ANALYSE ET TRAITEMENT D'UNE HTA RESISTANTE

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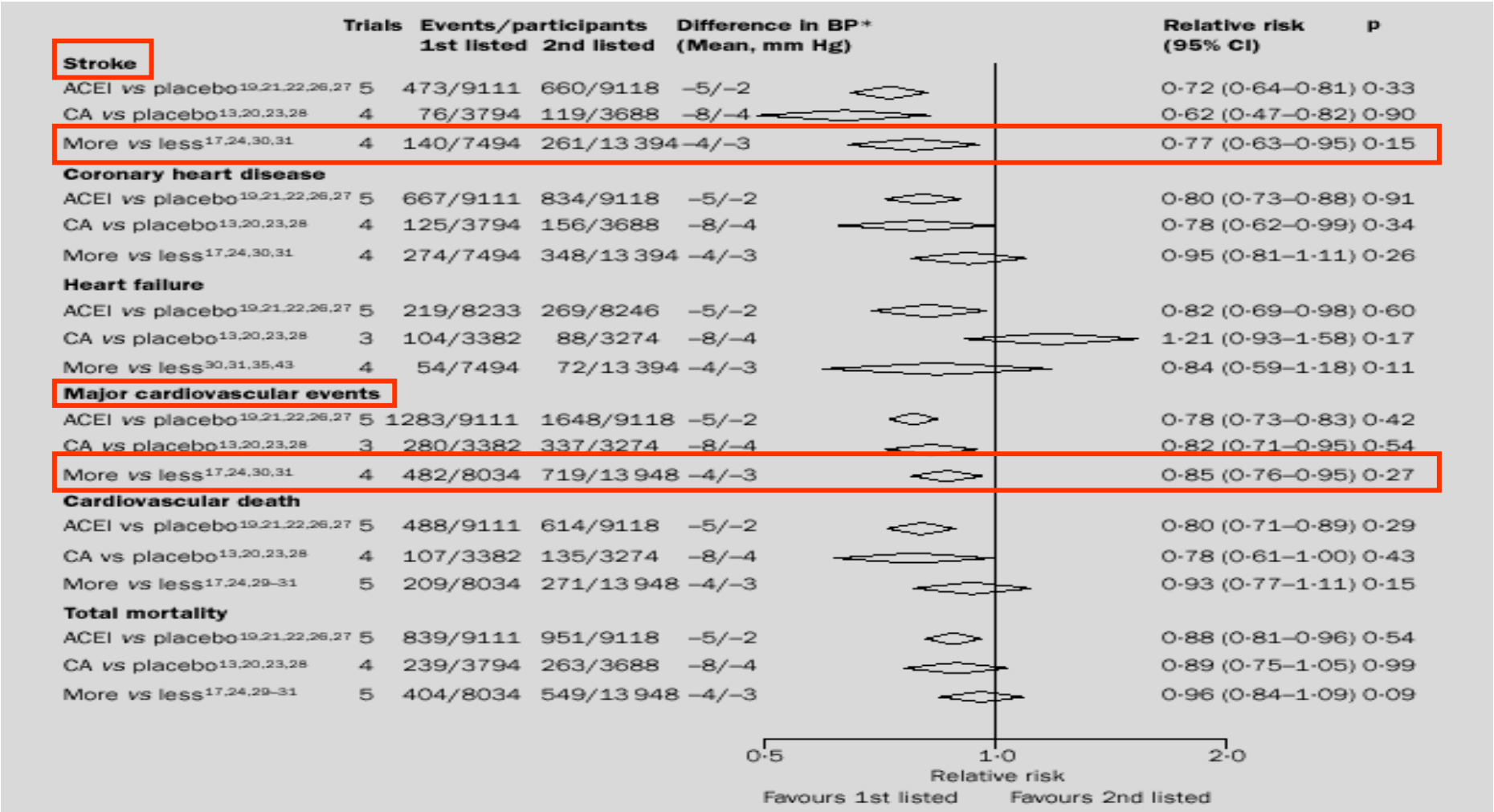
Blood pressure, stroke, and coronary heart disease : Part 2, short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context.

Collins R et al. The Lancet, 1990; 335: 827-838

Nb d'études	**14**
Suivi (ans)	**5**
Nb de sujets (25 à 70 ans)	**36 908**
∆ PAD 5 mmHg	
Réduction AVC	**42 %**
Réduction Ev. Coro.	**14 %**

Effects of different BP-lowering regimens on major CV events: results of prospectively-designed overviews of randomised trials.

BP Lowering Treatment Trialists' Collaboration. Lancet 2003; 362: 1527–35



	Trials	Events/participants 1st listed	2nd listed	Difference in BP* (Mean, mm Hg)	Relative risk (95% CI)	p
Stroke						
ACEI *vs* placebo[19,21,22,26,27]	5	473/9111	660/9118	−5/−2	0·72 (0·64–0·81)	0·33
CA *vs* placebo[13,20,23,28]	4	76/3794	119/3688	−8/−4	0·62 (0·47–0·82)	0·90
More *vs* less[17,24,30,31]	4	140/7494	261/13 394	−4/−3	0·77 (0·63–0·95)	0·15
Coronary heart disease						
ACEI *vs* placebo[19,21,22,26,27]	5	667/9111	834/9118	−5/−2	0·80 (0·73–0·88)	0·91
CA *vs* placebo[13,20,23,28]	4	125/3794	156/3688	−8/−4	0·78 (0·62–0·99)	0·34
More *vs* less[17,24,30,31]	4	274/7494	348/13 394	−4/−3	0·95 (0·81–1·11)	0·26
Heart failure						
ACEI *vs* placebo[19,21,22,26,27]	5	219/8233	269/8246	−5/−2	0·82 (0·69–0·98)	0·60
CA *vs* placebo[13,20,23,28]	3	104/3382	88/3274	−8/−4	1·21 (0·93–1·58)	0·17
More *vs* less[30,31,35,43]	4	54/7494	72/13 394	−4/−3	0·84 (0·59–1·18)	0·11
Major cardiovascular events						
ACEI *vs* placebo[19,21,22,26,27]	5	1283/9111	1648/9118	−5/−2	0·78 (0·73–0·83)	0·42
CA *vs* placebo[13,20,23,28]	3	280/3382	337/3274	−8/−4	0·82 (0·71–0·95)	0·54
More *vs* less[17,24,30,31]	4	482/8034	719/13 948	−4/−3	0·85 (0·76–0·95)	0·27
Cardiovascular death						
ACEI vs placebo[19,21,22,26,27]	5	488/9111	614/9118	−5/−2	0·80 (0·71–0·89)	0·29
CA vs placebo[13,20,23,28]	4	107/3382	135/3274	−8/−4	0·78 (0·61–1·00)	0·43
More *vs* less[17,24,29–31]	5	209/8034	271/13 948	−4/−3	0·93 (0·77–1·11)	0·15
Total mortality						
ACEI *vs* placebo[19,21,22,26,27]	5	839/9111	951/9118	−5/−2	0·88 (0·81–0·96)	0·54
CA *vs* placebo[13,20,23,28]	4	239/3794	263/3688	−8/−4	0·89 (0·75–1·05)	0·99
More *vs* less[17,24,29–31]	5	404/8034	549/13 948	−4/−3	0·96 (0·84–1·09)	0·09

Outcomes in hypertensive patients at high CV risk treated with regimens based on valsartan or amlodipine: the VALUE randomised trial. Weber MA et al. Lancet 2004; 363: 2022–31.

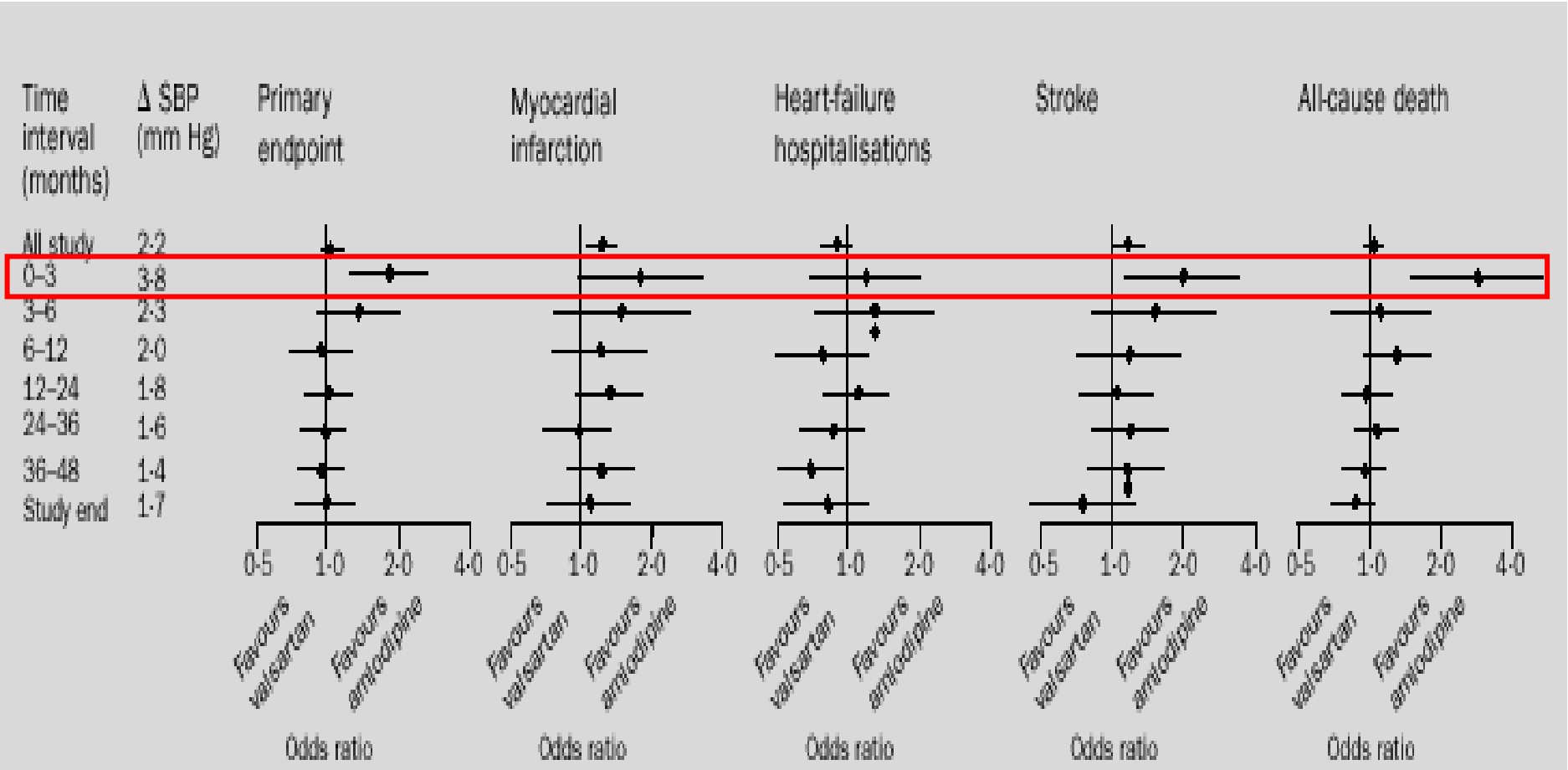
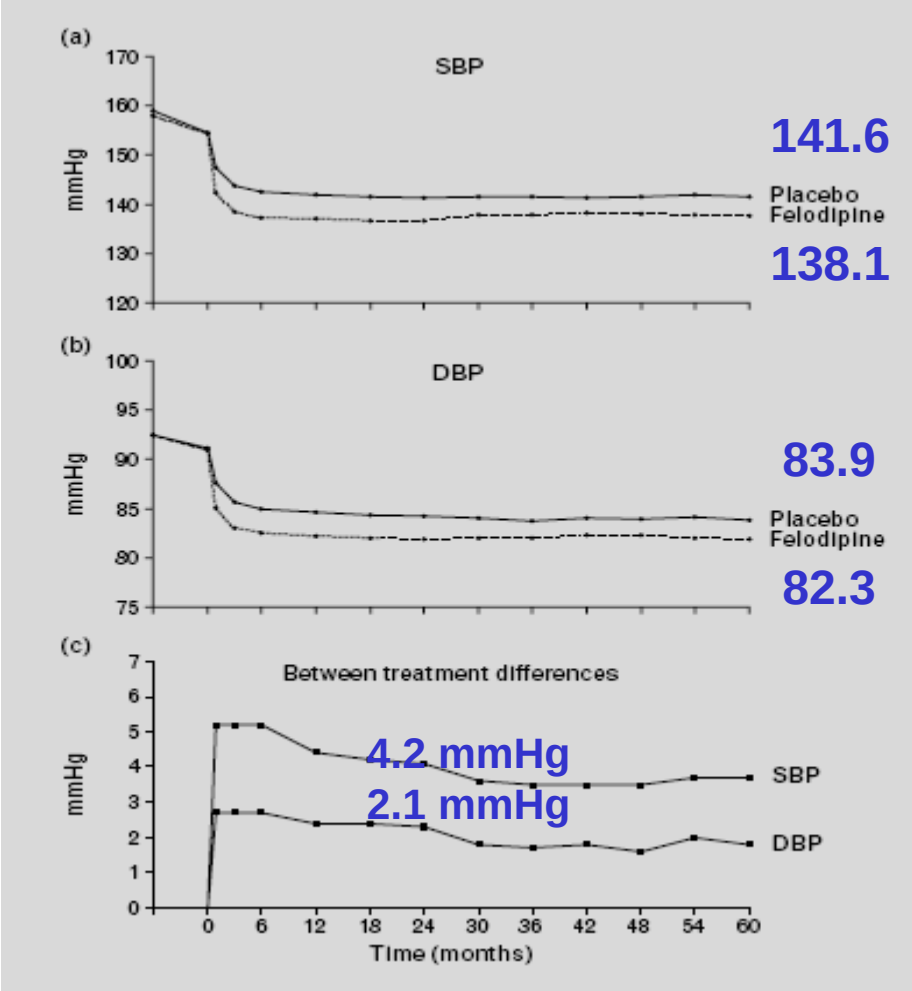
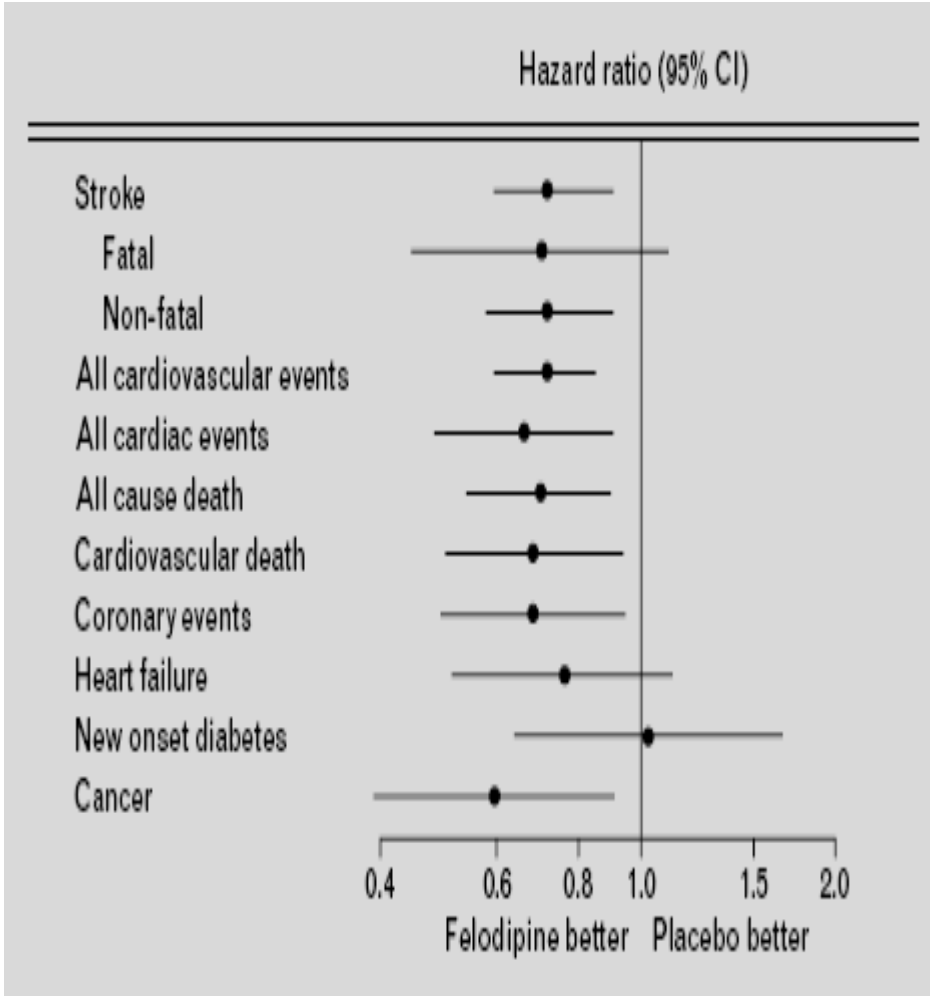


Figure 6: **Differences in BP between treatment groups with odds ratios for primary endpoint, secondary endpoints, and all-cause death during consecutive time periods in the study**

The Felodipine Event Reduction (FEVER) Study: a randomized long-term placebo-controlled trial in Chinese hypertensive patients. *Liu L. J Hypertens 2005; 23:2157–2172*





OBJECTIFS TENSIONNELS : RECOMMANDATIONS

	Général	Diabète	I. rénale
JNC7, 2003	<140/90	<130/80	<130/80
WHO/ISH, 2003	<140	<130/80	
BHS, 2004	140/85	≤130/80	≤130/80
ANAES, 2005	<140/90	<130/80	<130/80
ESH/ESC, 2007	<140/90	<130/80	<130/80

Niveau tensionnel moyen et prévalence de l'hypertension artérielle chez les adultes de 18 à 74 ans, Étude Nationale Nutrition Santé 2006-2007

Godet-Thobie H et al. BEH 16 décembre 2008

Hommes	18-34 ans	35-44 ans	45-54 ans	55-64 ans	65-74 ans	18-74 ans	[IC95 %]
Mesure dans l'année (%)	68,3	86,4	96,5	92,7	97,5	86,5	[83,1-89,9]
Prévalence de l'HTA (%)	4,0	19,5	42,6	62,4	69,9	34,1	[29,8-38,4]
HTA connue* (%)	21,5	22,9	40,5	55,2	59,9	46,9	[39,4-54,5]
HTA connue traitée* (%)	**	55,7	60,3	85,5	91,4	77,4	[67,2-87,6]
HTA traitée contrôlée* (%)	**	**	46,8	43,5	33,9	41,8	[32,3-51,3]
Femmes	**18-34 ans**	**35-44 ans**	**45-54 ans**	**55-64 ans**	**65-74 ans**	**18-74 ans**	**[IC95 %]**
Mesure dans l'année (%)	87,5	88,1	89,5	93,6	95,7	90,2	[87,9-92,6]
Prévalence de l'HTA (%)	5,6	13,1	31,4	43,7	65,0	27,8	[24,7-30,8]
HTA connue* (%)	22,3	55,5	52,9	62,0	68,6	58,8	[52,4-65,2]
HTA connue traitée* (%)	**	60,8	78,4	91,5	94,9	86,6	[81,1-92,1]
HTA traitée contrôlée* (%)	**	**	64	59,4	49,6	58,5	[51,1-65,8]

* HTA connue= proportion d'hypertendus connus parmi les hypertendus.
HTA connue traitée= proportion d'hypertendus traités par médicaments à action antihypertensive parmi les hypertendus connus.
HTA traitée contrôlée= proportion d'hypertendus contrôlés parmi les hypertendus traités.
** Effectifs insuffisants.
Champ : France métropolitaine 18-74 ans.
Source : Étude ENNS, 2006-2007.

Effects of intensive blood-pressure lowering and low-dose aspirin in patients with hypertension: principal results of the HOT randomised trial.

Hansson L et al. Lancet. 1998; 351: 1755-62.

	DBP target group (mmHg)		
	≤ 90	≤ 85	≤ 80
n	6264	6264	6262
Baseline DBP (mmHg)	105.4 (3.4)	105.4 (3.4)	105.4 (3.4)
Difference DBP (mmHg)	20.3 (5.6)	22.3 (5.4)	24.3 (5.8)
Diuretics (step 5) (%)	19	22	24
final DBP > 90 mmHg (%)	12	7	6

HTA RESISTANTE AU TRAITEMENT
DEFINITIONS

• **2007 ESH-ESC guidelines for the management of arterial hypertension.** ***J Hypertens 2007; 25: 1105-1187.*** **« When lifestyle measures and at least three drugs in adequate doses has failed to lower systolic and diastolic BP to goal. »**

• **ESH recommendations for BP measurement.** ***J Hypertens 2003; 21: 821-48.***

« Clinical BP measurement consistently greater than 140/90 mmHg with three antihypertensive drugs… ».

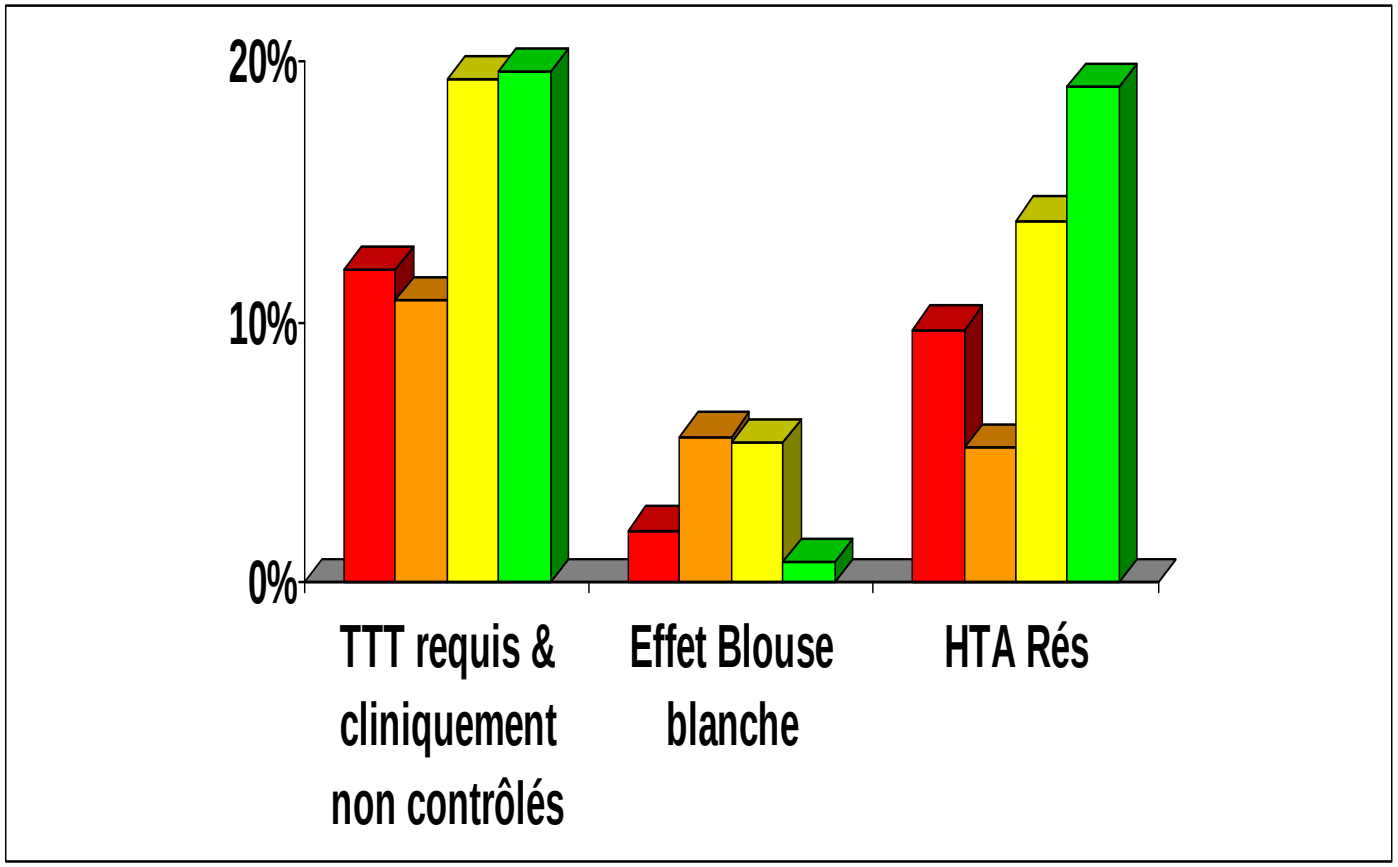
• **The Seventh Report of the Joint National Committee.** ***JAMA 2003; 289: 2560-72.***

« the failure to reach goal BP in patients who are adhering to full doses of an appropriate three-drug regimen that includes a diuretic. »

• **Diagnostic et prise en charge de l'HTA essentielle de l'adulte.** ***ANAES 2005*****.**

« PA restant au-dessus de la cible thérapeutique fixée (le plus souvent 140/90 mmHg) chez un patient traité par une association de 3 médicaments dont un diurétique ou parfois 2 médicaments antihypertenseurs à doses maximales ».

HTA RESISTANTE AU TRAITEMENT
PREVALENCE



ANALYSE ET TRAITEMENT D'UNE HTA RESISTANTE

LE MALADE

Characteristics of patients with uncontrolled patients in the United States

Hyman DJ, Pavlik VN. N Engl J Med 2001; 345: 479-86.

TABLE 4. RESULTS OF MULTIVARIATE ANALYSIS OF PREDICTORS OF THE LACK OF AWARENESS AND LACK OF CONTROL OF HYPERTENSION IN THE U.S. POPULATION.*

VARIABLE	LACK OF AWARENESS OF CONDITION†		ACKNOWLEDGED, UNCONTROLLED HYPERTENSION‡	
	ODDS RATIO (95% CI)	P VALUE	ODDS RATIO (95% CI)	P VALUE
Age ≥65 yr (vs. <65 yr)	7.69 (5.88–9.09)	<0.001	2.08 (1.64–2.53)	<0.001
Male sex (vs. female sex)	1.57 (1.36–1.82)	<0.001	1.30 (1.02–1.65)	0.03
Race or ethnic group (vs. non-Hispanic white)				
Non-Hispanic black	1.45 (1.18–1.79)	0.001	1.24 (0.97–1.59)	0.08
Mexican American	0.86 (0.66–1.13)	0.28	1.17 (0.92–1.48)	0.20
High-school graduation (vs. no high-school graduation)	0.87 (0.69–1.09)	0.21	1.00 (0.75–1.33)	0.98
Family income (vs. ≥$50,000/yr)				
<$20,000/yr	1.25 (0.90–1.74)	0.18	1.03 (0.69–1.55)	0.87
$20,000–$49,999/yr	1.06 (0.82–1.38)	0.63	1.26 (0.93–1.73)	0.14
Has health insurance (vs. has no health insurance)	0.91 (0.61–1.34)	0.62	1.30 (0.79–2.13)	0.29
Has a usual source of care (vs. has no usual source of care)	1.12 (0.87–1.43)	0.38	1.07 (0.63–1.84)	0.79
No visits to physician in past 12 mo (vs. ≥1 visits in past 12 mo)	1.41 (1.14–1.75)	0.002	1.89 (1.09–3.29)	0.03
Current smoking (vs. nonsmoking)	0.78 (0.62–0.98)	0.04	1.20 (0.92–1.57)	0.17

*Data are from phases I and II (1988 to 1994) of the third National Health and Nutrition Examination Survey. CI denotes confidence interval.

†The model included a total of 10,576 persons: 8928 persons without hypertension and 1648 who had hypertension but who were unaware of their condition.

‡The model included 3516 persons: 1117 with acknowledged, untreated hypertension and 2399 with treated hypertension.

Predictors of Uncontrolled Hypertension in Ambulatory Patients.

Knight EL et al. Hypertension 2001; 38: 809-814.

525 hypertendus, analyse multivariée

Variable	Odds of Poor Control	95% Confidence Interval
Age group*		
55–64 y	1.26	0.71–2.24
65–74 y	2.50	1.49–4.19
≥75 y	2.56	1.45–4.52
Site**		
VAMC	0.63	0.40–1.01
Hospital	0.94	0.42–2.11
No. of antihypertensive drugs during the study period***		
0	0.90	0.41–1.98
2	1.91	1.25–2.91
3	2.53	1.50–4.28
4 or 5	4.70	2.22–9.95
Angina	0.33	0.20–0.56
Lack of knowledge of appropriate SBP	1.55	1.09–2.20
Attributed a specific side effect to a specific antihypertensive medication	2.06	1.41–3.01

Adherence to Medication

Osterberg L, Blaschke T. N Engl J Med 2005; 353: 487-97.

« Drugs don't work in patients who don't take them ». **C. Everett Koop.**

Major Predictors of Poor Adherence to Medication

- Presence of psychological problems, particularly depression
- Presence of cognitive impairment
- Treatment of asymptomatic disease
- Inadequate follow-up or discharge planning
- Side effects of medication
- Patient's lack of belief in benefit of treatment
- Patient's lack of insight into the illness
- Poor provider–patient relationship
- Presence of barriers to care or medications
- Missed appointments
- Complexity of treatment
- Cost of medication, copayment, or both

Adherence to Medication

Osterberg L, Blaschke T. N Engl J Med 2005; 353: 487-97.

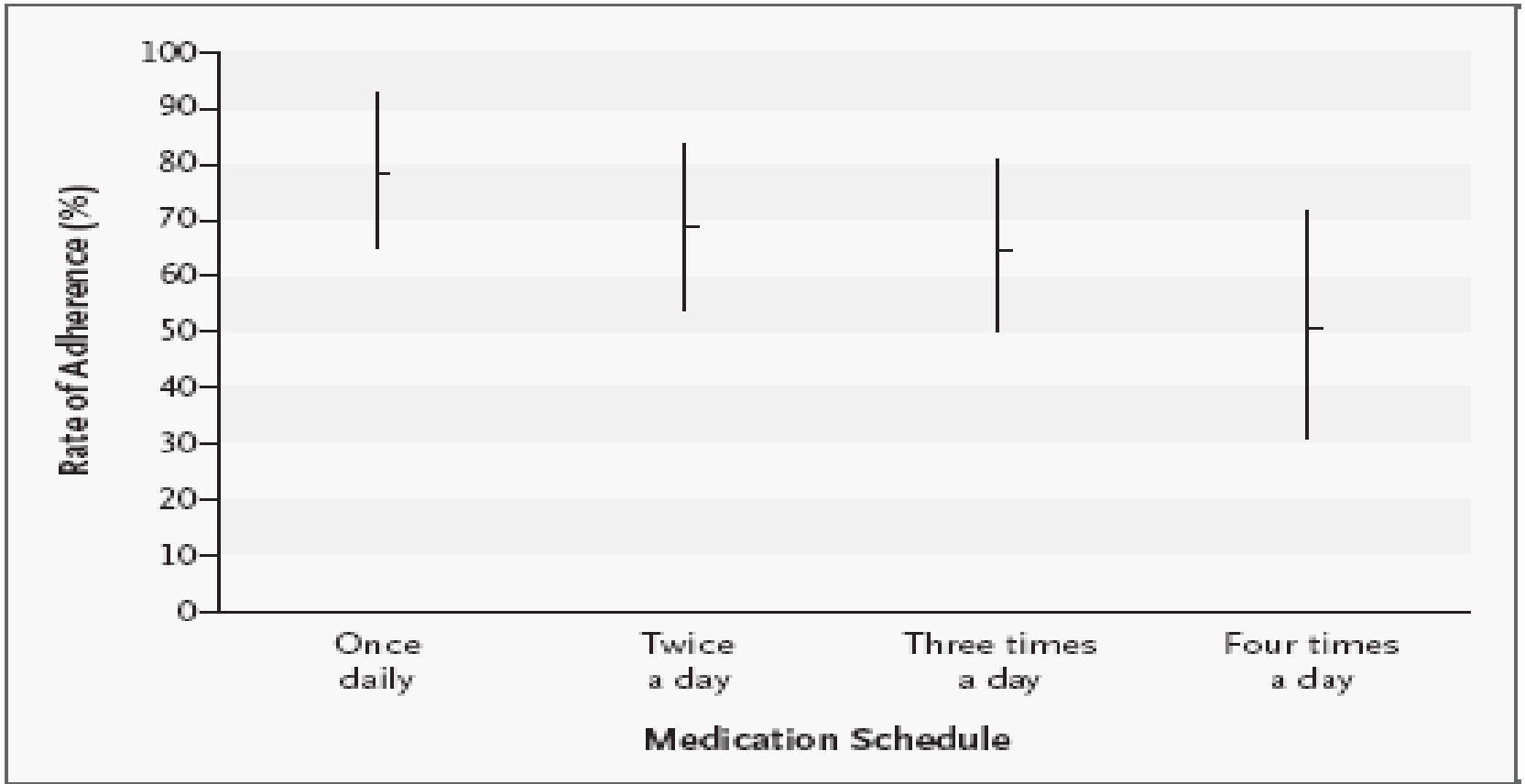


Figure 1. Adherence to Medication According to Frequency of Doses.

Vertical lines represent 1 SD on either side of the mean rate of adherence (horizontal bars). Data are from Claxton et al.[7]

Electronic monitoring of patient adherence to oral antihypertensive medical treatment: a systematic review

Christensen A et al. J Hypertens 2009; 27: 1540–1551

Table 2 Studies with feedback of electronic monitoring data to patients aiming to improve adherence

Reference	Participants receiving feedback (*n*)	Study length (weeks)	Adherence measures (%)	BP (mmHg)**	Change in BP during intervention (mmHg)
Baulmann *et al.* [30]	1	>20	Dosing b 50, a 91; timing b 17, a 76	b 190/80, a 137/71	−53/−9
Bertholet *et al.* [31]	69	4-9	Dosing 92	b 159/104, a 143/92; BP normalized in 33% of patients	−16/−12 ($P=0.001$)
Braam *et al.* [35]	30	24	Taking, cutoff 80:86	b 158/105, a 148/97	−10/−8 (<0.05)
Burnier *et al.* [36]	37	13–22	Dosing >90	b 156/106, a 145/97	−11/−9 ($P<0.01$)
Chiolero *et al.* [37]	1	>56	Taking 0–90	b 188/102, a 136/76	−52/−26
Kruse *et al.* [49]	24	30	Taking 89, 2/d 88; dosing 85, 2/d 80	Improved compliance led to reduced BP	Reduced
Mengden *et al.* [60]	24	12	Taking b 91, a 100; dosing b 78, a 97	SBPM b 154/84, a 145/80	−9/−4 ($P<0.01$)
Santschi *et al.* [67]***	21 (34)	52	Taking 97% throughout	Various	Reduced (NS)
Wetzels *et al.* [76]***	164	8	Refill b 81, dosing a 95	b 169/96, a 153/86; 3.1% more patients had normalized BP in intervention group; more dose escalations in usual care group	−14/−10
Mean	46.6	22			Adjusted average BP reduction: −13.6/−9.7****

Compliance values are given for once-daily dosing unless stated otherwise. 2/d, twice daily; BP, blood pressure; SBPM, self blood pressure measurement; SD, standard deviation. **Values represent office BP measurements unless stated otherwise. ***Feedback was given to the treating physician; b, before intervention; a, after intervention. **** SD not available as not reported in the article.

Relation between insufficient response to antihypertensive treatment and poor compliance with treatment: a prospective case-control study

Nuesch R et al. BMJ 2001; 323: 142–6

Table 2 Characteristics of 103 hypertensive patients by their responsiveness* to antihypertensive treatment. Values are numbers (percentages) unless stated otherwise

Characteristic	Responsive (n=54)	Non-responsive (n=49)
Compliant with treatment†	46 (85)	40 (82)
Percentage of doses taken:		
Mean (SD)	91 (19)	88 (18)
Median (range)	98 (11-100)	96 (11-100)
Mean (SD) age (years)	65 (10.4)	62 (9.5)
Male to female ratio	0.67	0.72
Mean (SD) body mass index (kg/m^2)	26.6 (4.5)	27.0 (3.5)
Tobacco smoker	10 (19)	9 (18)
Drink alcohol (>3 units/day)‡	3 (6)	3 (6)
Family history of hypertension	32 (59)	36 (73)
Diabetes	8 (15)	3 (6)
Hyperlipidaemia	19 (35)	13 (27)

*Patients responsive to treatment if 12 hour ambulatory blood pressure <135/85 mm Hg if aged ≤60 or <155/90 mm Hg if aged >60.
†Patients compliant if ≥80% of prescribed doses taken correctly.
‡1 unit defined as one standard drink.

A Systematic Review of the Effects of Home Blood Pressure Monitoring on Medication Adherence

Ogedegbe G and Schoenthaler A. J Clin Hypertens. 2006; 8: 174–180

Table I. Characteristics of Studies Included in the Systematic Review

Study (year)	Duration of Intervention	N	Completed Follow-Up (%)		Adherence Measure	Statistical Improvement in Adherence
			Intervention	Control		
Bailey et al.[6] (1999)	8 wk	62	97	97	Pill count	No
Binstock et al.[8] (1988)	1 yr	111	100	100	Self-report	No
Friedman et al.[11] (1996)	6 mo	267	85	92	Pill count	Yes
Haynes et al.[13] (1976)	6 mo	38	100	95	Pill count	Yes
Girvin et al.[12] (2004)	6 mo	136	97	97	Pill count	No
McKenney et al.[15] (1992)	24 wk	67	94	97	Electronic monitoring device	Yes
Mehos et al.[16] (2000)	6 mo	36	98	97	Pharmacy records	No
Ogbuokiri[18] (1980)	5 mo	24	79*		Pill count	Yes
Rudd et al.[19] (2004)	6 mo	150	94	91	Electronic monitoring device	Yes
Vrijens and Goetghebeur[22] (1997)	6 wk	628	n/a	n/a	Electronic monitoring device	Yes
Zarnke et al.[23] (1997)	8 wk	31	100	98	Self-report	No

n/a=not applicable; *lost to follow-up not differentiated among conditions

Of the 11 RCTs, six (54%) reported statistically significant improvement in medication adherence attributed to the intervention. Five of these six studies were complex interventions.

Influence of Weight Reduction on Blood Pressure
A Meta-Analysis of Randomized Controlled Trials

Neter J et al. Hypertension. 2003; 42: 878-884.

An average net weight reduction of 5.1 kg was associated with a reduction in SBP of 4.44 mm Hg (95% CI, 5.93-2.95) and in DBP of 3.57 mm Hg (95% CI, 4.88-2.25)

TABLE 2. Changes in SBP and DBP in 25 RCTs of Weight Reduction and BP, Overall and in Subgroups

		SBP, mm Hg†		DBP, mm Hg†	
Stratum	No. of Strata*	Unadjusted	Adjusted‡	Unadjusted	Adjusted‡
Overall	34	−4.44 (−5.93; −2.95)	−4.78 (−5.76; −3.80)	−3.57 (−4.88; −2.25)	−3.56 (−4.31; −2.81)
Age					
≤45 years	15	−4.19 (−6.19; −2.20)	−4.74 (−6.35; −3.12)	−3.17 (−5.04; −1.31)	−3.69 (−4.96; −2.43)
>45 years	19	−4.74 (−6.95; −2.52)	−4.80 (−6.48; −3.13)	−3.94 (−5.76; −2.12)	−3.43 (−4.63; −2.23)
Gender					
<50% females	21	−4.75 (−6.54; −2.97)	−5.05 (−6.10; −3.99)	−4.04 (−5.61; −2.48)	−3.89 (−4.66; −3.12)
≥50% females	13	−3.74 (−6.40; −1.07)	−3.91 (−5.69; −2.13)	−2.53 (−4.82; −0.24)	−2.50 (−3.93; −1.08)
Hypertension§					
No	17	−4.08 (−6.01; −2.16)	−4.46 (−5.71; −3.21)	−2.35 (−4.05; −0.65)	−2.62 (−3.83; −1.42)
Yes	17	−4.95 (−7.25; −2.64)	−4.73 (−6.40; −3.06)	−4.92 (−6.73; −3.12)	−4.36 (−5.72; −3.00)
Race‖					
White	14	−3.19 (−4.79; −1.59)	...	−2.50 (−3.00; −1.99)	...
Black	4	−4.67 (−8.86; −0.49)	...	−3.08 (−4.92; −1.23)	...
Asian	4	−8.77 (−11.91; −5.64)	...	−9.81 (−11.17; −8.44)	...
Intervention					
Energy restriction	19	−4.93 (−6.84; −3.02)	−4.33 (−5.70; −2.97)	−4.25 (−5.95; −2.55)	−2.84 (−3.80; −1.87)
Physical activity	6	−1.73 (−5.14; 1.69)	−4.74 (−7.60; −1.88)	−1.93 (−5.07; 1.22)	−4.65 (−6.84; −2.45)
Combined intervention	7	−5.15 (−7.78; −2.51)	−5.66 (−7.52; −3.81)	−3.12 (−5.60; −0.64)	−4.44 (−5.68; −3.19)
Initial BMI					
<30 kg/m²	15	−4.14 (−4.95; −3.33)	−4.59 (−5.70; −3.49)	−2.61 (−3.29; −1.93)	−3.11 (−4.01; −2.21)
≥30 kg/m²	13	−4.09 (−4.87; −3.31)	−4.05 (−5.06; −3.05)	−2.75 (−3.39; −2.11)	−2.77 (−3.50; −2.04)
Weight reduction					
≤5 kg	16	−2.44 (−4.38; −0.49)	−2.70 (−4.59; −0.81)	−1.97 (−3.71; −0.21)	−2.01 (−3.47; −0.54)
>5 kg	18	−6.24 (−8.06; −4.41)	−6.63 (−8.43; −4.82)	−4.97 (−6.62; −3.31)	−5.12 (−6.48; −3.75)
Antihypertensive drugs¶					
No	26	−3.77 (−5.33; −2.22)	−4.11 (−5.23; −3.00)	−2.97 (−4.39; −1.55)	−2.91 (−3.66; −2.16)
Yes	8	−7.00 (−10.02; −3.98)	−6.70 (−8.71; −4.69)	−5.49 (−8.06; −2.93)	−5.31 (−6.64; −3.99)

Association between refractory hypertension and obstructive sleep apnea

Ruttanaumpawana P et al. J Hypertens 2009; 27: 1439–1445

Table 2 Baseline polysomnographic data

	Controlled hypertension ($n=22$)	Refractory hypertension ($n=42$)	P
OSA, n (%)	12 (55)	34 (81)	0.03
AHI (numbers of hours of sleep)	16.5 ± 2.7	24.9 ± 3.2	0.13
Mean SaO_2 (%)	94.1 ± 0.5	94.5 ± 0.3	0.49
Lowest SaO_2 (%)	83.8 ± 1.7	84.0 ± 1.1	0.91
Time in bed (min)	406.9 ± 8.7	396.1 ± 11.9	0.46
Sleep onset latency (min)	15.0 ± 2.5	25.4 ± 4.4	0.30
Total sleep time (min)	321.4 ± 9.5	281.9 ± 14.1	0.02
Wake after sleep onset (min)	70.6 ± 6.7	84.7 ± 8.8	0.51
Sleep efficiency (%)	79.0 ± 1.7	69.7 ± 3.0	0.01
Stages 1 and 2 sleep (min)	219.9 ± 8.7	207.3 ± 10.4	0.43
Slow wave sleep (min)	38.3 ± 5.1	27.6 ± 4.0	0.11
REM sleep (min)	63.2 ± 4.9	47.0 ± 4.5	0.02
Arousal index (number per hour of sleep)	19.2 ± 2.6	26.8 ± 3.3	0.11

Values are expressed as means ± SEM. AHI, apnea–hypopnea index; OSA, obstructive sleep apnea; REM, rapid eye movement; SaO_2, oxygen saturation.

Table 4 Odds of having refractory hypertension, multivariate logistic regression

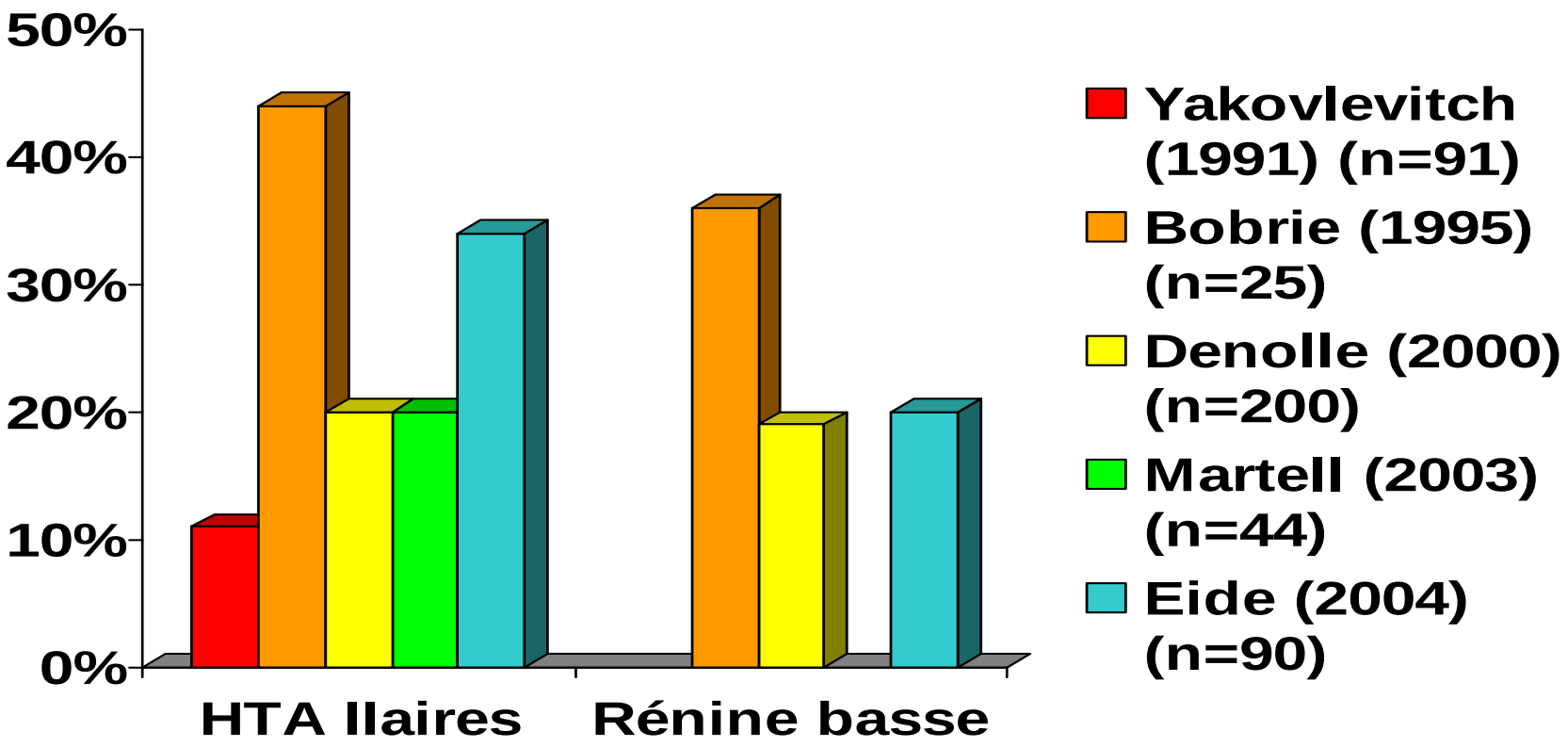
Factors	OR	95% CI	P
Presence of OSA	3.994	1.191–13.388	0.02
Reduced REM sleep time (min)	1.025	1.002–1.049	0.03

Variables included in the multivariate analysis were the presence of OSA, reduced total sleep time, reduced sleep efficiency and reduced REM sleep. CI, confidence interval; OR, odds ratio; OSA, obstructive sleep apnea; REM, rapid eye movement.

ANALYSE ET TRAITEMENT D'UNE HTA RESISTANTE

LA MALADIE HYPERTENSIVE

HTA RESISTANTE AU TRAITEMENT
HTA SECONDAIRES



NEPHROPATHIE ET I. RENALE

STENOSE ARTERIELLE RENALE

HAP & HYPERCORTICISME & PHEOCHROMOCYTOME

Lifestyle interventions to reduce raised blood pressure: a systematic review of randomised controlled trials.

Dickinson HO et al. J Hypertens 2006; 24: 215–233.

	Net reduction in blood pressure (mmHg)														
			Systolic blood pressure (SBP)				Diastolic blood pressure (DBP)				Withdrawals[a]			
Type of intervention	*n*	*N*	MD	(95% CI)	I^2	Size, *P*	MD	(95% CI)	I^2	Size, *P*	*n*	RD	(95% CI)	I^2
Diet	14	1339	−6.0	(−8.6 to −3.4)	72%	0.49	−4.8	(−6.9 to −2.7)	81%	0.25	12	0.04	(−0.02 to 0.09)	65%
Diet (excl. [28])	13	1256	−5.0	(−7.0 to −3.1)	52%	0.81	−3.7	(−5.1 to −2.4)	52%	0.59	12	0.04	(−0.02 to 0.09)	65%
Exercise	21	1346	−6.1	(−10.1 to −2.1)	87%	0.57	−3.0	(−4.9 to −1.1)	74%	0.45	17	0.03	(−0.01 to 0.08)	19%
Exercise (excl. [49])	20	1270	−4.6	(−7.1 to −2.0)	65%	0.13	−2.4	(−4.0 to −0.7)	58%	0.21	16	0.04	(−0.01 to 0.08)	26%
Relaxation	23	1231	−4.0	(−6.4 to −1.6)	62%	0.93	−3.1	(−4.7 to −1.5)	70%	0.68	12	0.04	(−0.01 to 0.09)	38%
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Calcium supplements	13	461	−2.5	(−4.4 to −0.6)	42%	0.90	−0.8	(−2.1 to 0.4)	48%	0.64	4	0.00	(−0.06 to 0.06)	0%
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Fish oil supplements	8	375	−2.3	(−4.3 to −0.2)	0%	0.10	−2.2	(−4.0 to −0.4)	34%	0.03	5	0.02	(−0.04 to 0.07)	28%

n, Number of included trials; *N*, number of participants assessed; MD, mean difference between treatment and control; CI, confidence interval; I^2, % of variation between trials not explained by sampling variation [11]; Size, *P*, *P* value for relationship between treatment effect and size of trial [12]; RD, risk difference. *, Not enough trials. [a]For parallel trials only.

Resistant Hypertension. Comparing Hemodynamic Management to Specialist Care.

Taler SJ et al. Hypertension 2002; 39: 982-988.

104 resistant hypertension patients randomized to drug selection:
• based on serial hemodynamic measurements (thoracic bioimpedance) and a predefined algorithm,
• directed by a hypertension specialist,
in a 3-month intensive treatment program.

Cardiac index	Systemic vascular resistance index	Medication choices
Low	high	1. Add or increase C, A or direct vasodilator 2. Reduce B 3. Evaluate TBI: if reduced, add or intensify D
high	low	1. Add B or central agonist 2. Reduce vasodilators 3. Evaluate TBI: if reduced, add or intensify D
normal	normal	Evaluate TBI: if reduced, add or intensify D

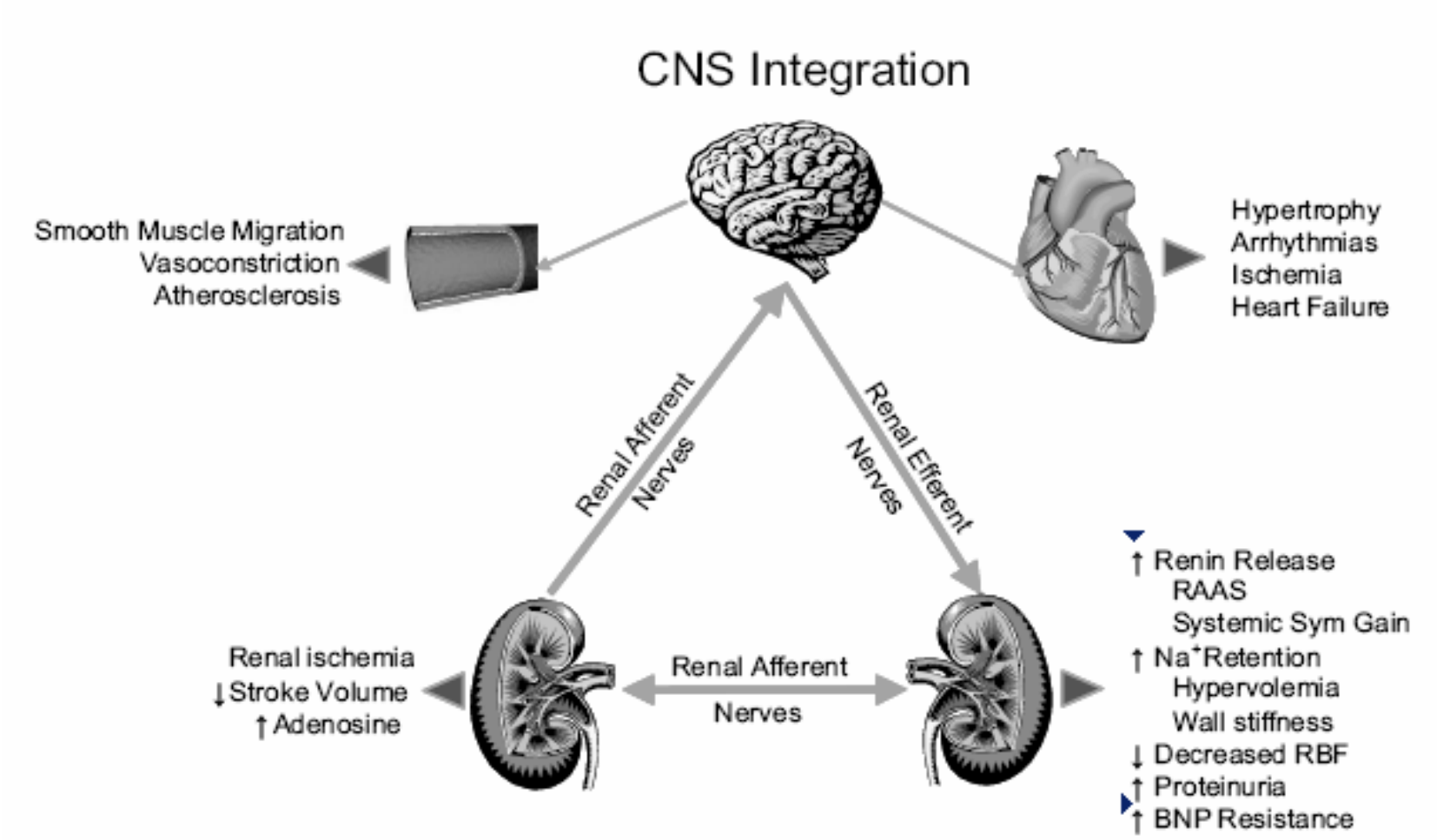
Resistant Hypertension. Comparing Hemodynamic Management to Specialist Care.

Taler SJ et al. Hypertension 2002; 39: 982-988.

	Hemodynamic care	p	Specialist care
n			
Age, y	67 ± 2		64 ± 2
BMI, kg/m²	31.4 ± 1.0		32.7 ± 1.2
Diabetes mellitus	16 (32)		18 (33)
BP, mmHg	169 ± 3 / 87 ± 2		173 ± 3 / 91 ± 2
HR, bpm	66 ± 1	*	72 ± 2
No. of medications	3.6 ± 0.1		3.6 ± 0.1
DDD	1.1 ± 0.1		1.2 ± 0.2
renal artery stenosis	6 (12)		8 (15)
primary aldosteronsim	3 (6)		4 (7)
obstructive sleep apnea	9 (18)		11 (20)
	After 3 months of treatment		
BP, mmHg	139 ± 2 / 72 ± 1	*/*	147 ± 2 / 79
No. of medications	3 ± 0.1	*	4.1 ± 0.1
DDD	2.1 ± 0.2	*	1.4 ± 0.1
Control ≤ 140/90 mmHg	28 (56)	*	18 (33)

Renal Denervation as a Therapeutic Approach for Hypertension Novel Implications for an Old Concept

Schlaich MP et al. Hypertension. 2009; 54:



Catheter-based renal sympathetic denervation for resistant hypertension: a multicentre safety and proof-of-principle cohort study

***Krum H et al. Lancet* 2009; 373: 1275–81.**

Diffuse visceral non-radiating abdominal pain.
No complication in 43 of 45 pts.
One renal artery dissection.
One pseudoaneurysm at the femoral access site.

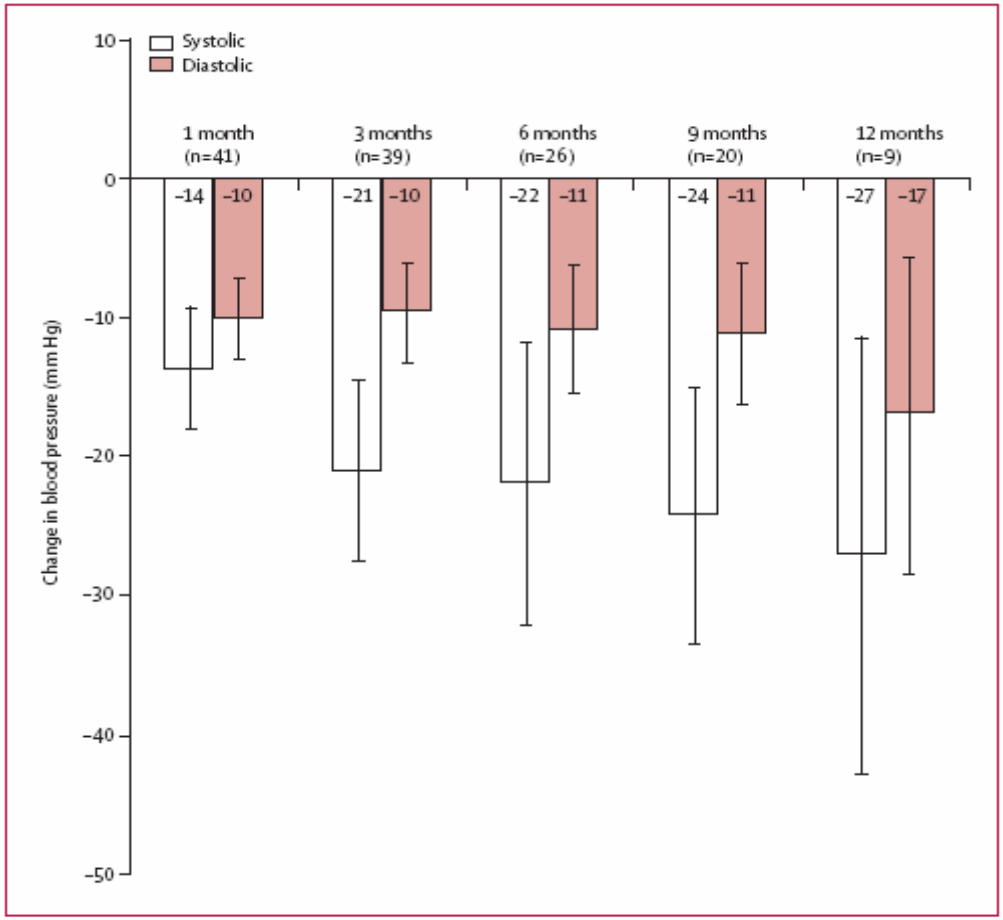


Figure 2: Change in office blood pressure (95% CI) at 1, 3, 6, 9, and 12 months
Numbers in parentheses indicate patients who had attended each predefined visit at the time of submission of this publication.

Management of Uncontrollable Hypertension With a Carotid Sinus Stimulation Device

Mohaupt MG et al. Hypertension. 2007; 50: 825-828.

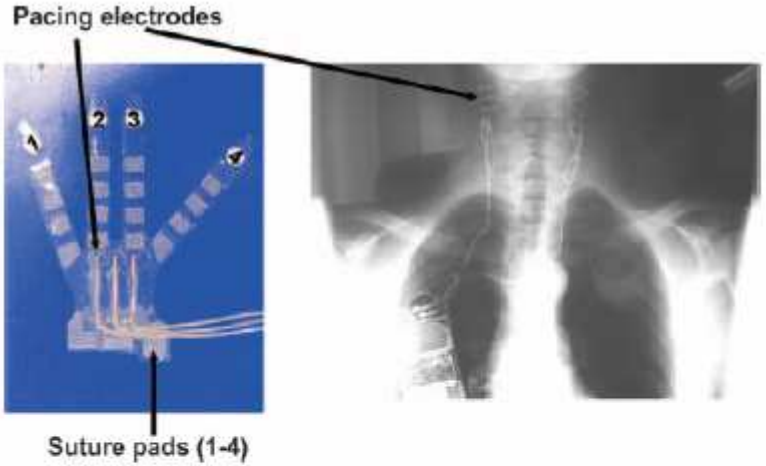


Figure 1. A, Electrode system that is implanted on both carotid sinuses is shown. The adventia is stimulated directly. Pacing electrodes and suture pads of the electrodes are prepared to accommodate placement close to the carotid bifurcation. B, Chest roentgenogram after implantation showing the electrodes in place and the stimulator that is somewhat larger than a conventional pacemaker.

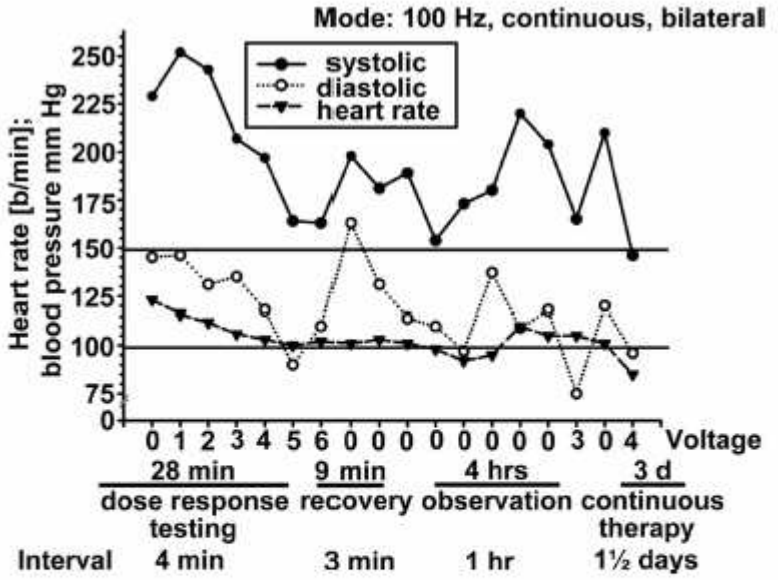


Figure 2. Dinamap blood pressure measurements of the patient during a hypertensive crisis are shown. Systolic blood pressure decreased >45 mm Hg, and diastolic blood pressure decreased 50 mm Hg. Thereafter, the device was shut off, and blood pressure increased over 4 hours. Continuation of the stimulus resulted in blood pressure decreases to the previous stimulation values. Voltage is indicated on the *x* axis. The stimulation was bilateral with on a continuous square-wave pattern at a frequency of 100 Hz and a pulse width of 480 μs.

Effects of Chronic Baroreceptor Stimulation on the Autonomic Cardiovascular Regulation in Patients With Drug-Resistant Arterial Hypertension

Wustmann K et al. Hypertension. 2009; 54: 530-536.

Table 2. Twenty-Four–Hour HRV Time-Domain and Frequency-Domain Measures and Office BP Values in 21 Patients Before and With Chronic Electrical Baroreflex Stimulation

Variable	Stimulator Off	Stimulator On	*P*
Time-domain measures			
Heart rate, 1/min	81±11	76±10	0.001
R-R intervals, ms	743±182	818±107	0.002
SDNN, ms	89±20	95±23	0.2
SDNN index, ms	37±10	44±13	<0.001
pNN50, %	1.3 (2.2)	2.6 (4.8)	<0.001
RMSSD, ms	18.6±6.7	24.3±9.5	<0.001
Frequency-domain measures (FFT)			
HF power, ms^2	42 (59)	67 (105)	<0.001
LF power, ms^2	150 (196)	117 (135)	<0.001
Ratio LF:HF	2.78 (2.75)	2.24 (2.09)	<0.001
Office BP			
Systolic BP, mm Hg	184±31	154±23	<0.001
Diastolic BP, mm Hg	109±23	95±15	0.002

SDNN index indicates the mean of the SDNN intervals for all 5-minute segments in 4 hours. Data are mean±SD or median (mean).

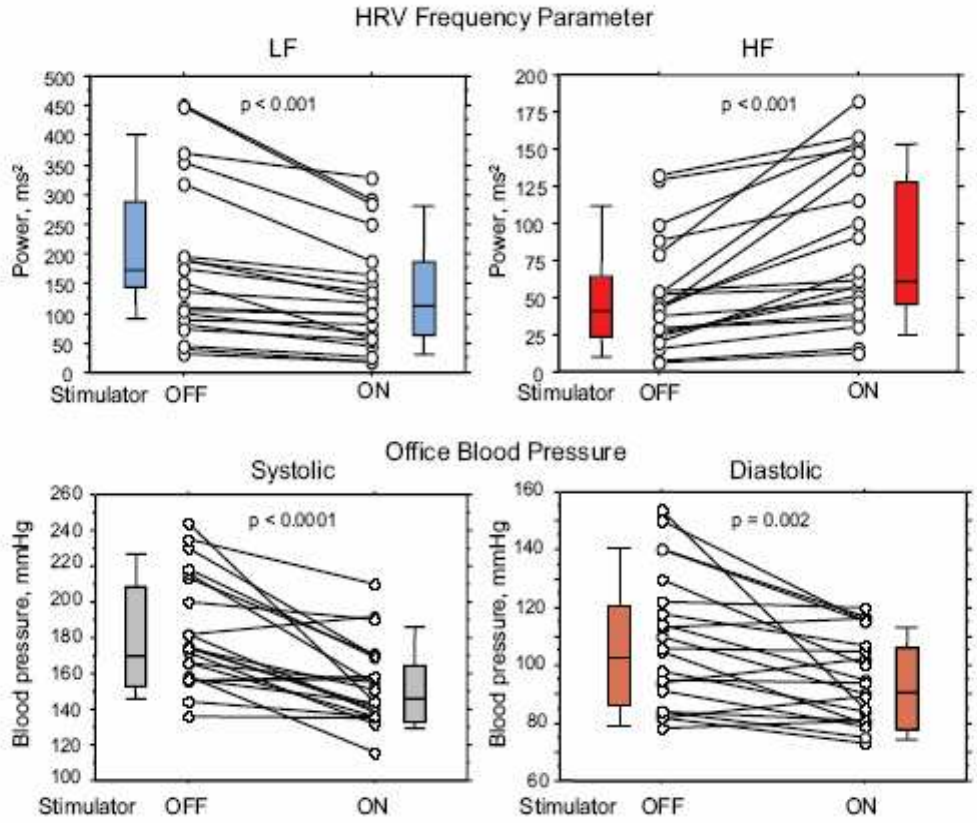


Figure 1. Top, Effect of chronic electric baroreceptor stimulation on LF and HF power, reflecting significant changes in the sympatho-vagal activity consistent with an enhanced vagal outflow and a decreased sympathetic activity during chronic carotid receptor stimulation (ON) vs the control without stimulation (OFF). Bottom, Effect of chronic electric baroreceptor stimulation on office systolic (left) and diastolic (right) BPs.

A selective endothelin-receptor antagonist to reduce BP in patients with treatment-resistant hypertension.

Weber MA et al. Lancet 2009; 374: 1423–31

	Placebo (n=132)	Darusentan 50 mg (n=81)	Darusentan 100 mg (n=81)	Darusentan 300 mg (n=85)
Age (years)				
<65 (n=233)	-8·8 (1·5)	-16·4 (2·3); p=0·0020	-17·2 (2·4); p=0·0014	-19·5 (2·4); p<0·0001
≥65 (n=146)	-8·3 (2·1)	-16·8 (2·3); p=0·0091	-19·4 (2·9); p=0·0038	-15·8 (3·4); p=0·0554
Sex				
Women (n=191)	-9·9 (1·8)	-19·9 (2·4); p=0·0002	-18·3 (2·9); p=0·0035	-20·2 (2·8); p=0·0009
Men (n=188)	-7·1 (1·7)	-13·5 (2·3); p=0·0222	-18·0 (2·3); p=0·0003	-16·1 (2·7); p=0·0036
Comorbidity status				
Diabetes (n=153)	-7·2 (1·9)	-13·7 (2·3); p=0·0104	-18·4 (3·1); p=0·0013	-13·4 (3·0); p=0·0724
CKD (n=96)	-7·6 (2·4)	-11·1 (4·1); p=0·2722	-17·5 (4·0); p=0·0292	-16·0 (4·3); p=0·0514
Neither diabetes nor CKD (n=176)	-10·1 (1·9)	-18·9 (2·6); p=0·0051	-17·8 (2·3); p=0·0114	-22·2 (2·8); p=0·0003
Number of background antihypertensive drugs				
Exactly three (n=159)	-8·7 (1·6)	-15·1 (2·6); p=0·0136	-19·8 (3·1); p=0·0007	-18·3 (2·7); p=0·0009
≥Four (n=220)	-8·5 (1·8)	-17·4 (2·2); p=0·0017	-17·1 (2·3); p=0·0040	-17·9 (2·8); p=0·0070

Data are mean (SE). p values indicate changes from baseline compared with placebo. CKD=chronic kidney disease.

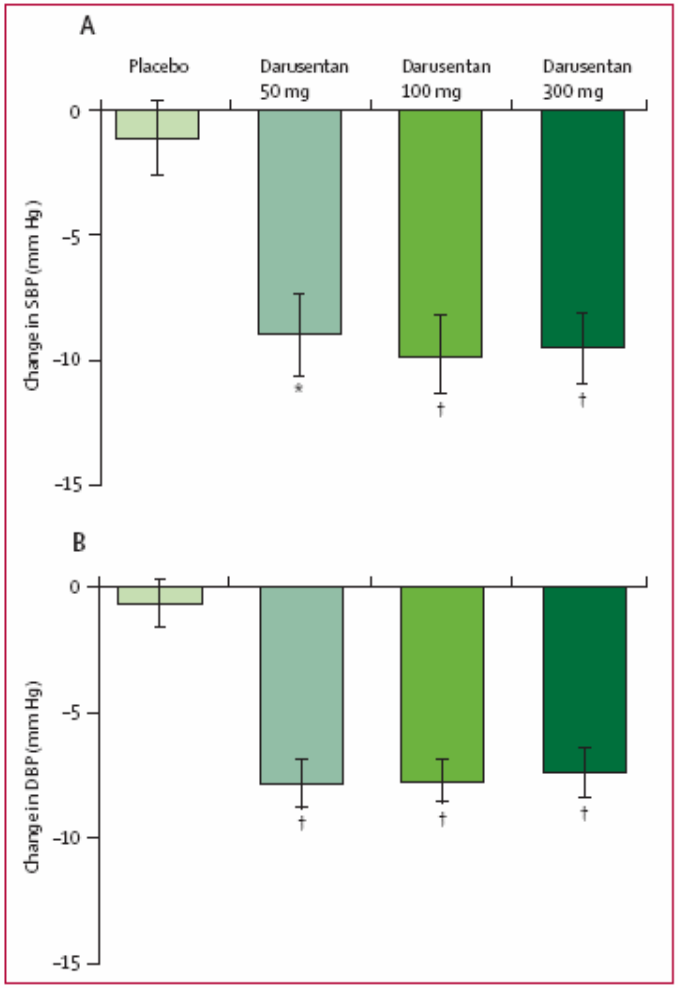


Figure 3: Changes from baseline in mean 24-h ambulatory blood pressure after 14 weeks

Oedema or fluid retention occurred in 67 (27%) patients given darusentan compared with 19 (14%) given placebo. One patient in the placebo group died (sudden cardiac death), and 5 patients in the 3 darusentan dose groups combined had cardiac-related SAE.

ANALYSE ET TRAITEMENT D'UNE HTA RESISTANTE

LES MEDICAMENTS

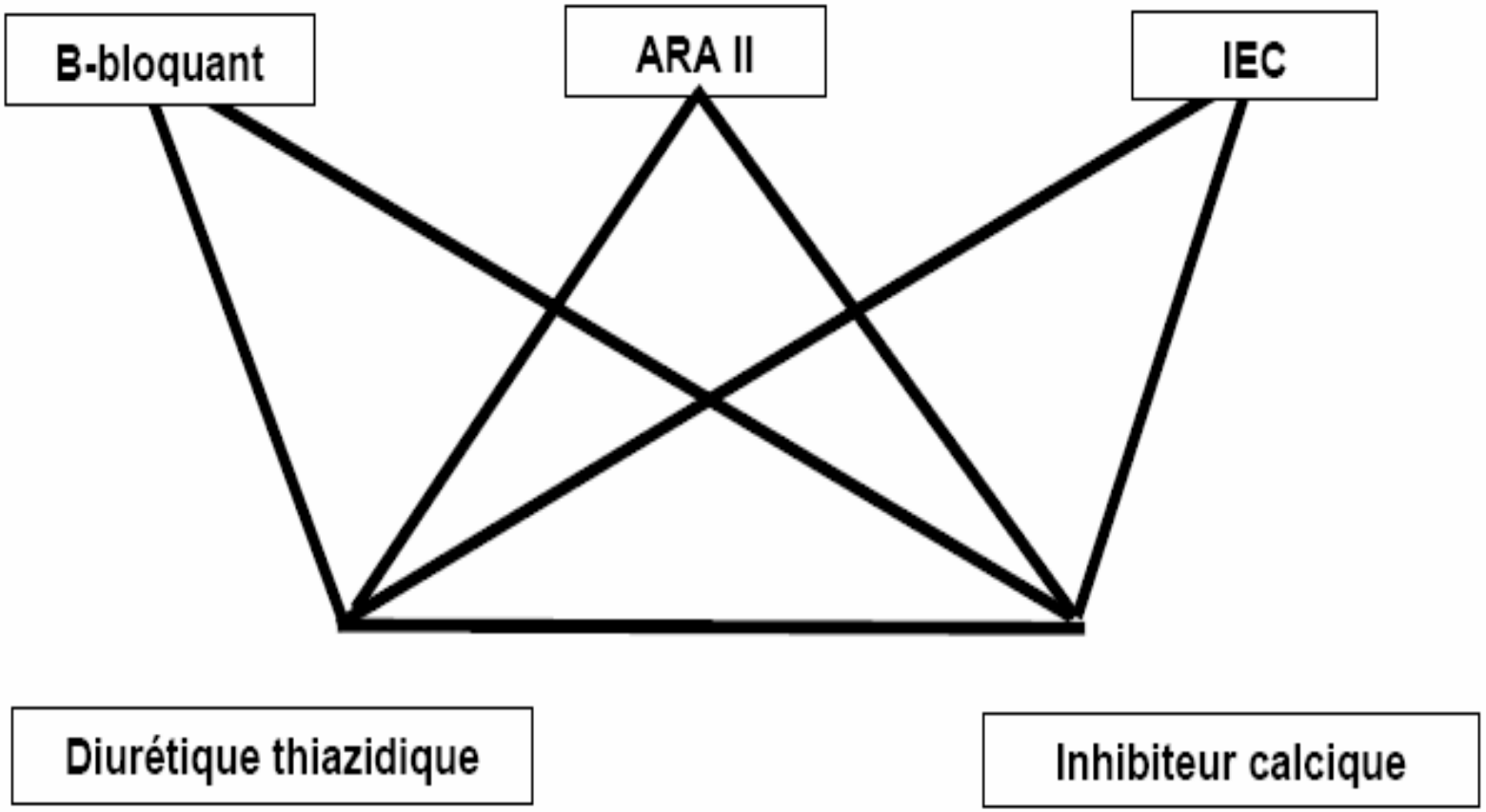
ANTI HYPERTENSEURS

ET AUTRES

QUALITE DU TRAITEMENT

- **doses**
- **synergie des associations**
- **délai de jugement**
- **passage hépatique et cytochrome P 450**
- **biodisponibilité**
- **relation concentration / effet (vallée / pic)**
- **distribution et adaptation au poids**
- **élimination et insuffisances hépatique et/ou rénale**
- **activité du SRAA et autres systèmes hormonaux**
- **interactions médicamenteuses / déplétion sodée**

ASSOCIATIONS SYNERGIQUES



Traits pleins : effet additif sur la baisse tensionnelle

SUBSTANCES VASOPRESSIVES

AINS

Alcool

Cocaïne

Réglisse

Sympathicomimétiques

Anti-VEGF

Corticoïdes

Erythropoïétine

Oestrogènes de synthèse

Tacrolimus (*FK-506, Prograf*®)

Ciclosporine (*Sandimmun*®, *Neoral*®)

INHIBITION DE L'ACTION DES ANTI HYPERTENSEURS PAR LES AINS

Pope JE ***(Arch Intern Med. 1993)***

54 études, 1324 participants (46 ans) dont 1213 hypertendus (92%)

Après ajustement sur les apports sodés, Δ PAM :

+ 3,59 mmHg / indométhacine, + 3,74 mmHg / naproxène,

+ 0,49 mmHg / piroxicam,

- 0,16 mmHg / sulindac, - 0,83 mmHg / ibuprofène,

- 1,76 mmHg / aspirine,

- 2,59 mmHg / placebo.

Johnson AG ***(Ann Intern Med. 1994)***

50 RCT dont 38 contre placebo et 12 comparant ≥ 2 AINS

Δ PAM : + 5,0 mmHg (95% IC : 1,2 – 8,7 mmHg)

Effet sur action des b-bloquants > action des vaso-dilatateurs et diurétiques.

Effet du piroxicam > du sulindac et de l'aspirine.

The effects of cyclooxygenase-2 inhibitors and NSAI therapy on 24-h BP in patients with hypertension, osteoarthritis, and type 2 diabetes mellitus.

Sowers JR et al. Arch Intern Med 2005; 165: 161-8.

Characteristic	Celecoxib (n = 136)	Rofecoxib (n = 138)	Naproxen (n = 130)
Patient Baseline Characteristics			
Age, y	61.8	63.6	63.6
Sex, % (M/F)	38/62	41/59	40/60
Race, %			
White	75	76	77
Black	15	15	13
Other	10	9	10
Weight, kg	90.6	90.9	92.2
24-h SBP, mm Hg	131.9	132.1	134.3
24-h DBP, mm Hg	75.8	76.2	76.0
24-h pulse pressure, mm Hg	56.2	55.9	58.3
Glycosylated hemoglobin, %	7.0	7.0	7.0
Nonfasting plasma glucose, mg/dL	154.2	138.0	142.0
Serum creatinine, mg/dL	0.86	0.87	0.87
Osteoarthritis index, %			
Hip	16	11	10
Knee	84	89	90
Antihypertensive Therapies†			
Combination	84 (62)	85 (64)	84 (66)
ACE inhibitor	114 (84)	109 (83)	106 (83)
ARB	24 (18)	24 (18)	19 (15)
Calcium channel blocker	38 (28)	40 (30)	39 (30)
β-Blocker	24 (18)	30 (23)	28 (22)
Diuretic	55 (40)	54 (41)	52 (41)
Other	17 (13)	13 (10)	7 (6)

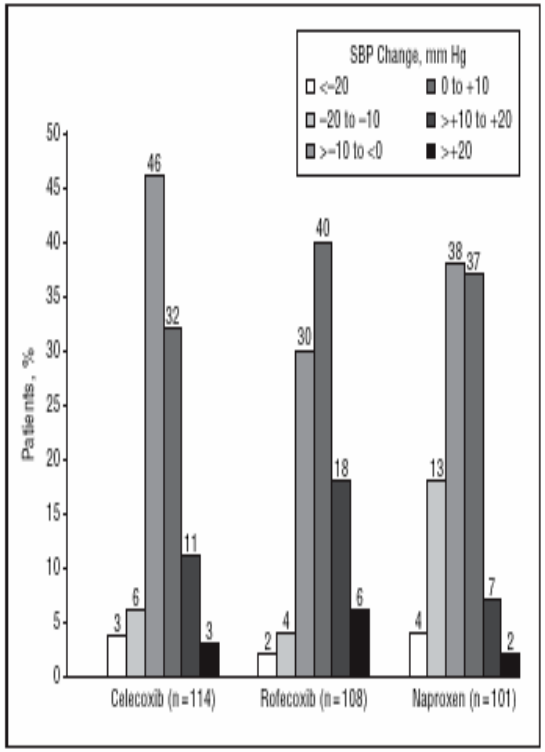


Figure 1. Distribution of changes from baseline in ambulatory systolic blood pressure (SBP) at week 6. Fewer rofecoxib-treated patients had changes in ambulatory SBP of less than 0 mm Hg than celecoxib- or naproxen-treated patients. The percentage of rofecoxib-treated patients with elevations in ambulatory SBP across the distribution of increasing BP levels was consistently greater than for either celecoxib- or naproxen-treated patients.

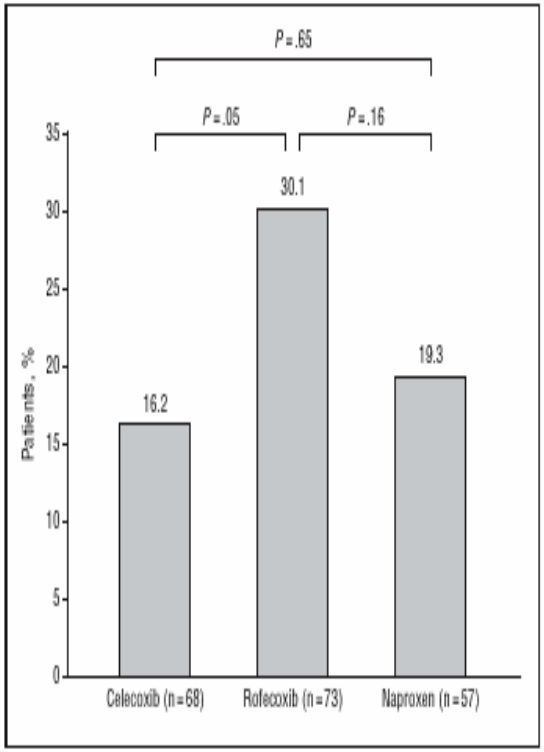


Figure 2. Percentage of baseline normotensive patients who became hypertensive at week 6. *Normotensive* is defined as an ambulatory systolic blood pressure (SBP) lower than than 135 mm Hg. *Hypertensive* is defined as an ambulatory SBP of 135 mm Hg or higher. *P* values are based on a χ^2 test. Nearly twice as many patients in the rofecoxib treatment group became hypertensive compared with the celecoxib and naproxen treatment groups.

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Home Blood-Pressure Monitoring in Patients Receiving Sunitinib.

Azizi M et al. N Engl J Med. 2008; 358: 95-7.

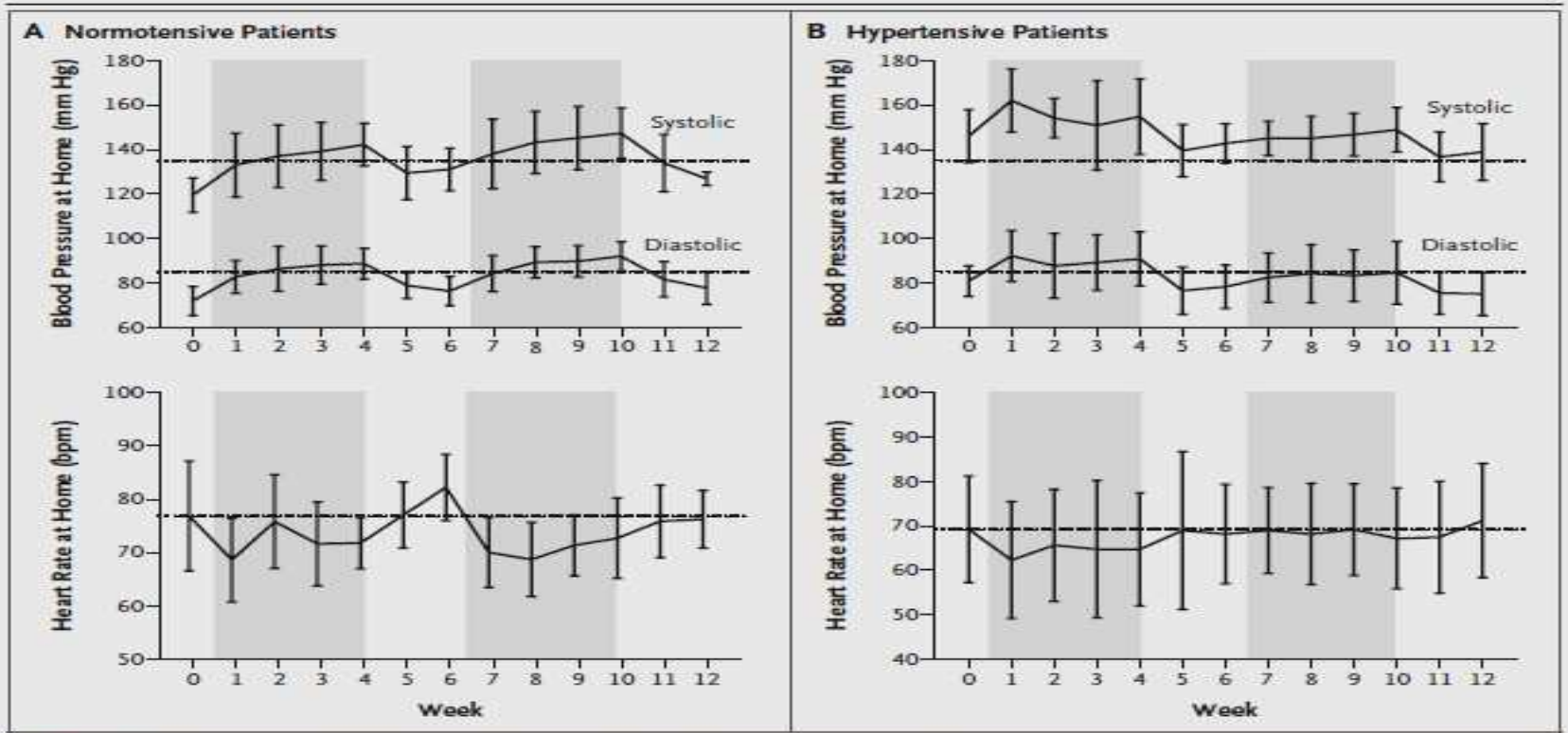


Figure 1. Changes in Systolic and Diastolic Blood Pressure and Heart Rate.

The graphs show the changes in mean blood pressure and heart rate as measured by teletransmitted results of home monitoring in patients with metastatic renal-cell carcinoma who were treated with two cycles of sunitinib at a dose of 50 mg daily for 4 weeks (shaded area), followed by 2 weeks without treatment. The results are shown separately for patients who were normotensive (Panel A) and those who were hypertensive (Panel B) before starting sunitinib treatment. In the graphs of home blood-pressure monitoring, the dotted line shows the blood-pressure threshold for the diagnosis of hypertension (systolic pressure, >135 mm Hg; or diastolic pressure, >85 mm Hg).[5] For changes in heart rate, the dotted line represents the baseline value. The I bars indicate the standard deviation.

ANALYSE ET TRAITEMENT D'UNE HTA RESISTANTE

LE MEDECIN

Inadequat management of blood pressure in a hypertensive population.

Berlowitz DR et al. NEJM 1998; 339: 1957-1963

800 hommes hypertendus, 66 ans, suivis 2 ans

	basal	final	p
PA (mmHg)	146/84	145/82	NS/< 0,001
patients PA ≥ 160/90 mmHg (%)	46	39	0,001
augmentation traitement (%)			
si PA ≥ 155/90		26	
si PA ≥ 165 (<90)		22	

Comparison of hypertension management after stroke and MI. Results from ECLAT1 – a french national wide study.

Amar J et al. Stroke 2004; 35: 1579-83.

Enquête 7 décembre 2000

3 009 généralistes

4 346 patients

	IDM	AVC	p
n	846	570	
PA ≥ 140/90 mmHg (%)	66	75	<0,001
PAS (mmHg)	141±14	144 ±15	<0,0001
PAD (mmHg)	81±8	82±9	<0,05
Monothérapie (%)	31,4	43,2	<0,0001
Trithérapie dont diurétique (%)	20	16	<0,05

Outpatient Hypertension Treatment, Treatment Intensification, and Control in Western Europe and the US

Wang YR et al. Arch Intern Med. 2007; 167: 141-147

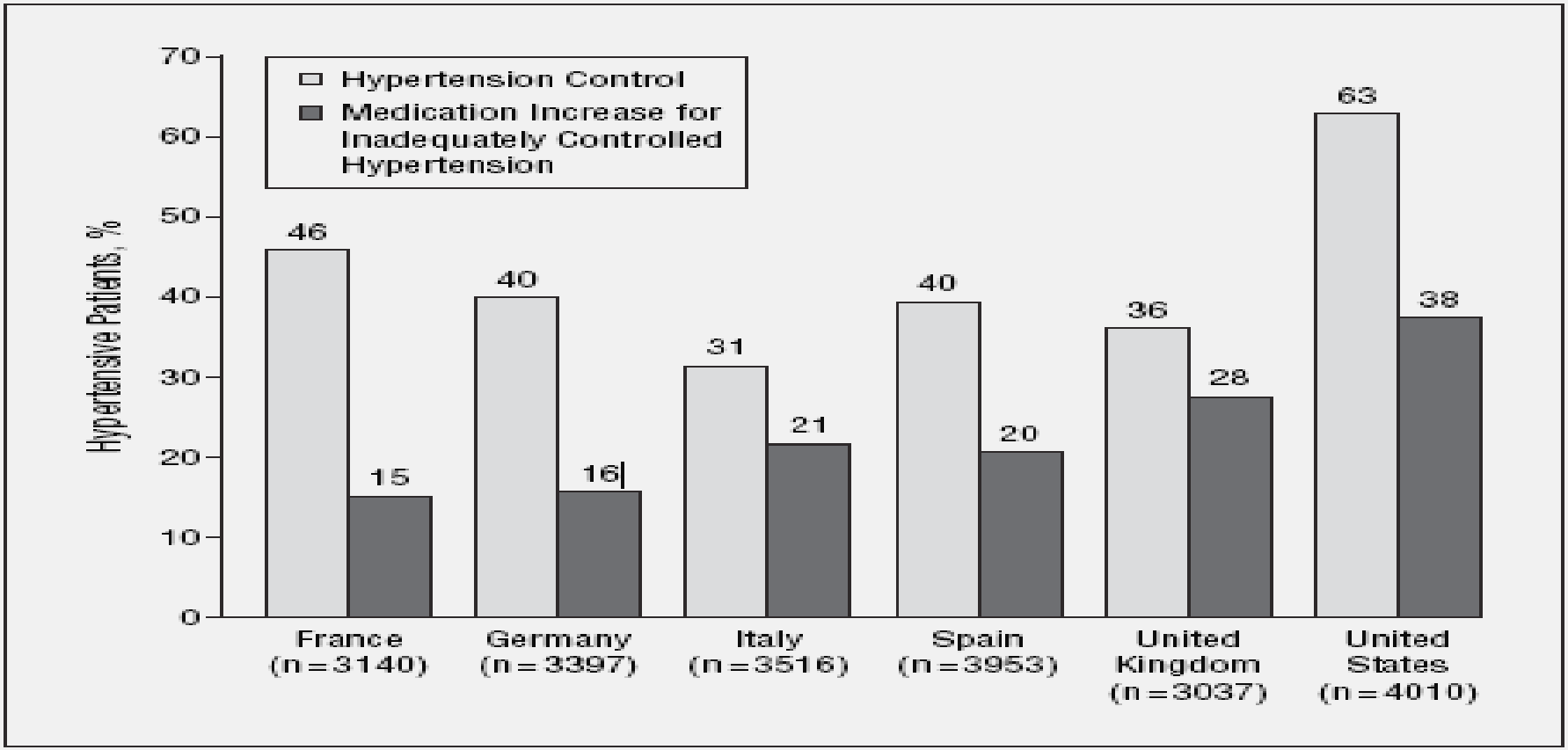
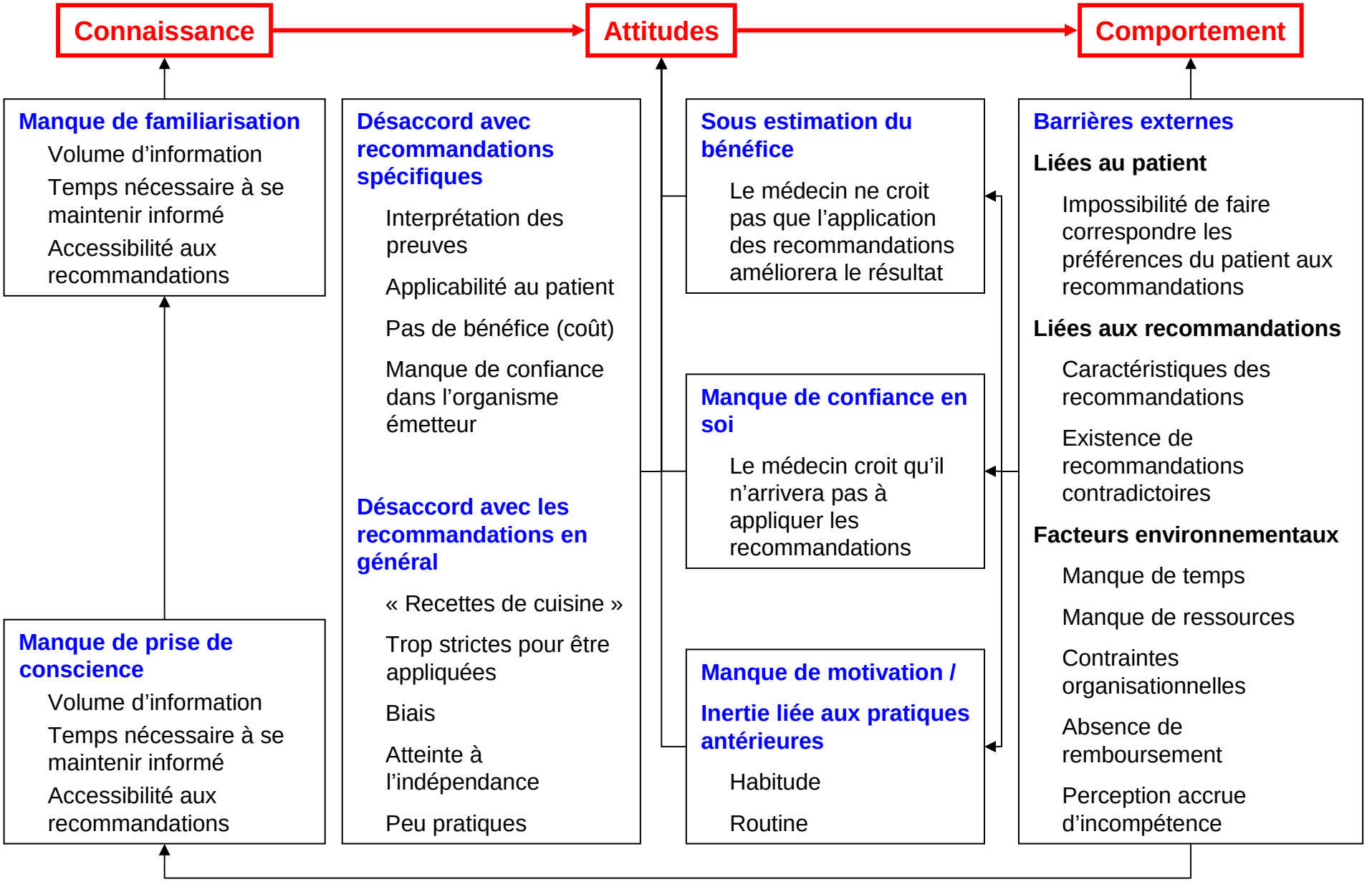


Figure 2. Cross-national differences in hypertension control (defined as a latest systolic blood pressure level of <140 mm Hg and a diastolic blood pressure level of <90 mm Hg) and medication increase for those with inadequately controlled hypertension.

Why Don't Physicians Follow Clinical Practice Guidelines?: A Framework for Improvement

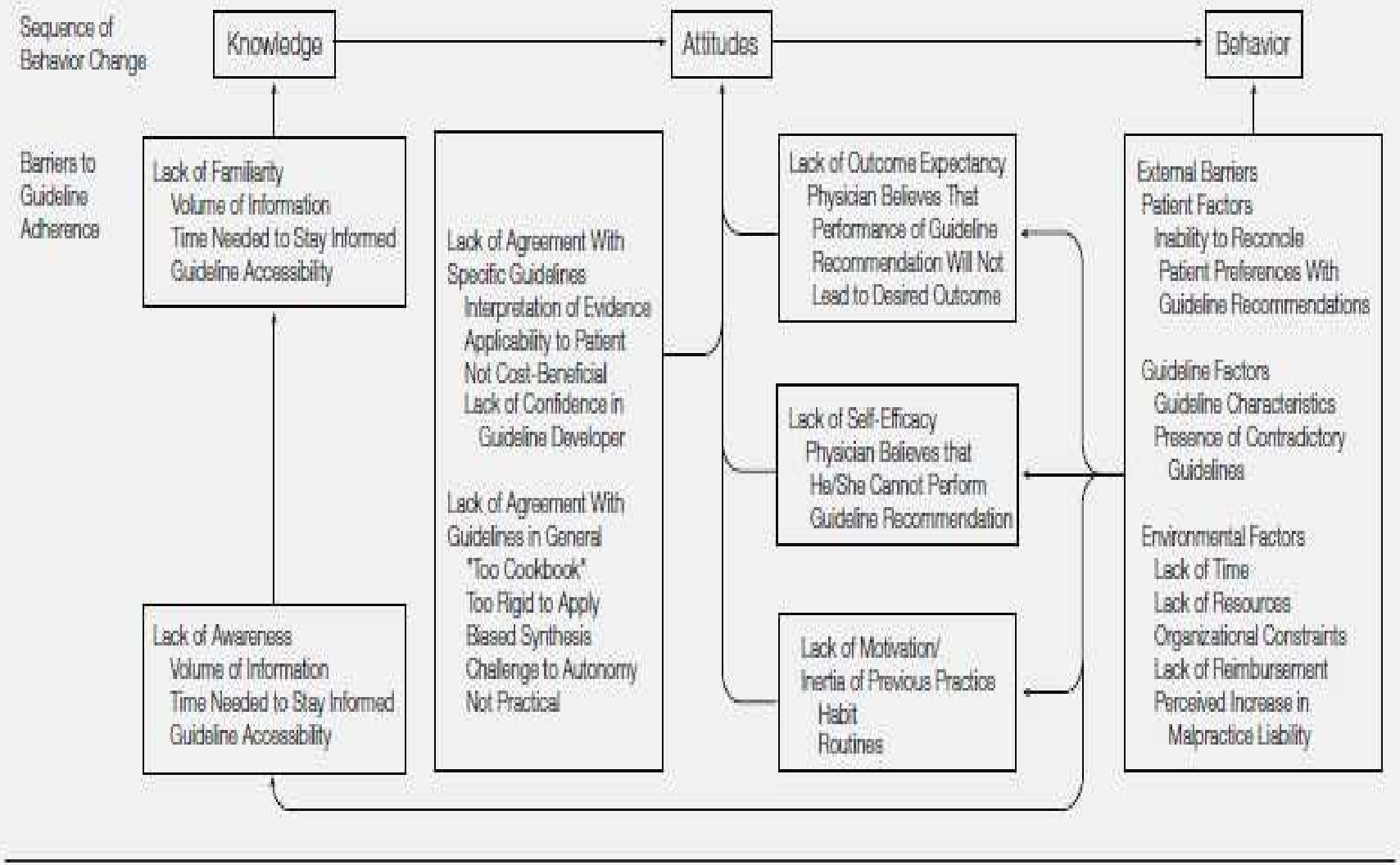
Cabana MD et al. JAMA. 1999; 282:1458-1465



Why Don't Physicians Follow Clinical Practice Guidelines?: A Framework for Improvement

Cabana MD et al. JAMA. 1999; 282:1458-1465

Figure. Barriers to Physician Adherence to Practice Guidelines in Relation to Behavior Change



Physician-related barriers to the effective management of uncontrolled hypertension.

Oliveria SA et al. Arch Intern Med 2002; 162: 413-420.

- **5 145 patients avec diagnostic d'HTA (CIM 9) en 6 mois**
- **314 patients non contrôlés dont 231 interviews téléphoniques :**
  69 ans ; 50% blancs; 152/84 mmHg ; 94% traités.
- **21/ 26 (81%) médecins ont répondu au questionnaire et donné informations sur 270 visites patients (taux de réponse : 86%).**

Connaissance du JNC VI (%)	**52**
En accord avec JNC VI (%)	**76**
Appliquent JNC VI (toujours ou habituellement) (%)	**76**

- **Motifs de non augmentation (%)**

Poursuivre mesures PA avant changement traitement	**35**
Satisfait de la réponse tensionnelle	**30**
Motif de la visite indépendant de l'HTA	**29**
PAD satisfaisante	**16**
HTA limite	**10**

- **Analyse multivariée (OR)**

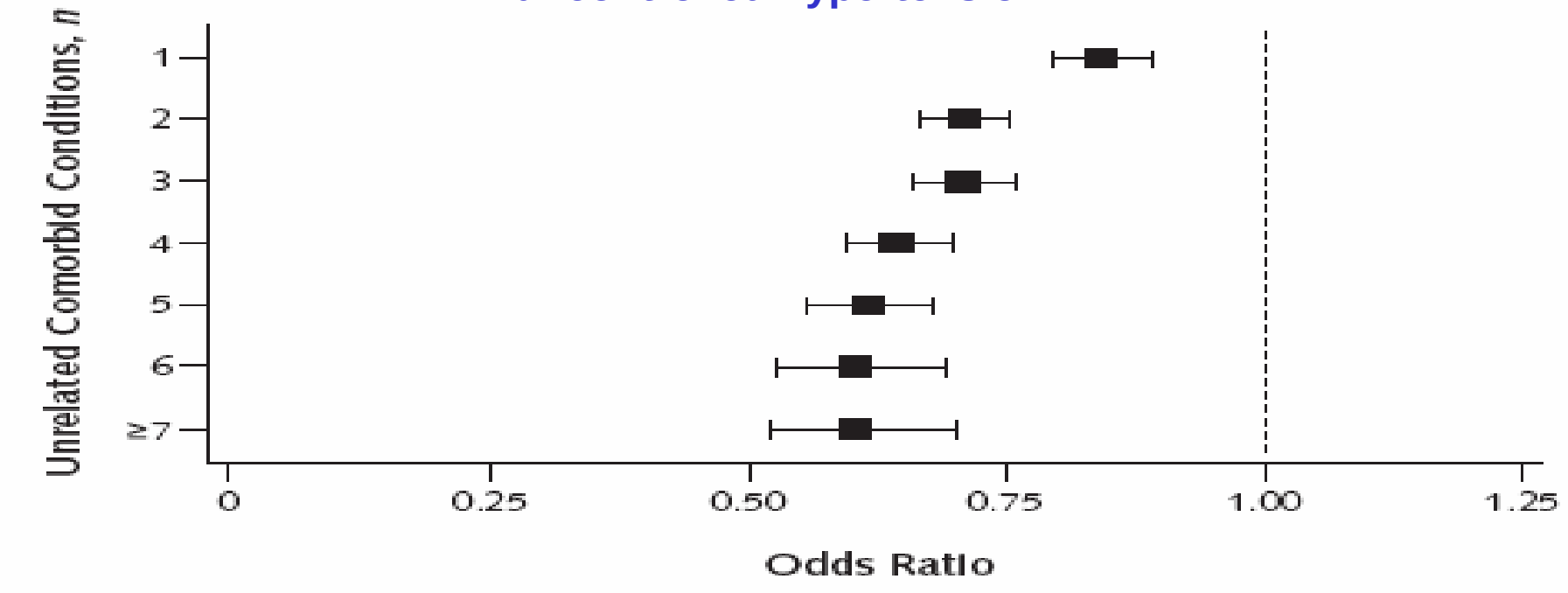
Augmentation de TTT dans les 6 mois précédents	**2.88 (1.42-5.96)**
Niveau tensionnel obtenu	**2.96 (1.53-5.83)**

Effect of Unrelated Comorbid Conditions on Hypertension Management.

Turner BJ. Ann Intern Med. 2008; 148: 578-586.

Examination of a database derived from electronic medical records collected during routine care of a cohort of primary care: 15 459 patients with uncontrolled hypertension who made 70 557 visits to 200 clinicians (01/2004 – 12/2006).

Adjusted association of unrelated comorbid conditions with management of uncontrolled hypertension



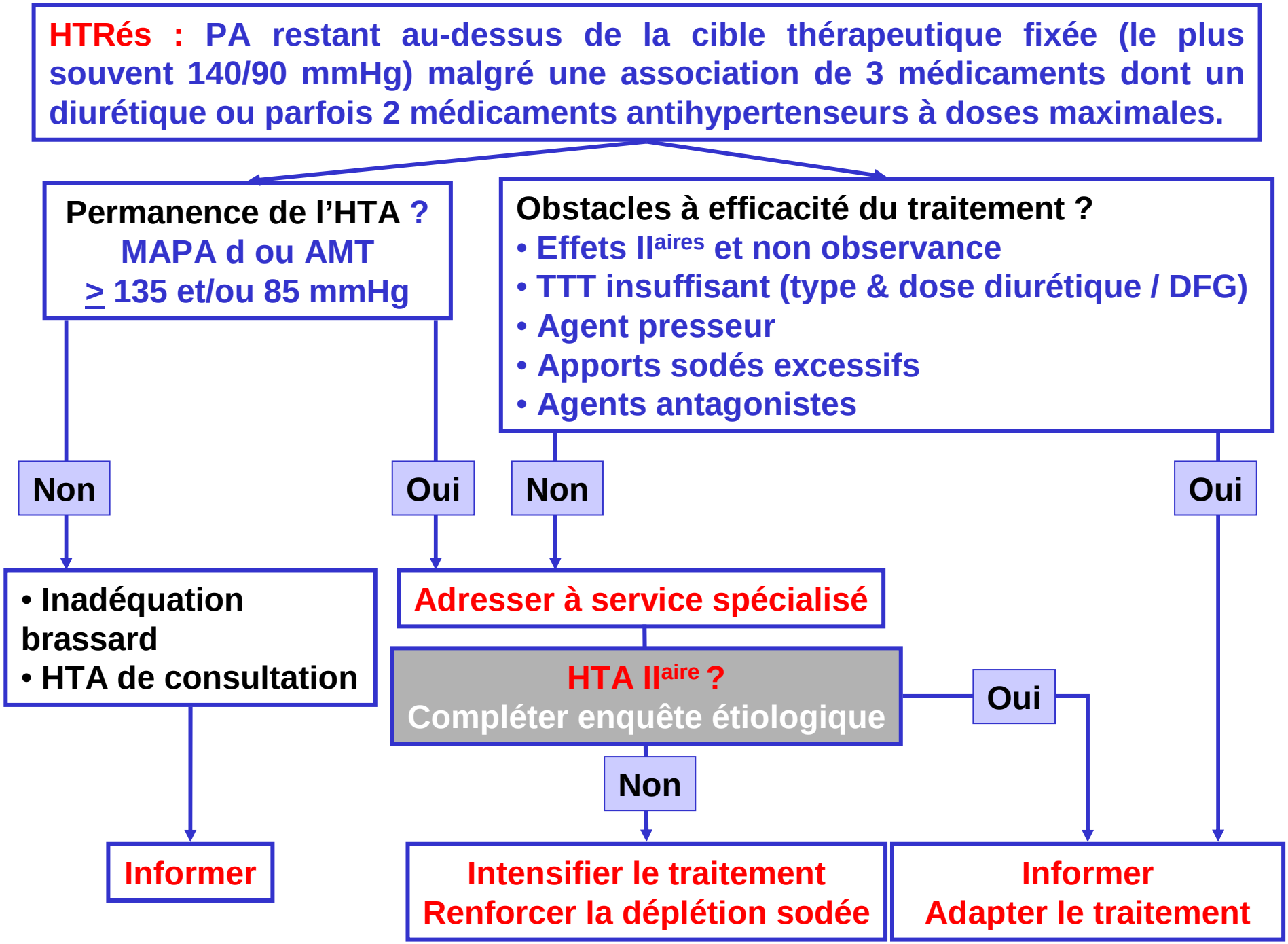
The role of clinical uncertainty in treatment decisions for diabetic patients with uncontrolled blood pressure.

Kerr EA et al. Ann Intern Med. 2008; 148: 717-727.

1169 diabetic patients (2005-2006).

Despite an average SBP of 154 mmHg, only 49% of patients had a change in a BP treatment (medication intensification or planned follow-up within 4 weeks).

Factors of intensification			p
OBP < 140/90 vs. ≥ 140/90 mmHg or no OBP	13%	61%	<0.001
HBPM < 140/90 vs. ≥ 140/90 mmHg or no HBPM	18%	52%	<0.001
OSBP goal > 130 mmHg vs < 130 mmHg	33%	52%	0.008
Discussion of medication issues vs no	23%	52%	<0.001


HTRés : PA restant au-dessus de la cible thérapeutique fixée (le plus souvent 140/90 mmHg) malgré une association de 3 médicaments dont un diurétique ou parfois 2 médicaments antihypertenseurs à doses maximales.
Permanence de l'HTA ?
MAPA d ou AMT
≥ 135 et/ou 85 mmHg
Obstacles à efficacité du traitement ?
• Effets IIaires et non observance
• TTT insuffisant (type & dose diurétique / DFG)
• Agent presseur
• Apports sodés excessifs
• Agents antagonistes
Non
Oui
Non
Oui
• Inadéquation brassard
• HTA de consultation
Adresser à service spécialisé
HTA IIaire ?
Compléter enquête étiologique
Oui
Non
Informer
Intensifier le traitement
Renforcer la déplétion sodée
Informer
Adapter le traitement