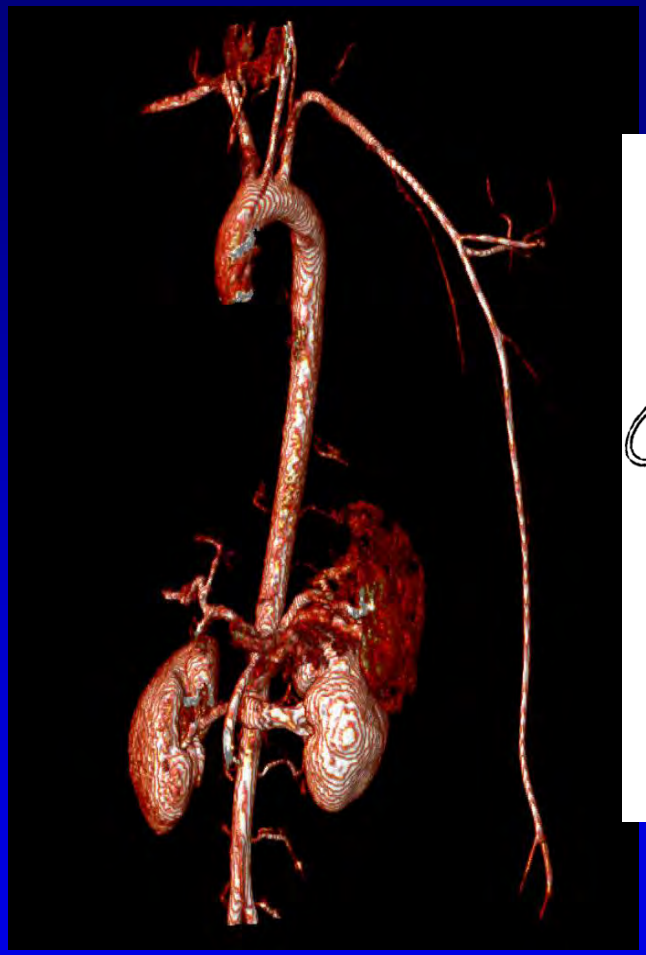
PA moyenne	PA pulsée	
• Débit cardiaque • Résistances périphériques **PAM =** **débit cardiaque** **x résistances périphériques**	■ **Rigidité artérielle** ■ **Ondes de réflexion** ■ **Résistances périphériques**	**+++** **++** **+**

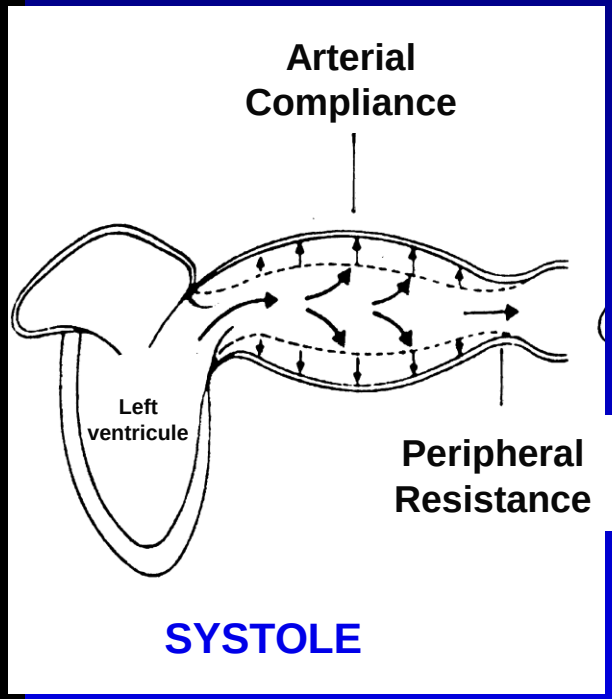
Décomposition de l'onde de pression

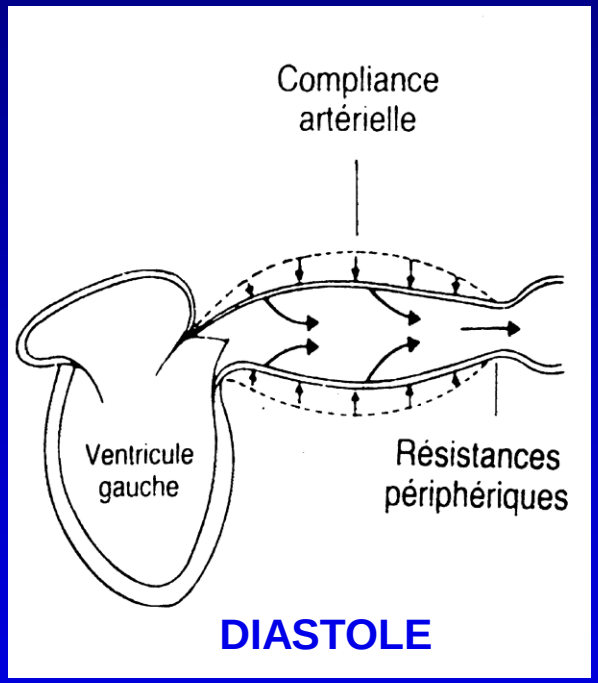




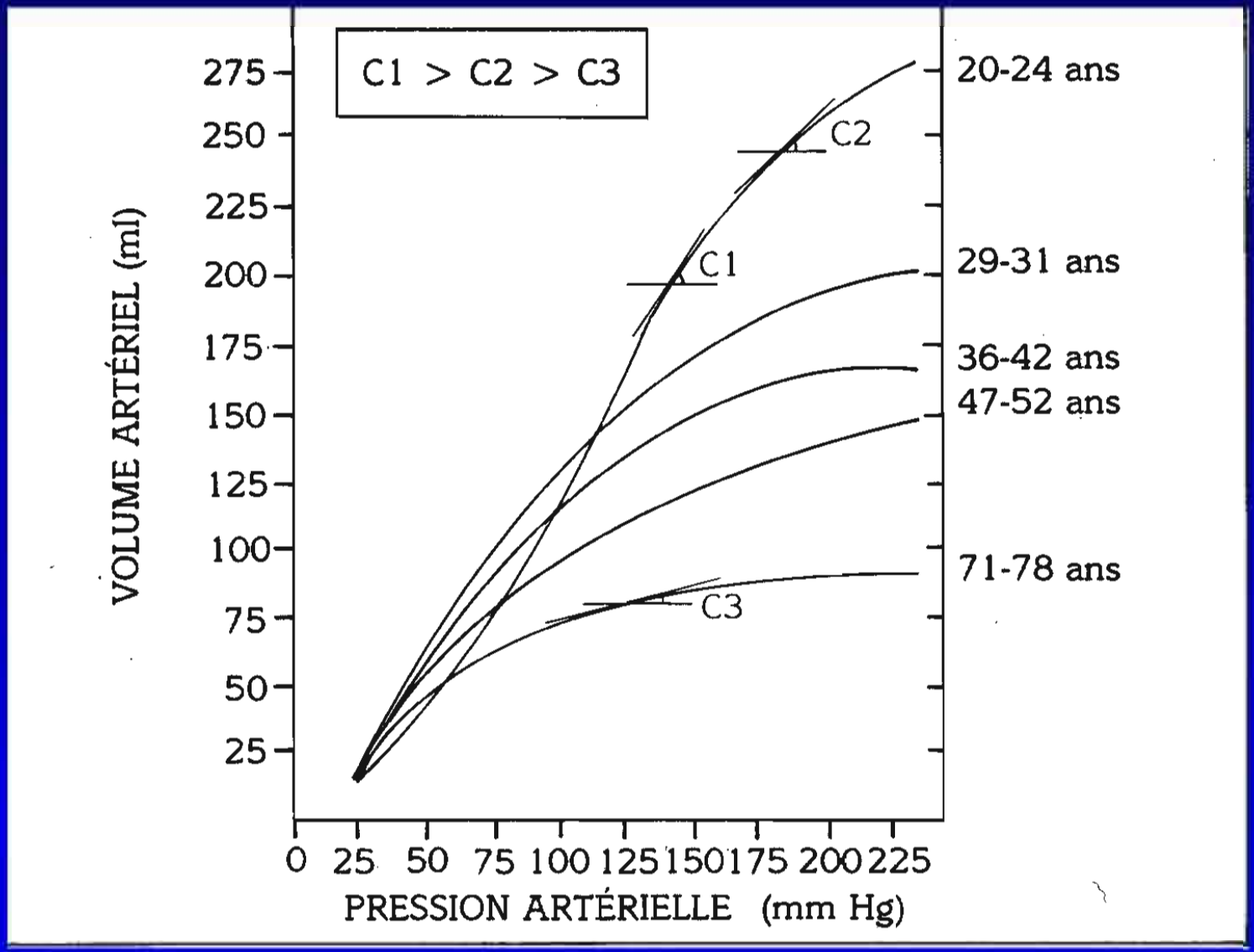
ARTERIAL COMPLIANCE

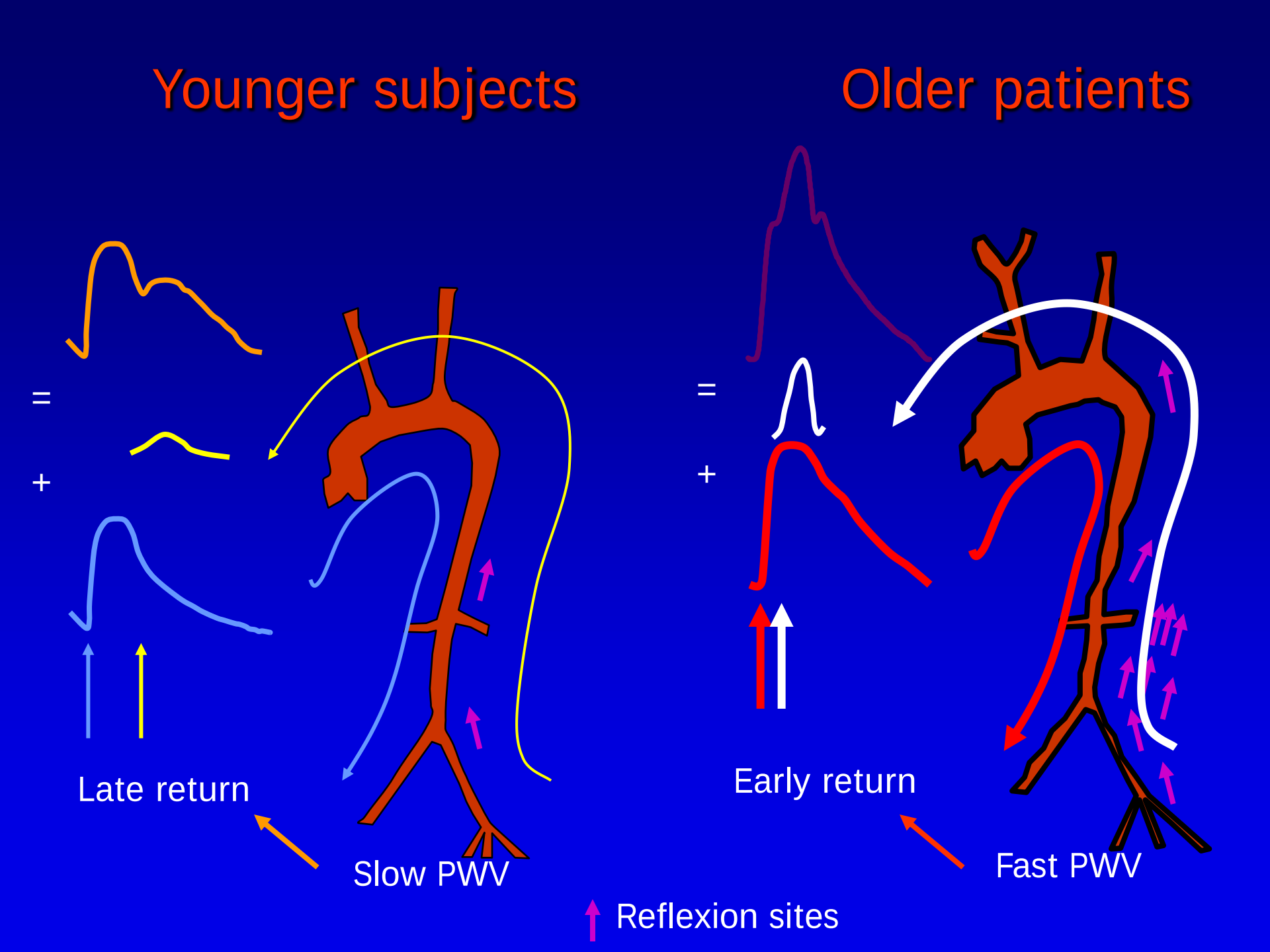






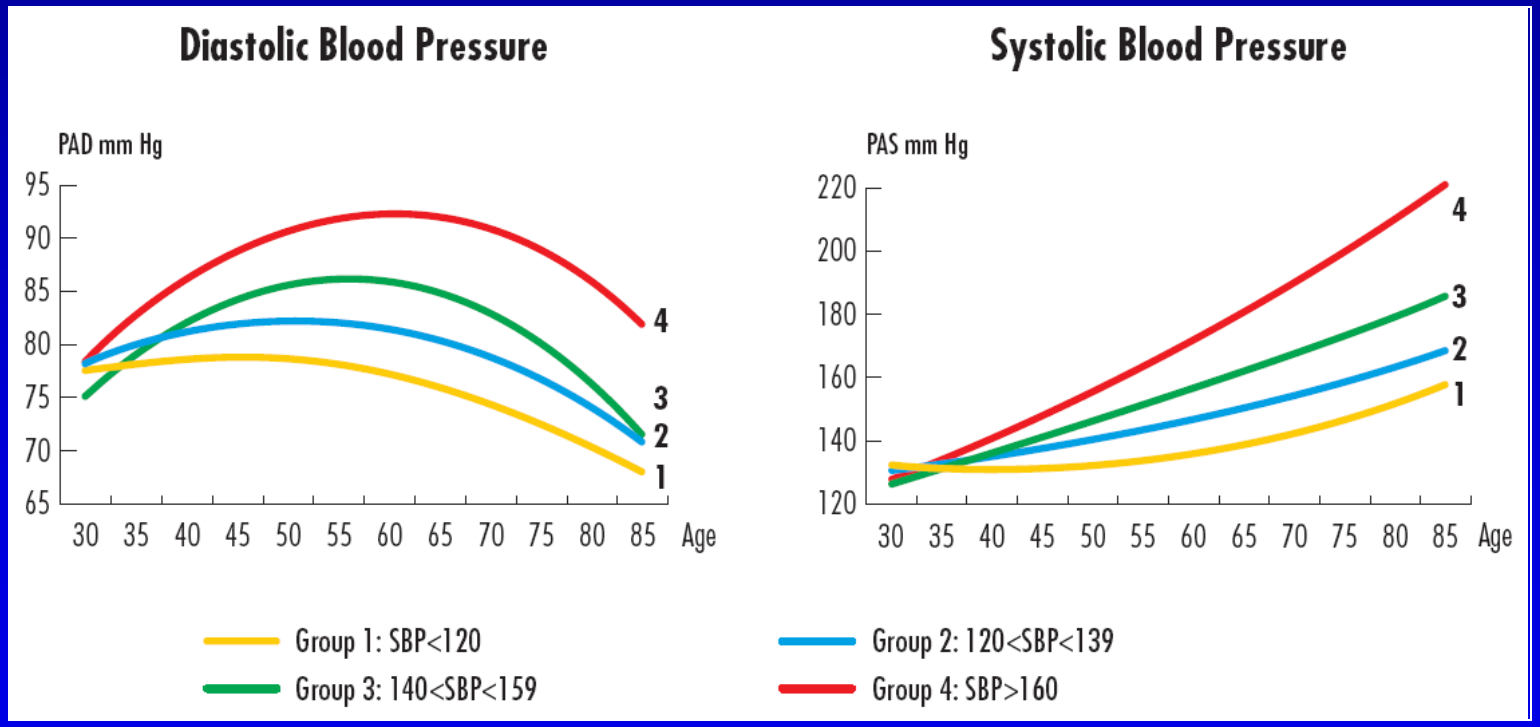
By courtesy of CARIM


C1 > C2 > C3
C1
C2
C3
20-24 ans
29-31 ans
36-42 ans
47-52 ans
71-78 ans
VOLUME ARTÉRIEL (ml)
275
250
225
200
175
150
125
100
75
50
25
0 25 50 75 100 125 150 175 200 225
PRESSION ARTÉRIELLE (mm Hg)


Younger subjects
Older patients
=
+
=
+
Late return
Early return
Slow PWV
Fast PWV
Reflexion sites

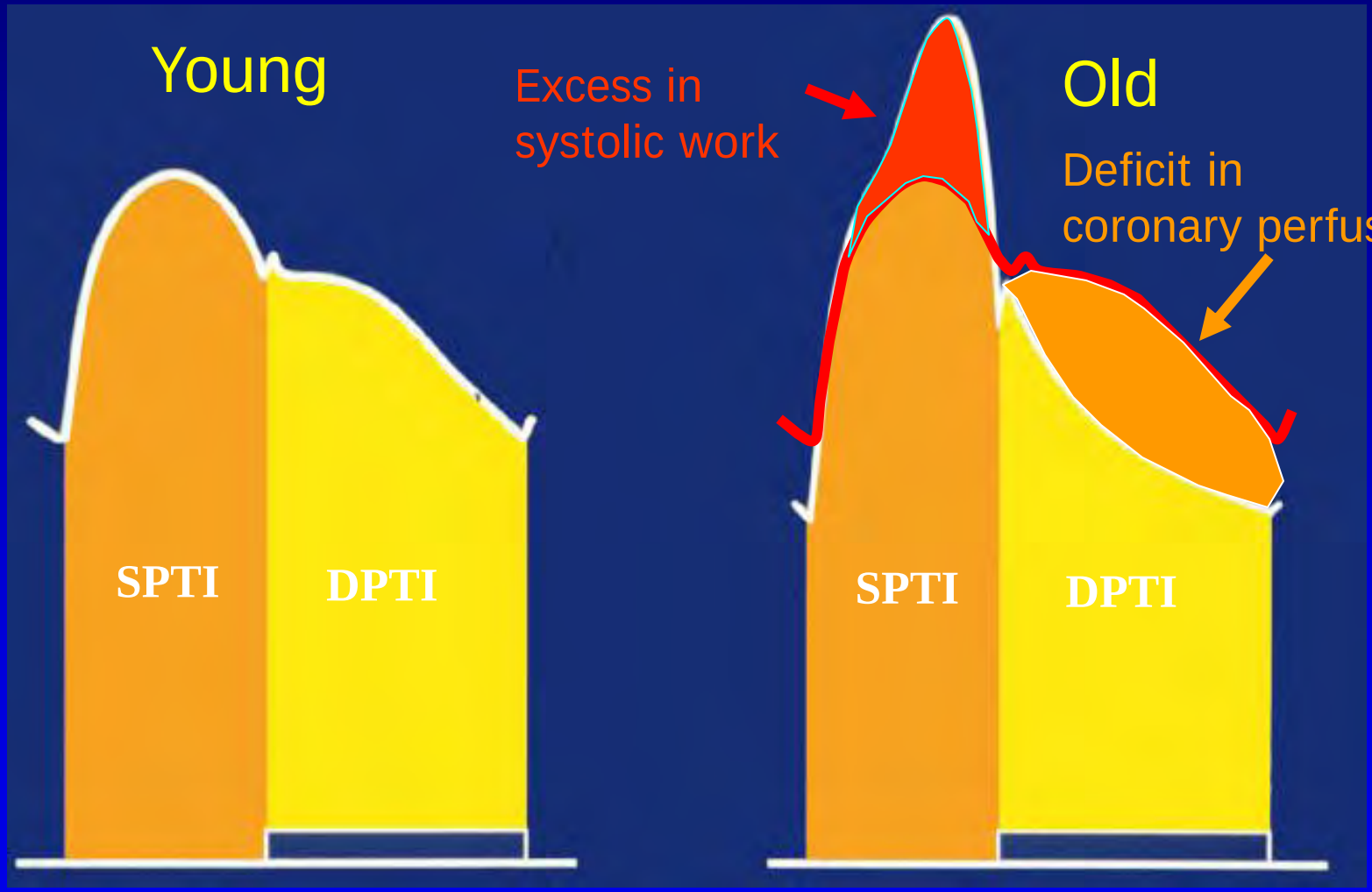
Evolution de la PA avec l'âge

- La systolique augmente avec l'âge
- La diastolique augmente jusqu'à 60 ans puis se stabilise et décroît



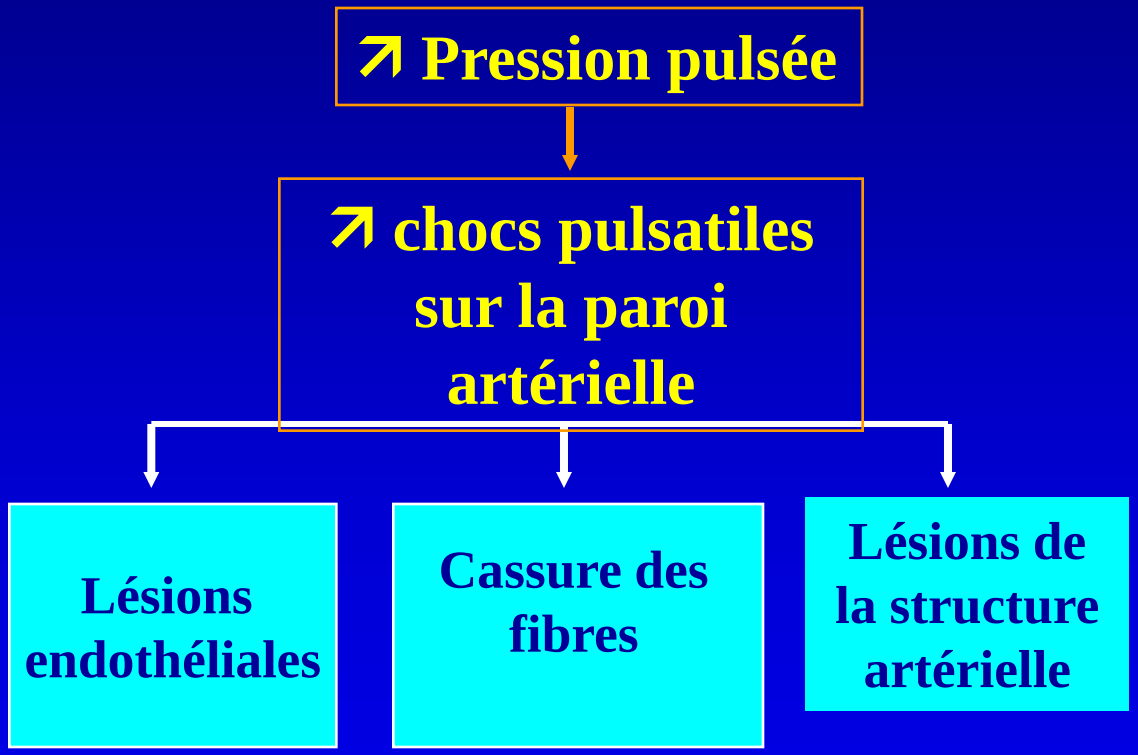
Franklin SS et al. *Circulation.* 1997;96:308-315.

Effects of arterial stiffening on aortic systolic pressure time index (orange area) and aortic diastolic pressure time index (yellow area)



O'Rourke MF : the arterial pulse, lea Fibiger, 1992

CONSEQUENCES PHYSIOPATHOLOGIQUES sur les artères



MESURE DE LA PRESSION ARTERIELLE

LA PRESSION PULSEE

VALEURS DE REFERENCES – PP
Mesure clinique – Etude population

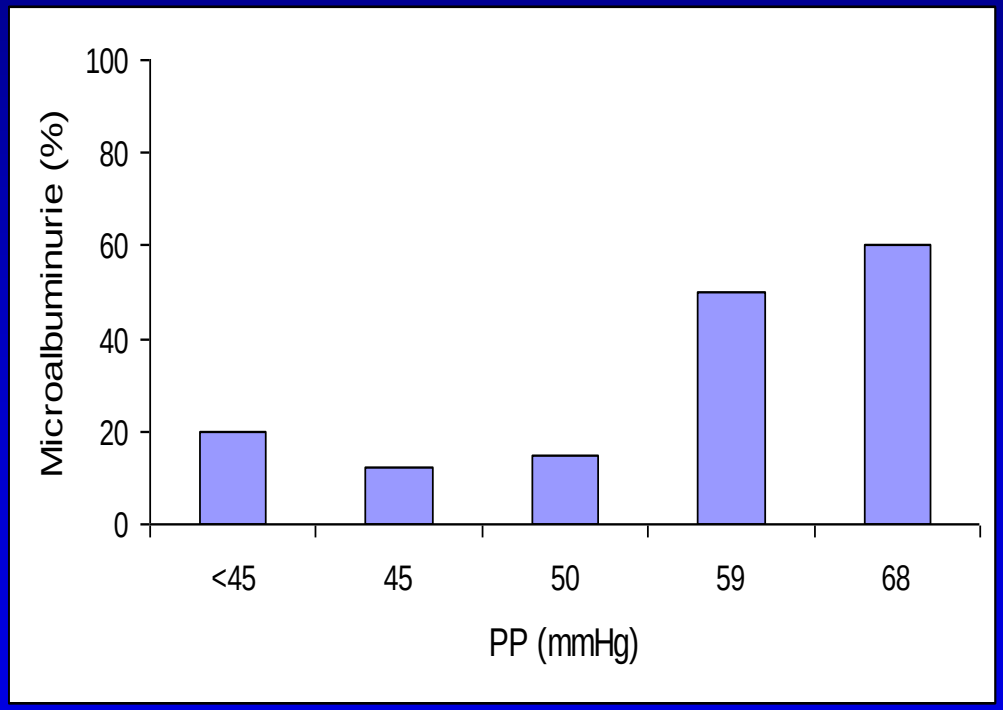
		PP (mmHg)			
	n	moyenne	DS	50ème P	95ème P
Hommes	29692	52	10	50	70
Femmes	31416	49	10	50	65

Asmar et al., 2001

CONSEQUENCES PHYSIOPATHOLOGIQUES

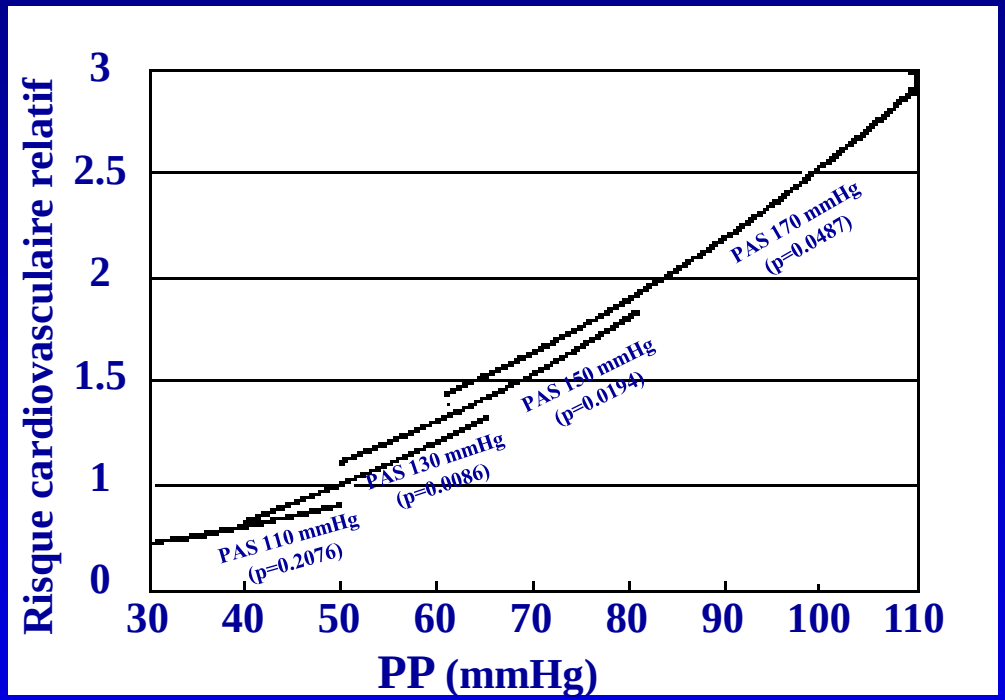
Rein

Corrélation entre PP et microalbuminurie



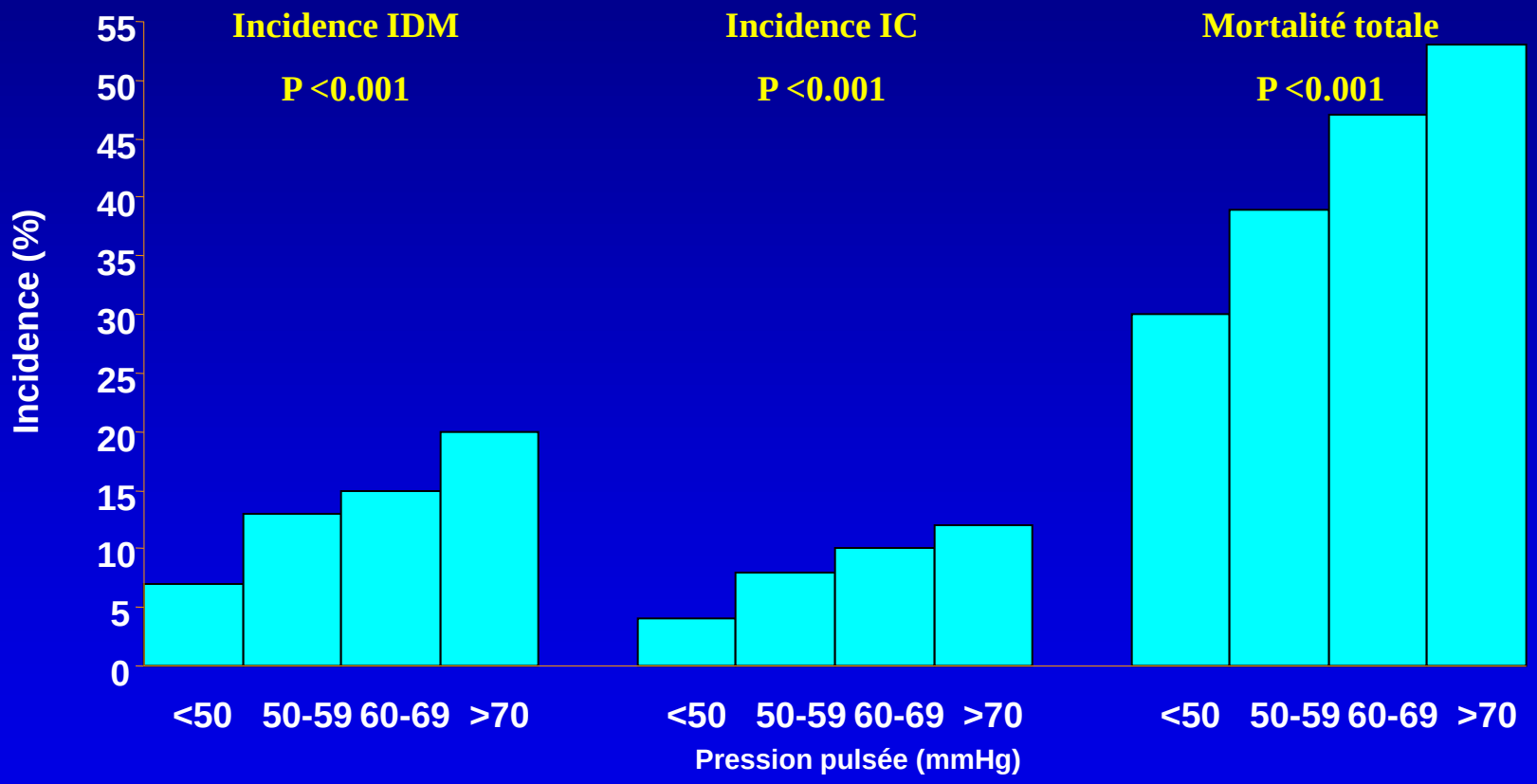
Pedrinelli et al, 1999

PA SYSTOLIQUE, PP ET RISQUE CORONARIEN



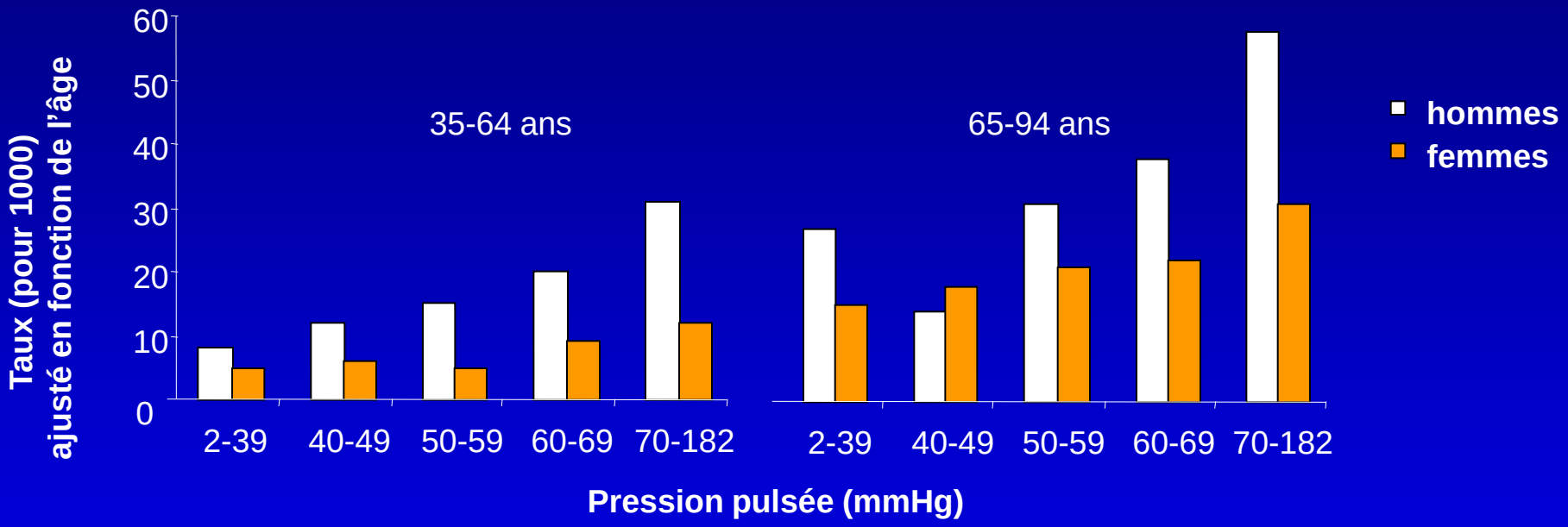
Franklin et al., Circulation 1999, 100 : 354-360

Relations entre PP de base et l'incidence d'IDM, IC et mortalité globale



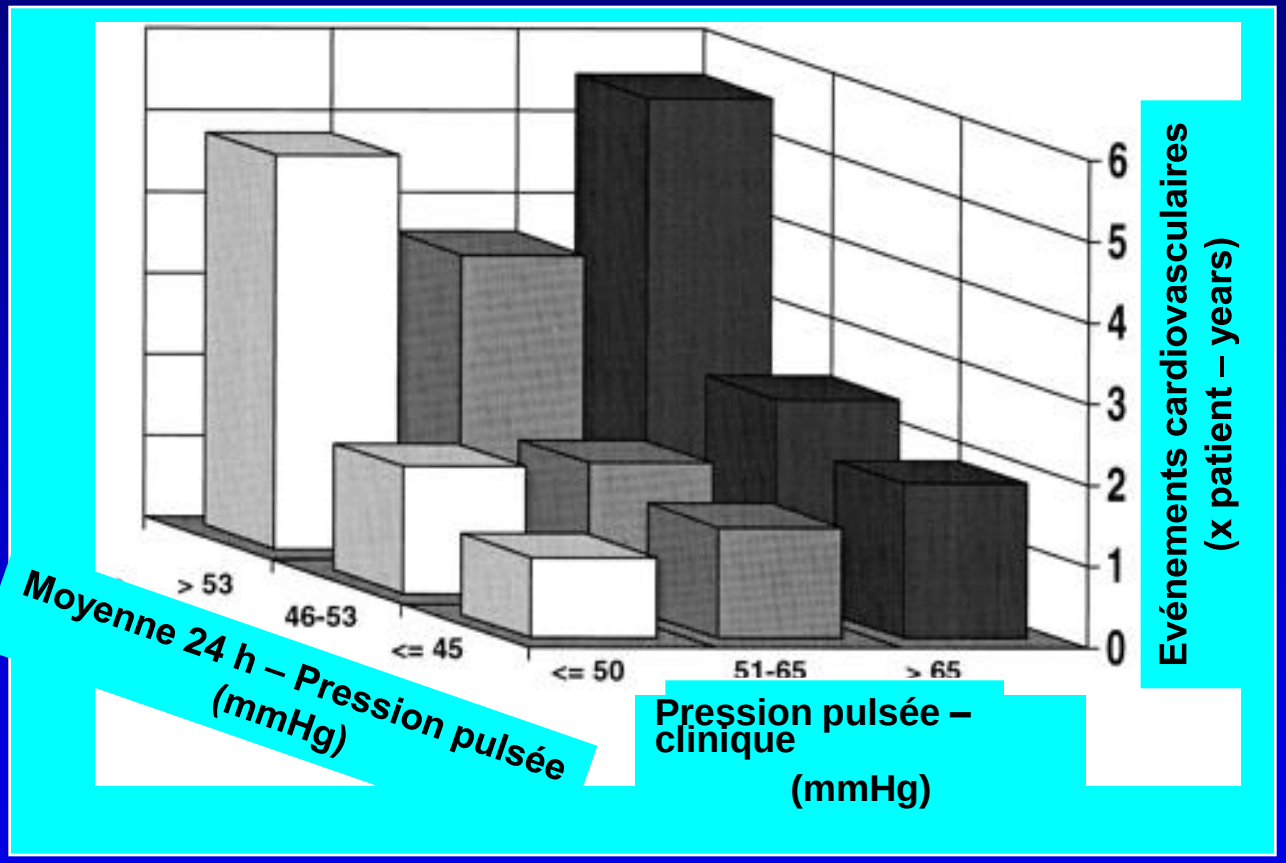
Vaccarino et al, JACC; 2000 - 36: 130

PP ET RISQUE D'EVENEMENTS CARDIOVASCULAIRES



Suivi de 30 ans, étude de Framingham. Toutes les tendances sont significatives à p < 0,05.

PP & RISQUE CARDIOVASCULAIRE

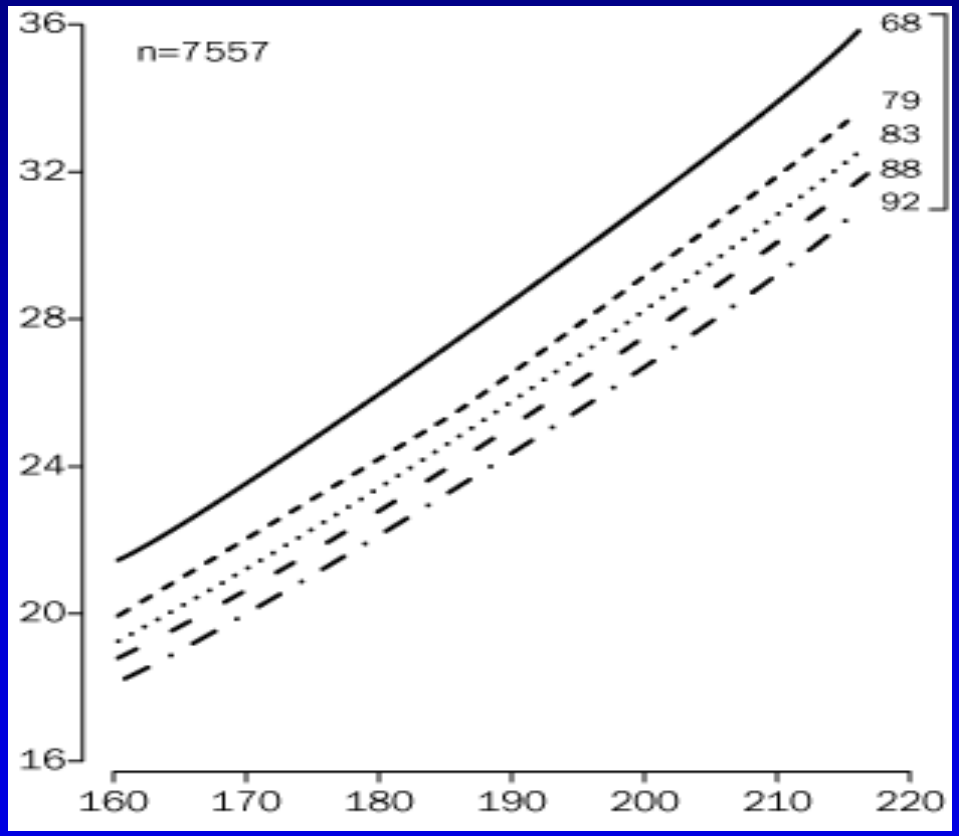


Verdecchia et al., Hypertension 1998

Le rôle pronostique de la pression pulsée sur la mortalité totale chez l'hypertendu âgé (62-76 ans)

(méta-analyse sur 15 693 sujets, 8 essais randomisés)

Risque de mortalité pour 100 sujets à 2 ans (groupe placebo)



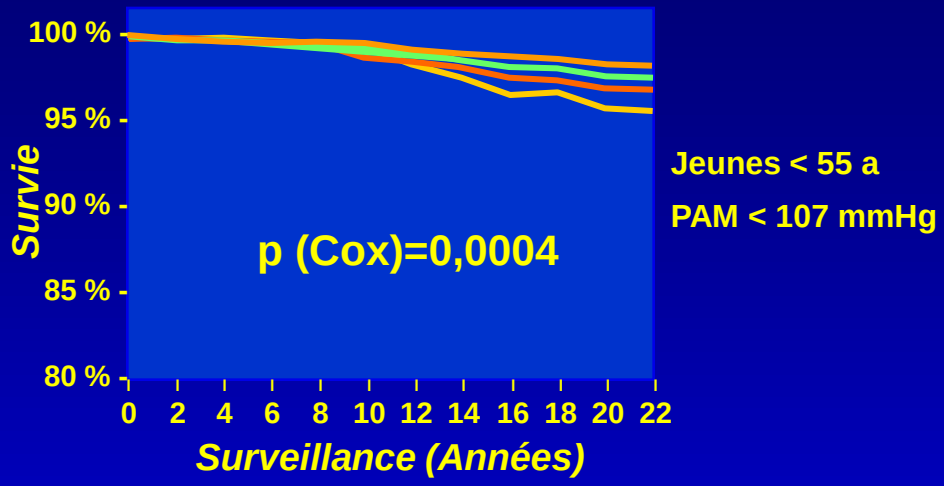
PAD mmHg au début de la surveillance

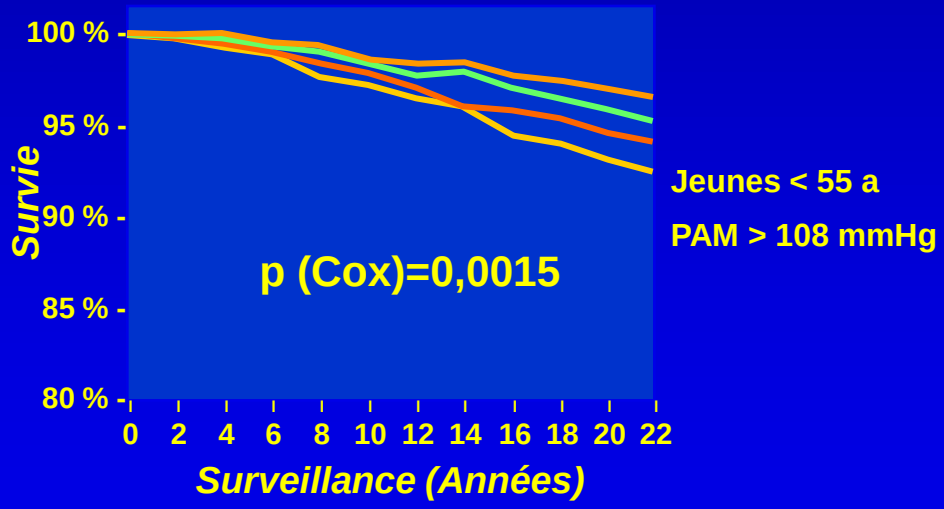
PAS mmHg au début de la surveillance

Staessen J Lancet 2000;355:865-872.

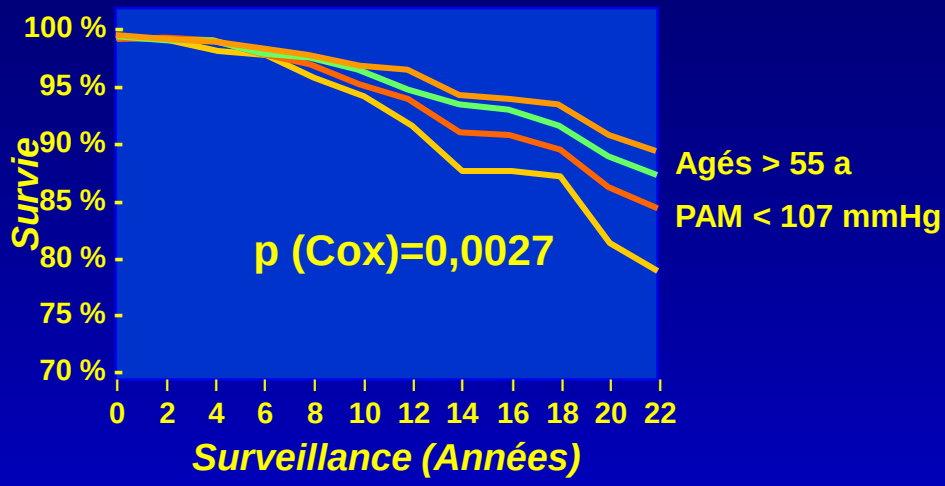
PP ET MORTALITE

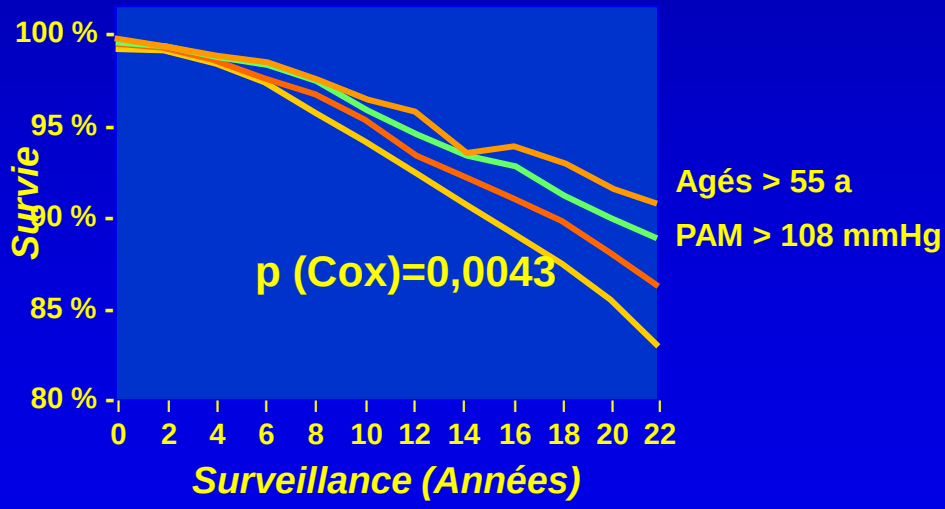
Sujet jeune





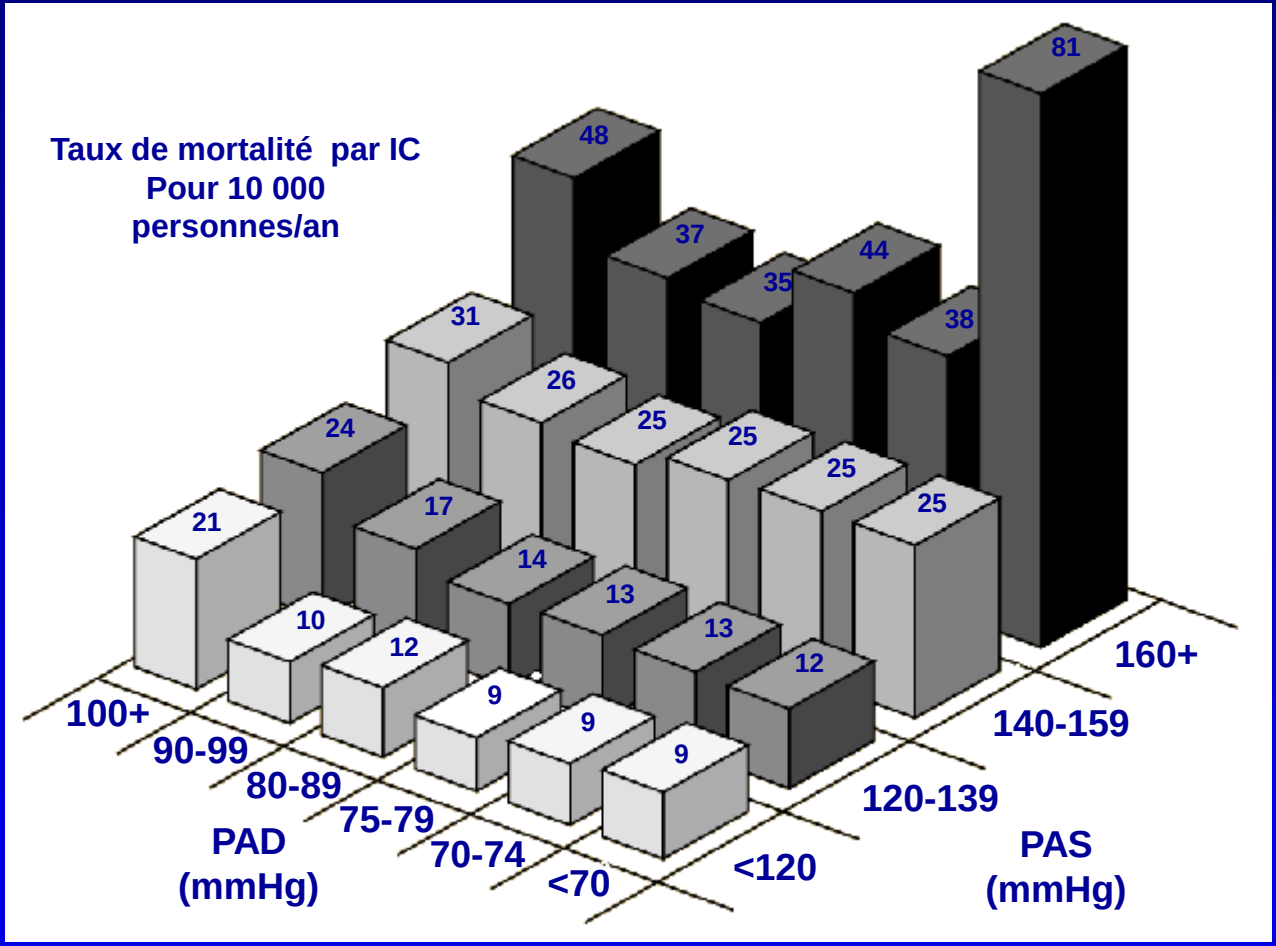
Sujet âgé





Benetos et al., 1997

PRESSION PULSEE ET MORTALITE CORONARIENNE



Black et al., J Hypertens 1999, 17(suppl 5): S3-514

VALEUR PRONOSTIQUE DE LA PP

- Une PP élevée est observée en présence d'athérosclérose et de maladies artérielles (HTA, diabète, artérite …).
- La PP est corrélée à de nombreuses atteintes organiques : ↗ épaisseur intima-média, hypertrophie ventriculaire gauche, ↗ rigidité artérielle, microalbuminurie … etc.
- La PP est un facteur indépendant de mortalité globale et cardiovasculaire.
- Une PP élevé ≥ 65 mmHg
  - Augmente la mortalité
  - Est un facteur prédicteur indépendant d'événements cardiovasculaires.

PRESSION PULSEE

- La PP peut être mesurée à différents niveaux de l'arbre artériel.
- La mesure de la PP brachiale peut être réalisée en clinique ou en ambulatoire.
- La mesure de la PP centrale peut être réalisée par des appareils automatiques : Sphygmocor®, Complior ®.
- Des valeurs de référence sont disponibles pour :
  - La mesure clinique : limite supérieure = 65 mmHg
  - La mesure ambulatoire : limite supérieure = 55 mmHg

ARTERIAL STIFFNESS

Pulse Wave Velocity

PULSE WAVE VELOCITY

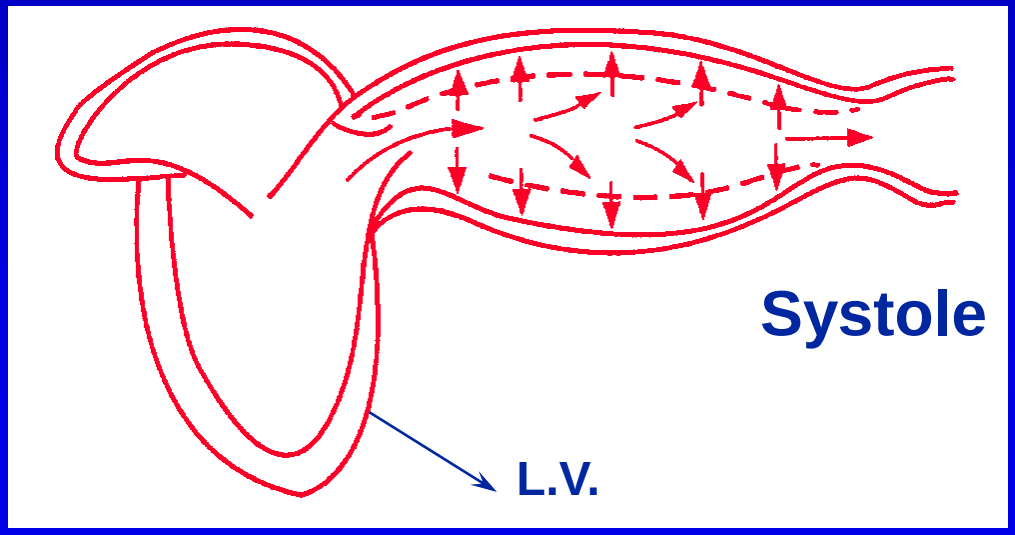
Principles

L.V.E. generates a pulse wave which propagates throughout the arterial wall at a finite speed.

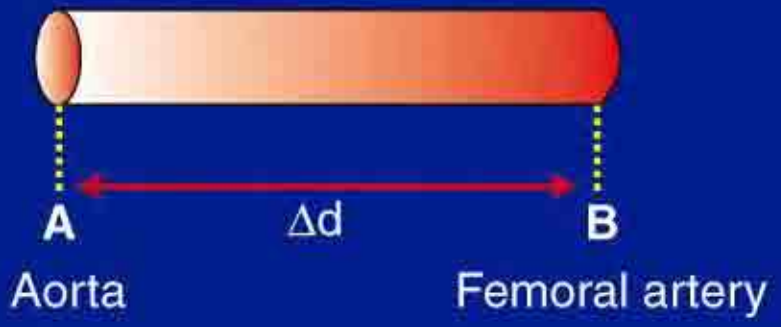
Blood = incompressible fluid

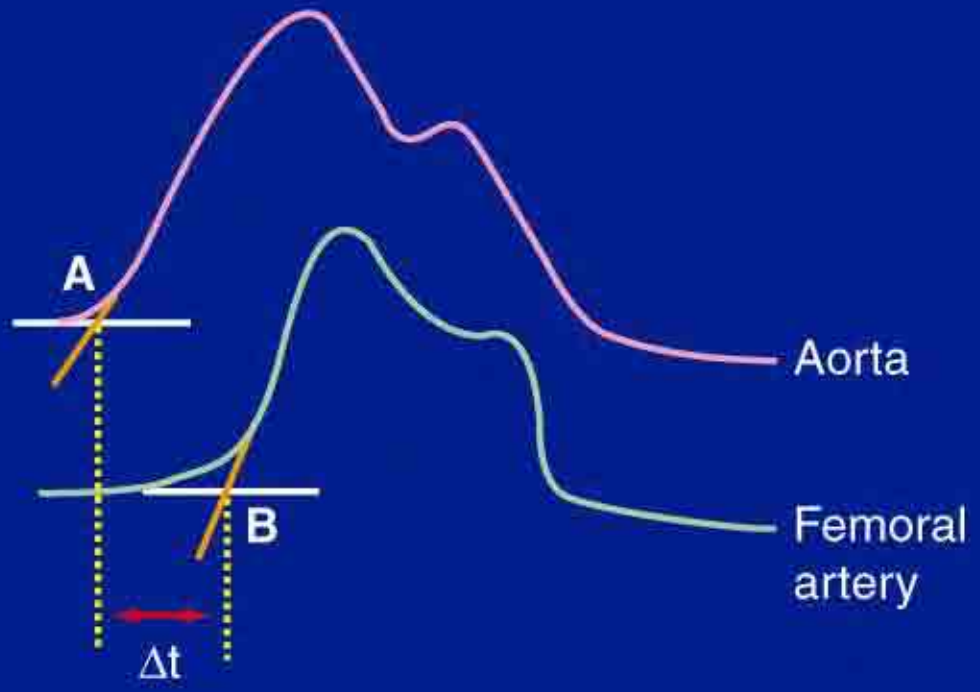
Artery = elastic conduit

} → **Propagation accurs along the arterial wall**



PWV: Measurement





PWV = Distance (Δd) / Time delay = (Δt m/sec)

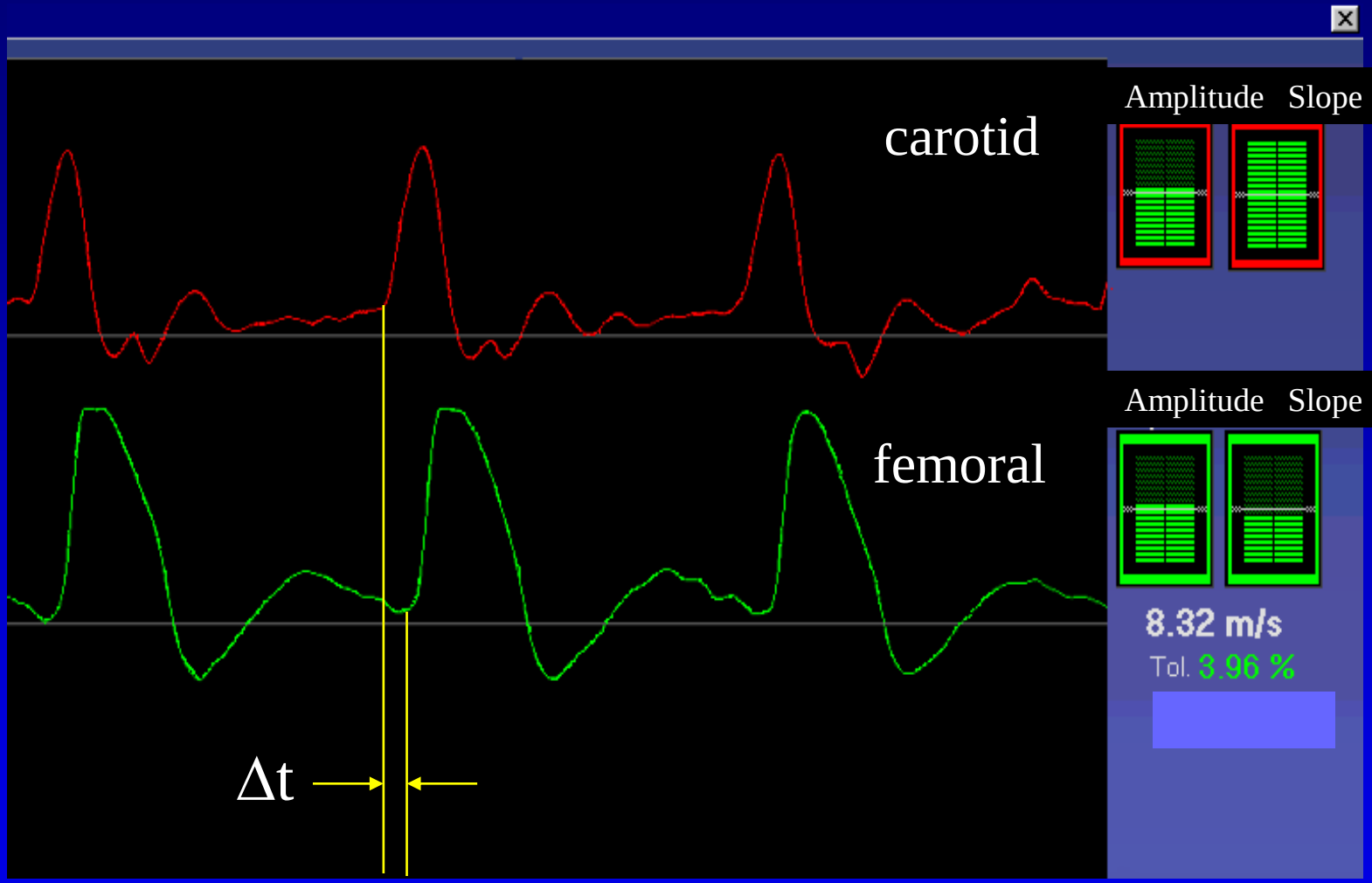
Usually measured on 10 heartbeats
PWV = pulse wave velocity

PWV AUTOMATIC DEVICES

Complior®

Colin®

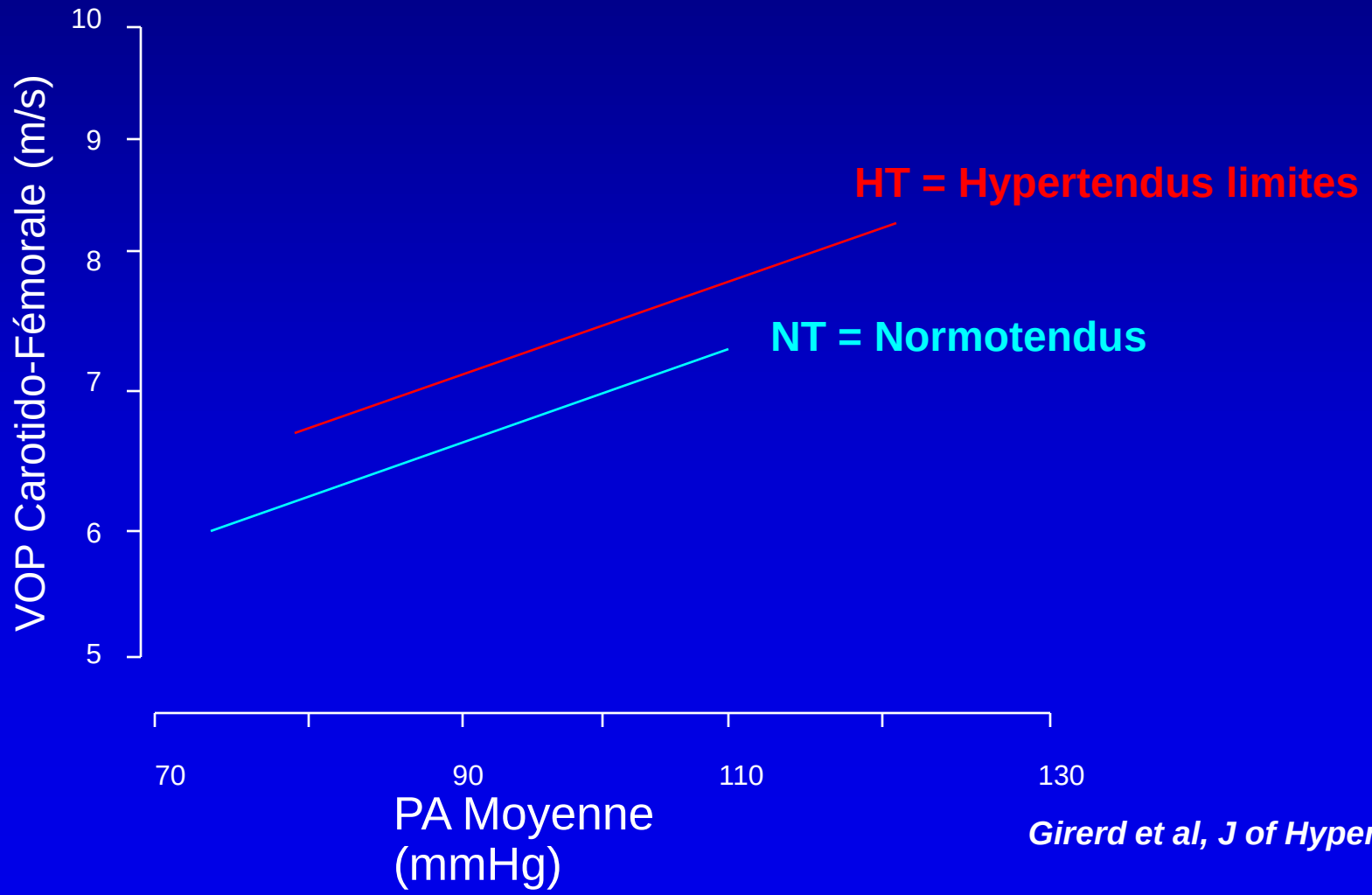
PWV - Simultaneous recordings



ARTERIAL STIFFNESS
&
SURROGATE MARKERS

Rigidité artérielle & HTA

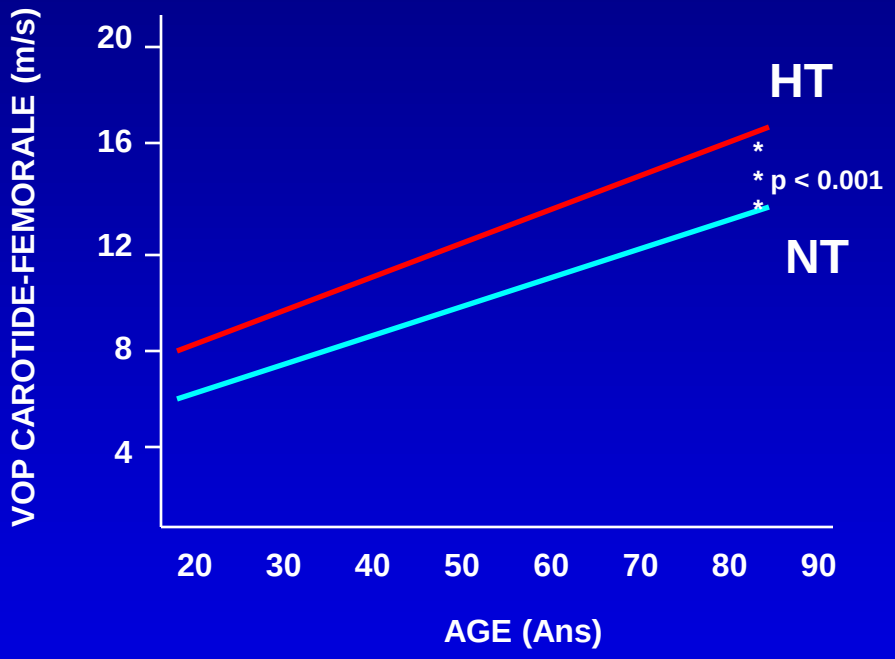
VOP chez des NT et HT limites

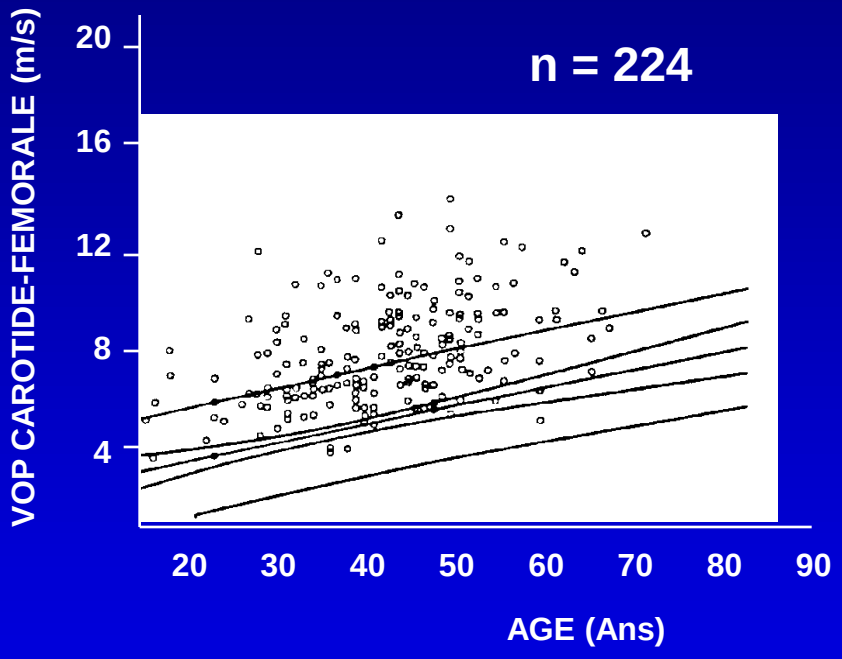


Girerd et al, J of Hyper, 1989

Rigidité Artérielle & HTA

VOP chez les hypertendus en comparaison aux normotendus





NT ——— **HT** ———

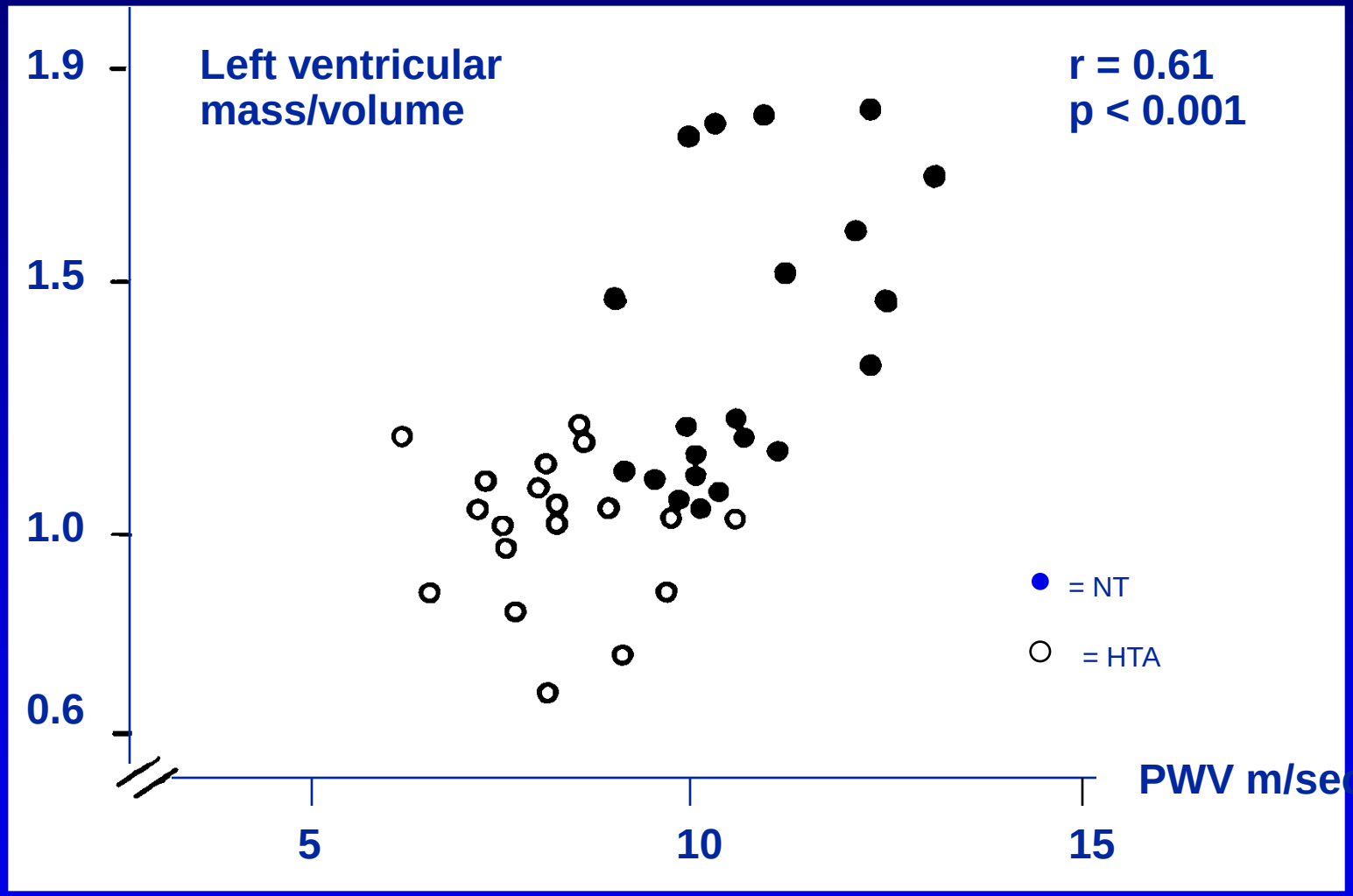
y = 0.0628x + 5.728 **y = 0.123x + 6.27**

r = 0.48* ; y = 0.123x + 6.27**

Asmar et al, Blood Pressure, 1995

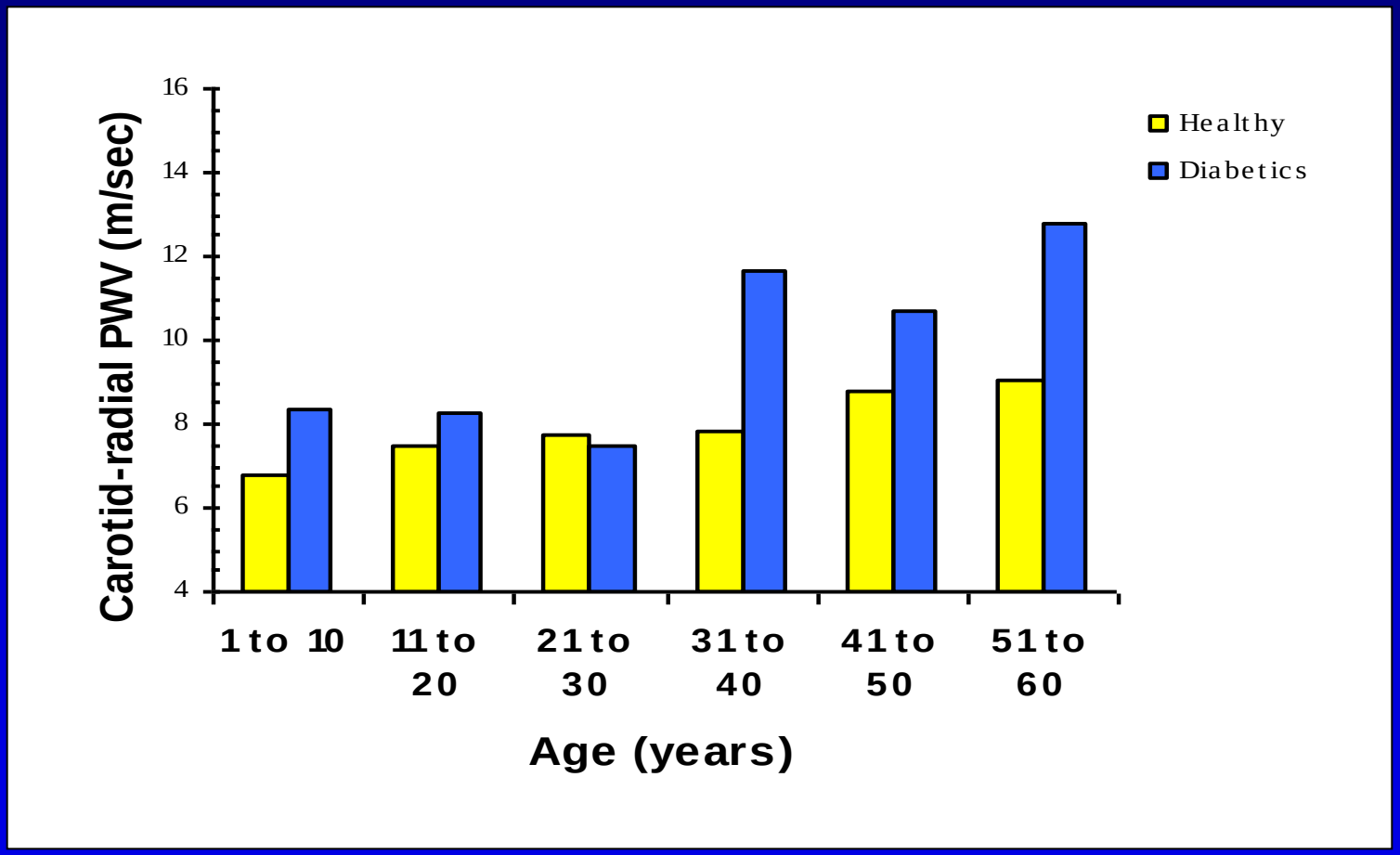
PULSE WAVE VELOCITY

P.W.V. is an independant determinant of L.V.H.



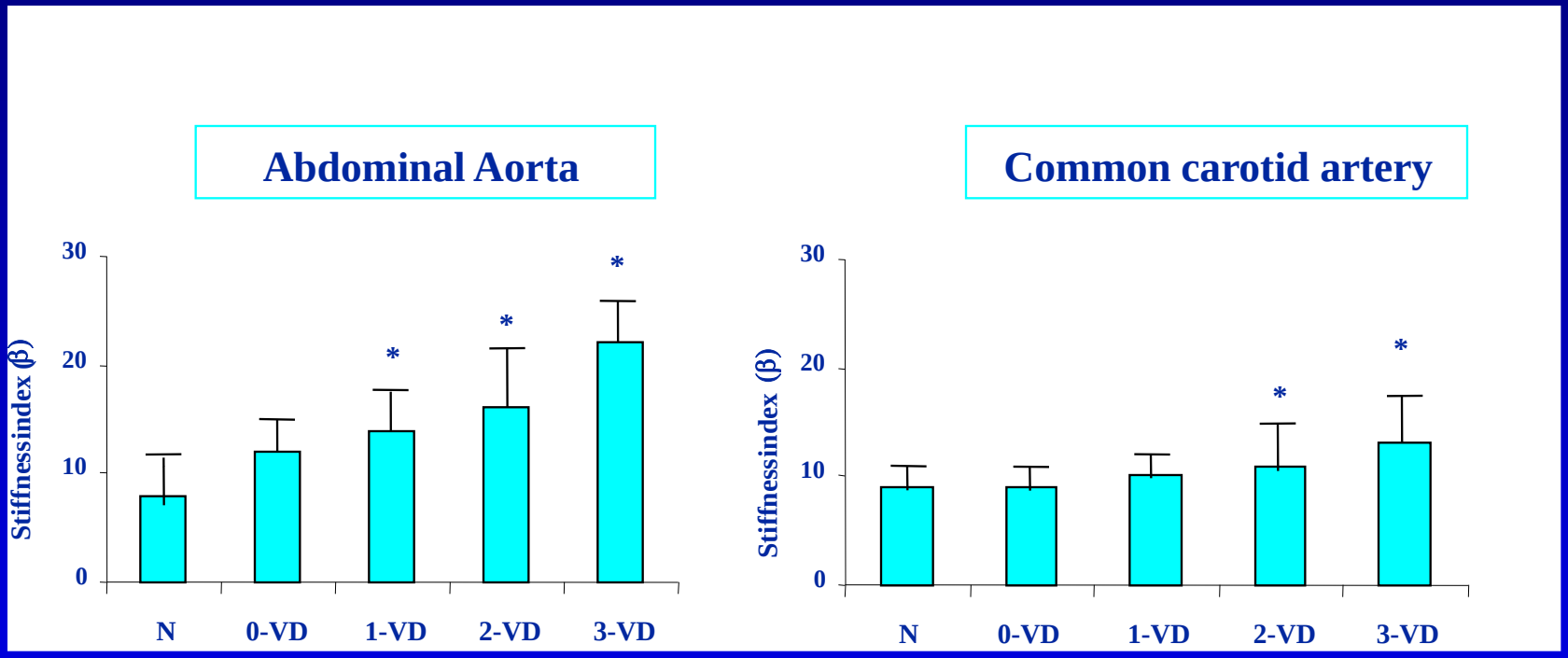
Bouthier et al, Am Heart J 1985

ARTERIAL STIFFNESS in DIABETIC SUBJECTS



(Adapted from Woolam et al.

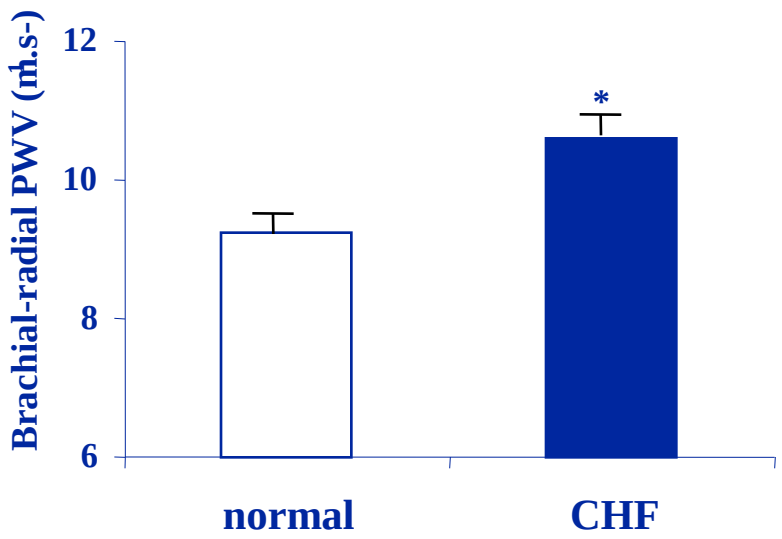
ARTERIAL STIFFNESS INDEX & CAD

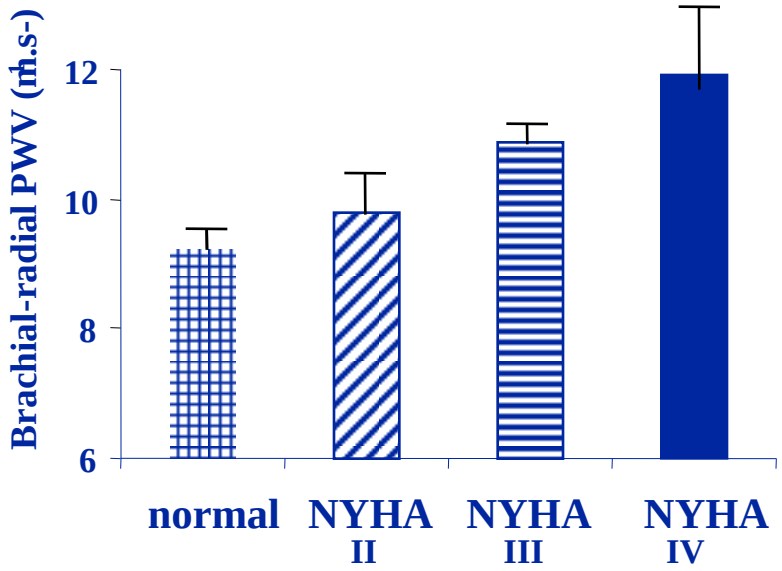


VD: vessel disease

adapted from Hirai et al.

PWV IN PATIENTS WITH CHF





adapted from Arnold et al.

ARTERIAL STIFFNESS

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PREDICTOR OF MORBIDITY-MORTALITY

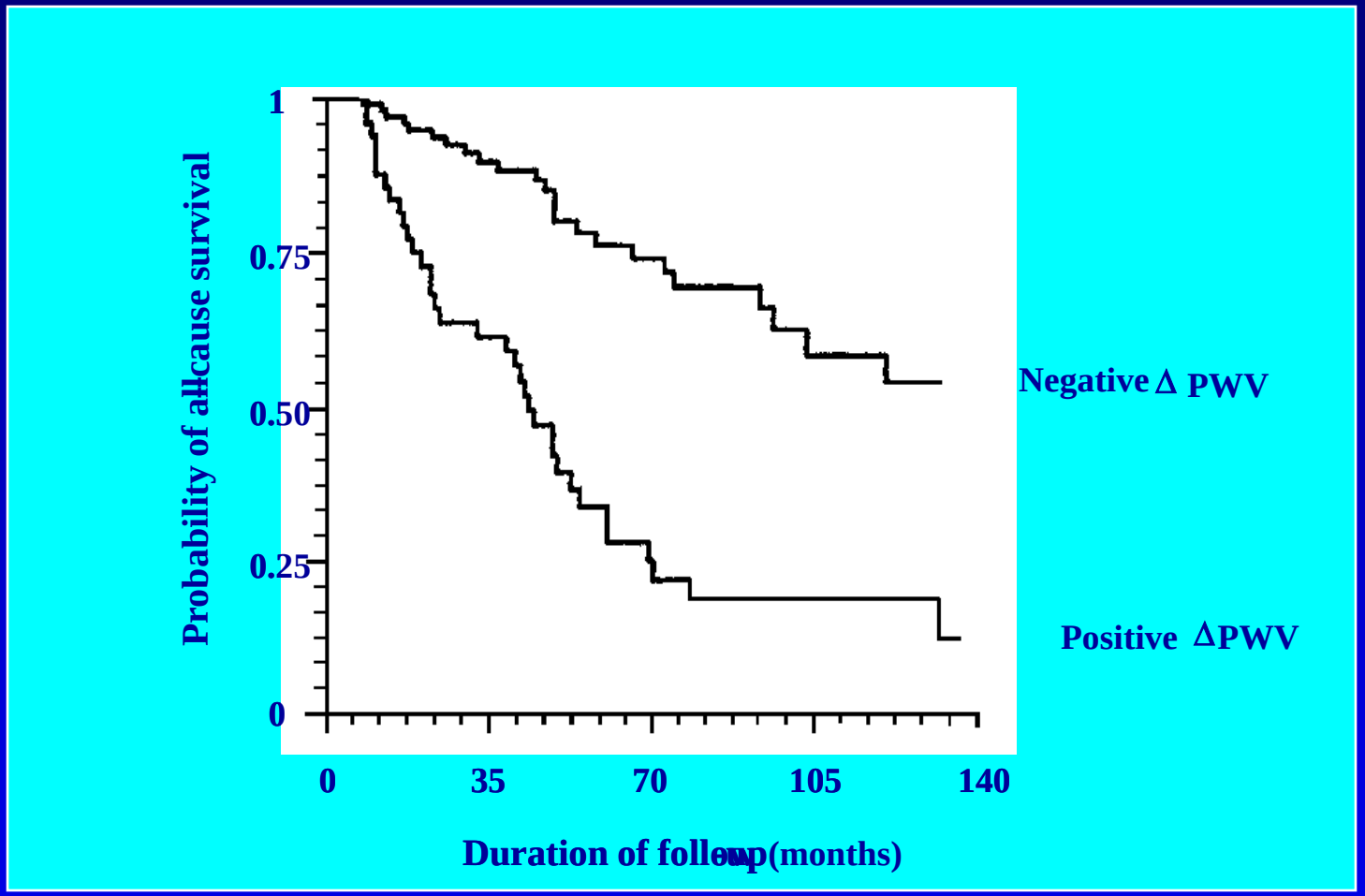
Independent predictive value of C-F PWV for CV events

First author (year, country)	Events	Follow-up (years)	Type of patient (number)	Mean age at entry
Blacher (1999, Fr)	CV mortality	6.0	ESRD (241)	51
Laurent (2001, Fr)	CV mortality	9.3	Hypertension (1980)	50
Meaume (2001, Fr)	CV mortality	2.5	Elderly (>70) (141)	87
Shoji (2001, Jp)	CV mortality	5.2	ESRD (265)	55
Boutouyrie (2002, Fr)	CHD events	5.7	Hypertension (1045)	51
Cruickshank (02, GB)	All cause M.	10.7	Diabetes and MS (571)	51
Laurent (2003, Fr)	Fatal strokes	7.9	Hypertension (1715)	51
Sutton-Tyrrell (2005, USA)	CV events	4.6	Elderly (2488)	74
Shokawa (2005, Jp)	CV mortality	10	General pop. (492)	64
Willum-Hansen (2006, Dk)	CV mortality	9.4	General pop. (1678)	55
Mattace-Raso (2006, Neth.)	CV mt, CHD	4.1	Elderly (2835)	72
Roman (2007, USA)	CV events	4.8	Gen. Pop. (3520)	58

12 studies and # 13 000 subjects

Laurent S et al. Eur Heart J 2006

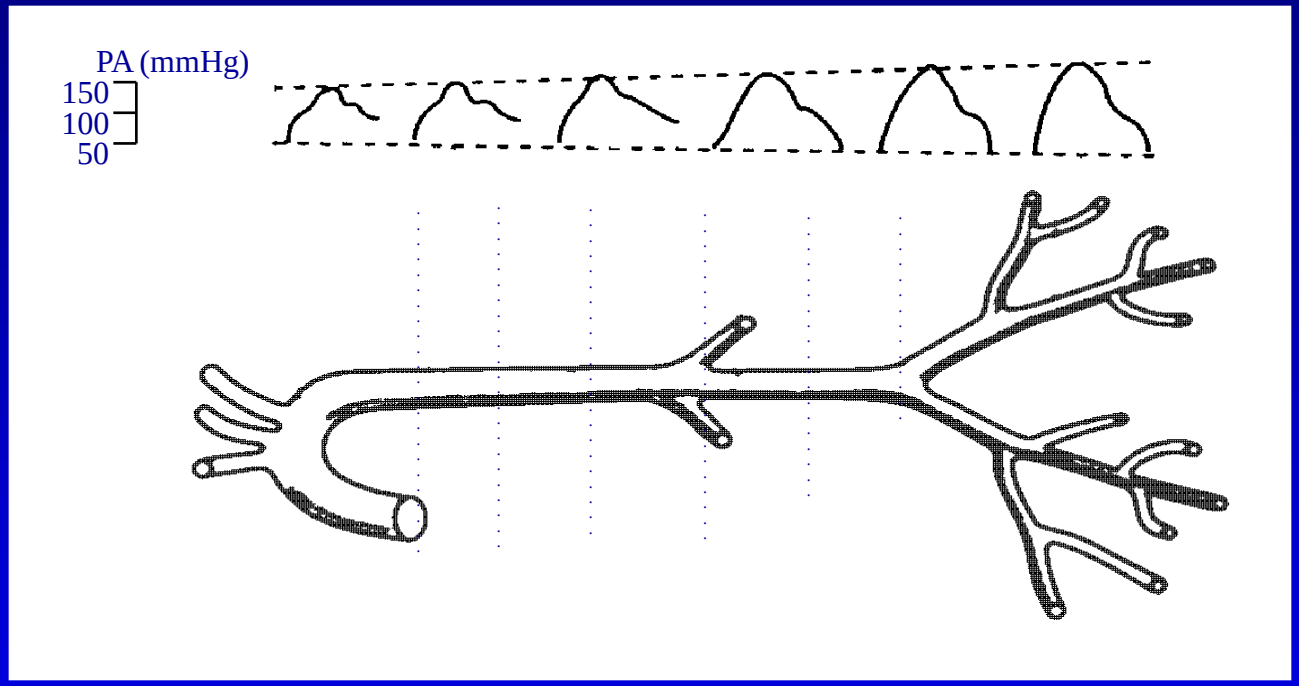
PROGNOSIS VALUE OF PWV CHANGES



London & al., Hypertension. 2001

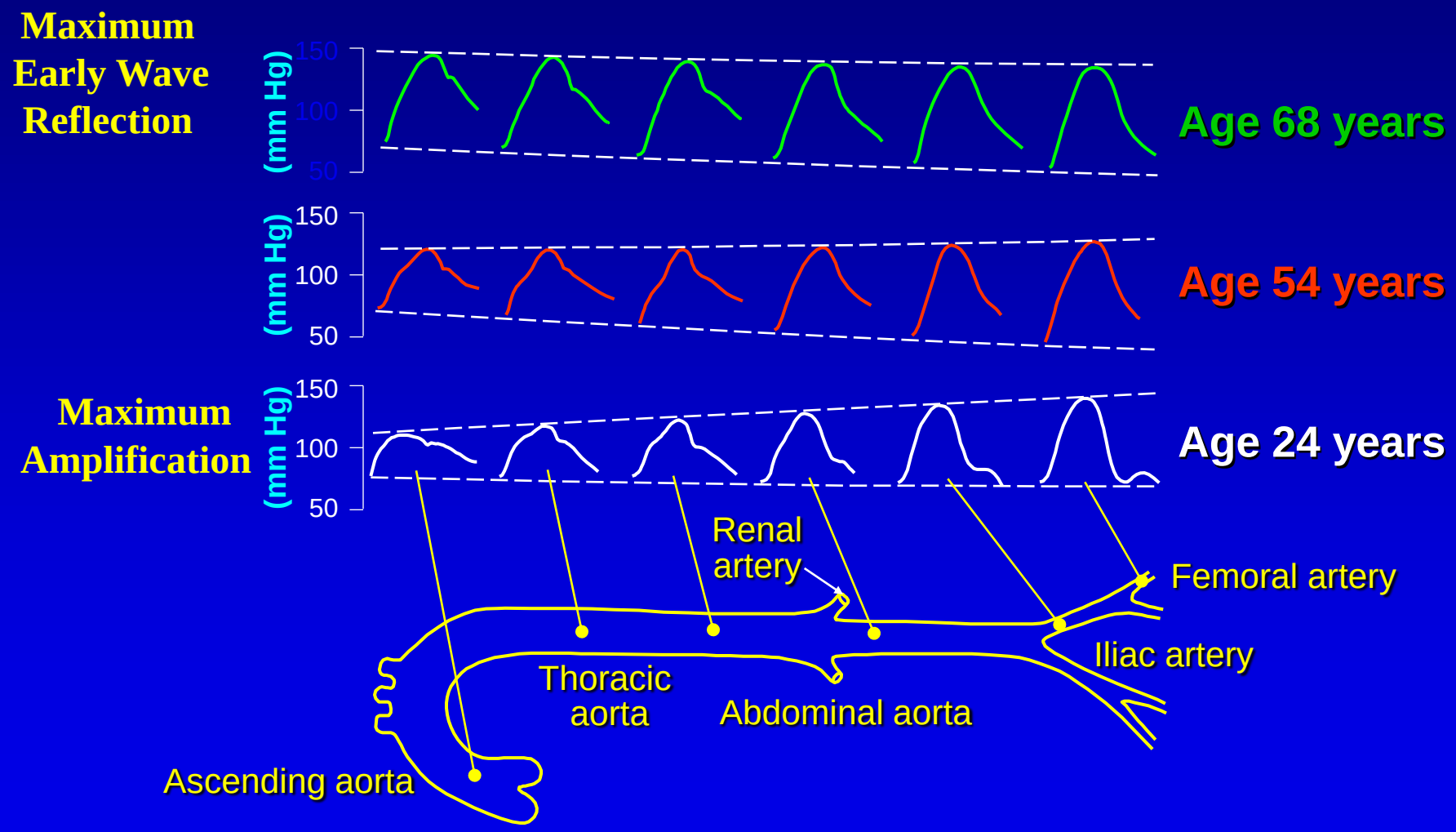
BASES HEMODYNAMIQUES

Amplification de la pression pulsée



La PP augmente des artères centrales vers les artères périphériques.

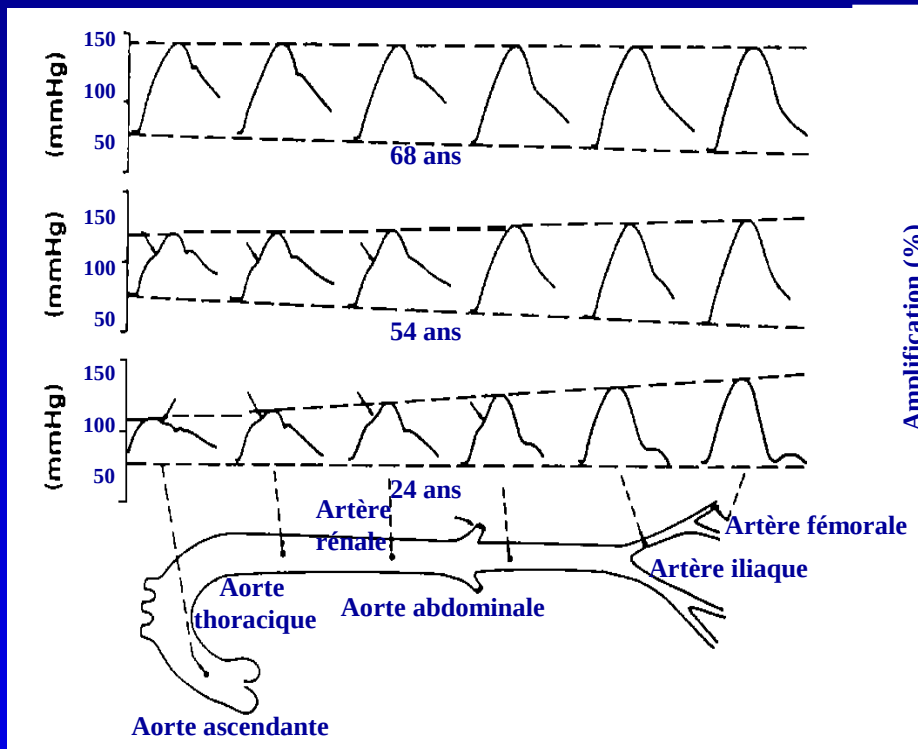
Blood pressure curves

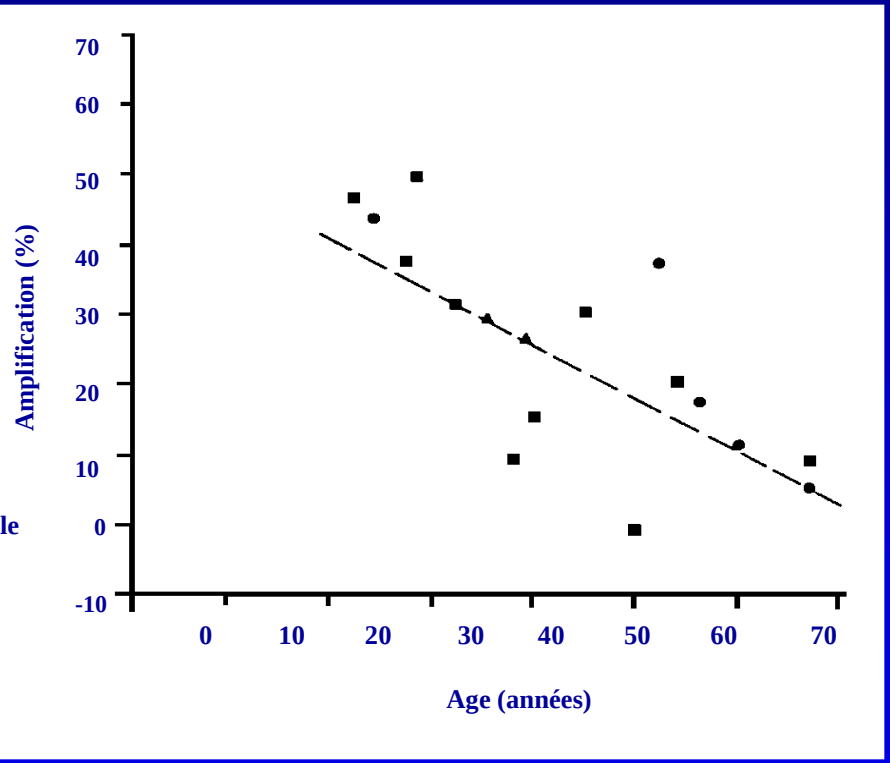


Nichols WW, et al. Arterial Vasodilation. Philadelphia,1993;32.

BASES HEMODYNAMIQUES

Amplification des ondes de pression





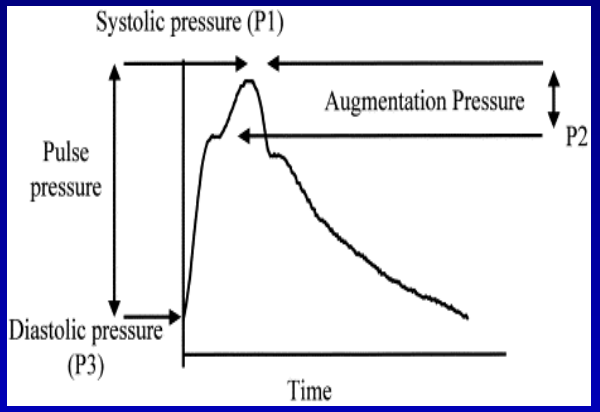
MESURE DE LA PRESSION PULSEE

- PP brachiale
  - **Clinique**
  - **Ambulatoire**
  - **Automesure**
- PP locale et centrale
  - **Sphygmocor®**
- PP centrale et rigidité artérielle
  - **Complior®**

CENTRAL BP

Central PP and augmentation index (AIx)

- can be measured non invasively at the aortic or carotid levels
- are independent predictors of CV events
- are affected by antihypertensive drugs differently from brachial BP



AIx = augmentation pressure / pulse pressure

The prognosis role of central as opposed to brachial BP needs to be further confirmed in large scale observational and interventional studies, before being used routinely as a diagnostic tool.

The measure of central BP can help understanding the results of large clinical trials

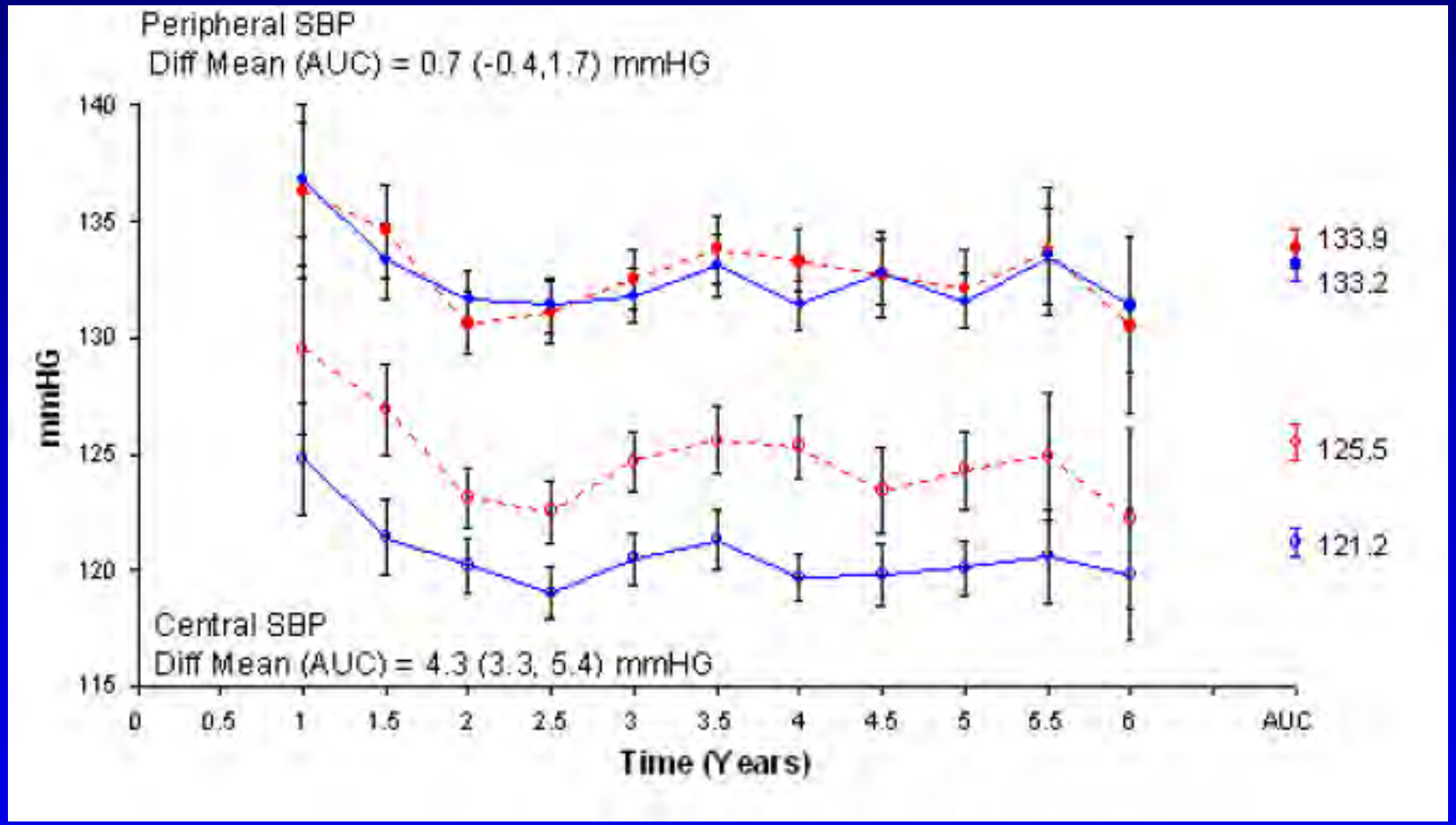
2007 ESH Guidelines. J Hypertens 2007, 25:1105

Central BP and AIx - Independent predictive value for CV events

7 studies, including 7,441 subjects

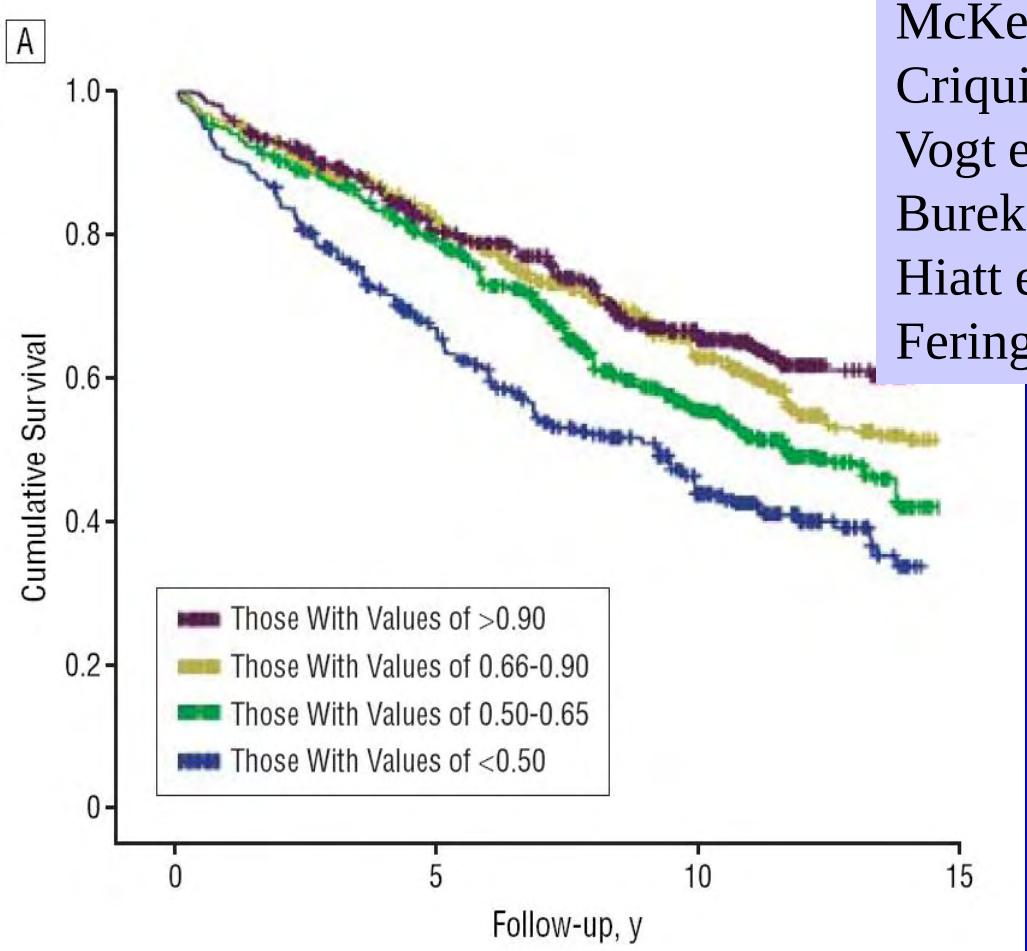
First author (year, country) Mean age at entry	Events	Follow-up (years)	Type of patient (number)
a. Central pulse pressure			
Safar (2002, Fr) 54	All cause mortality	4.3	ESRD (180)
Williams (2006, UK) 63	CV events	3.4	HT, ASCOT study (2073)
Roman (2007, USA) 58	CV events	4.8	Strong Heart Study (3520)
Jankowski (2008, Pol)	CV events	4.5	Coronary patients (1109)
b. Carotid augmentation index (AIx)			
London (2001, Fr)	All and CV mortal.	4.3	ESRD (180) 54
Williams (2006, UK)	CV events	3.4	HT, ASCOT study (2073) 63
Weber (2005, Austria)	CV events	2.0	CHD undergoing PCI (262) 66
Chirinos (2005, USA)	CV events	3.5	CHD undergoing PCI (297) 64

The Conduit Artery Functional Evaluation (CAFE) ASCOT SUBSTUDY



Williams & al, 2006

Long-term prognostic value of resting ankle-brachial index



McKenna et al.	Atherosclerosis	1991
Criqui et al.	NEJM	1992
Vogt et al.	JAMA	1993
Burek et al.	JACC	1999
Hiatt et al.	NEJM	2001
Feringa et al.,	Arch Int Med	2006

Feringa et al., Arch Int Med 2006

JAMA®

Online article and related content current as of September 13, 2008.

Ankle Brachial Index Combined With Framingham Risk Score to Predict Cardiovascular Events and Mortality: A Meta-analysis

Ankle Brachial Index Collaboration

JAMA. 2008;300(2):197-208 (doi:10.1001/jama.300.2.197)

The studies included a total of 24 955 men and 23 339 women.

10-year cardiovascular mortality (%) according to baseline ABI

Group	Low ABI (≤0.90)	Normal ABI (1.11–1.40)
Men	18.7	4.4
Women	12.6	4.1

The risks remained elevated after adjustment for Framingham risk score, with a low ABI associated with approximately twice the 10-year risk of total mortality, cardiovascular mortality, or major coronary events compared with the overall rate in each Framingham risk score category.

"Conclusion Measurement of the ABI may improve the accuracy of cardiovascular risk prediction beyond the FRS…"

Noninvasive assessment of IMT and plaque by ultrasound

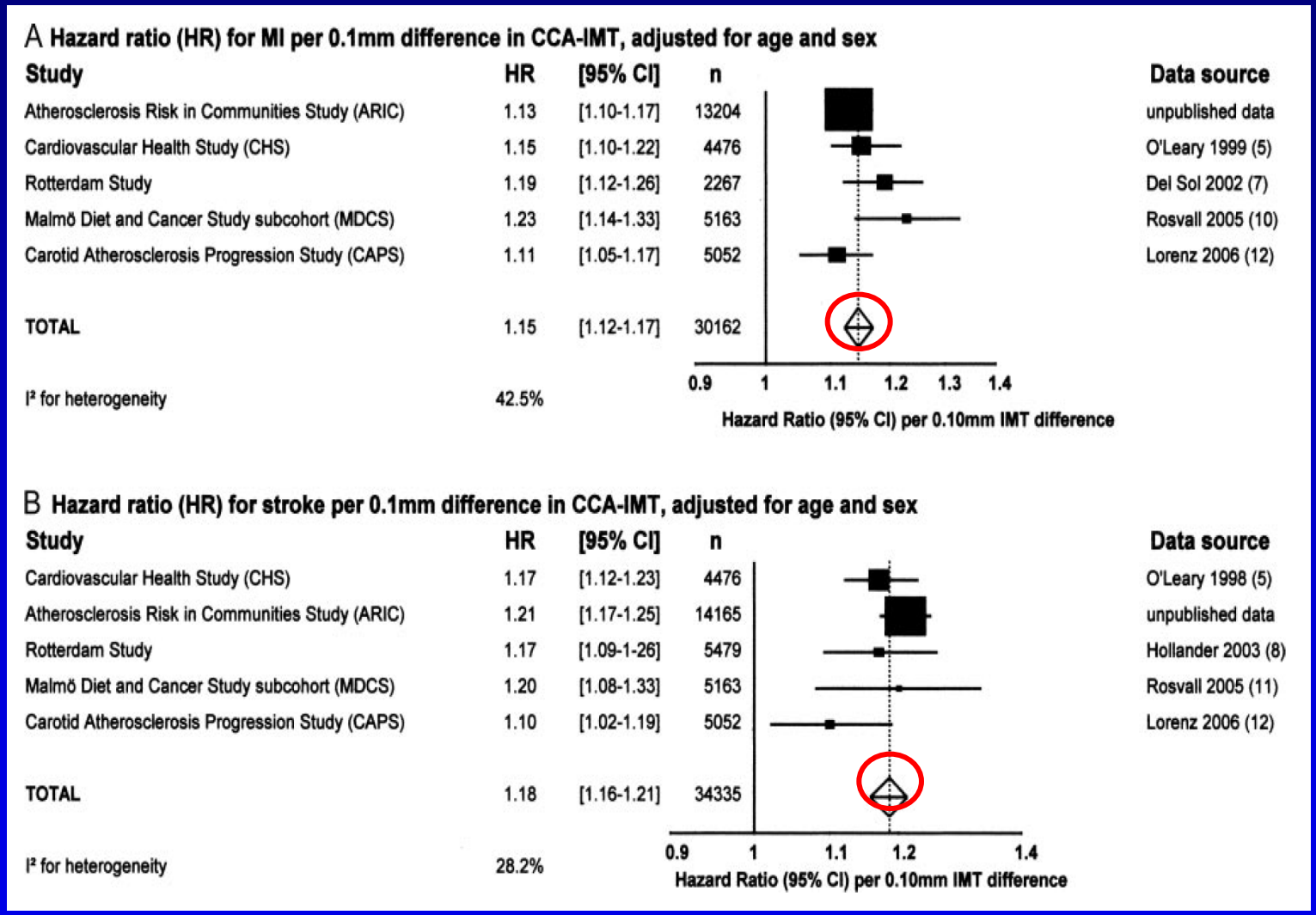
Artlab system wall motion real time, RF

X14 RF

Dist ± 1.7 µm

Prediction of Clinical Cardiovascular Events With Carotid Intima-Media Thickness

A Systematic Review and Meta-Analysis



Lorenz M et al Circulation 2007; 115: 459

Carotid Intima-Media Thickness and Antihypertensive Treatment

A Meta-Analysis of Randomized Controlled Trials

Ji-Guang Wang, MD, PhD; Jan A. Staessen, MD, PhD; Yan Li, MD, PhD; Luc M. Van Bortel, MD, PhD; Tim Nawrot, PhD; Robert Fagard, MD, PhD; Franz H. Messerli, MD; Michel Safar, MD

22 trials published between 1996 and 2005:

- 8 trials antihypertensive drugs vs placebo or no-treatment

- 9 trials new with old drug classes

- 5 trials ACE inhibitors and CCBs

"…Conclusions: Calcium channel blockers reduce carotid intima-media thickening. This mechanism might contribute to their superior protection against stroke."

Stroke online June 8, 2006

ESH - Subclinic organ damage

- **EKG LVH** (Sokolow-Lyon > 38 mm; Cornell > 2440 mm*ms)

  or:

- **LVH at echocardiography** (LVMI M ≥ 125 g/m^2, F ≥ 110 g/m^2) *
- **Thickening of the carotid arteries** (IM T > 0.9 mm) **or plaques**
- **Carotid-femoral pulse wave velocity** >12 m/sec
- **Ankle-brachial index < 0.9**
- **Slight increase in Serum Creatinine** (M: 1.3-1.5 mg/dl; F:1.2-1.4 mg/dl)
- **Reduced GFR** † (< 60 ml/min/1.73m^2)

  or of creatinine clearance◊ (< 60 ml/min)
- **Microalbuminuria** (30-300 mg/24h **or albumin/creatinine ratio:** ≥ 22 (M) o ≥ 31 (F) mg/g)

*higher risk for concentric LVH; ◊ Cockroft Gault's formula; † MDRD formula;