

Top ~~10~~ Coups de coeur 2019

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Déclaration de conflit d'intérêt potentiel

- J'ai préparé le matériel moi-même.
- J'ai utilisé des extraits des références citées.
- Je reçois un honoraire de la SQHTA pour cette présentation.
- Je n'ai pas reçu d'honoraires ou quelconque avantage direct ou indirect d'une compagnie tiers ayant un intérêt en hypertension et son traitement.

Méthode

- PubMed:
 - 124 articles: Hypertension, clinical trial, drug therapy
- QxRead: Hypertension
- Practice Update: Hypertension, High Blood pressure
- NEJM Journal Watch; Hypertension
- Collègues



Oral antihypertensive regimens (nifedipine retard, labetalol, and methyldopa) for management of severe hypertension in pregnancy: an open-label, randomised controlled trial

Thomas Easterling, Shuchita Mundle, Hillary Bracken, Seema Parvekar, Sulabha Mool, Laura A Magee, Peter von Dadelszen, Tara Shochet, Beverly Winikoff

***Lancet* 2019; 394: 1011–21**

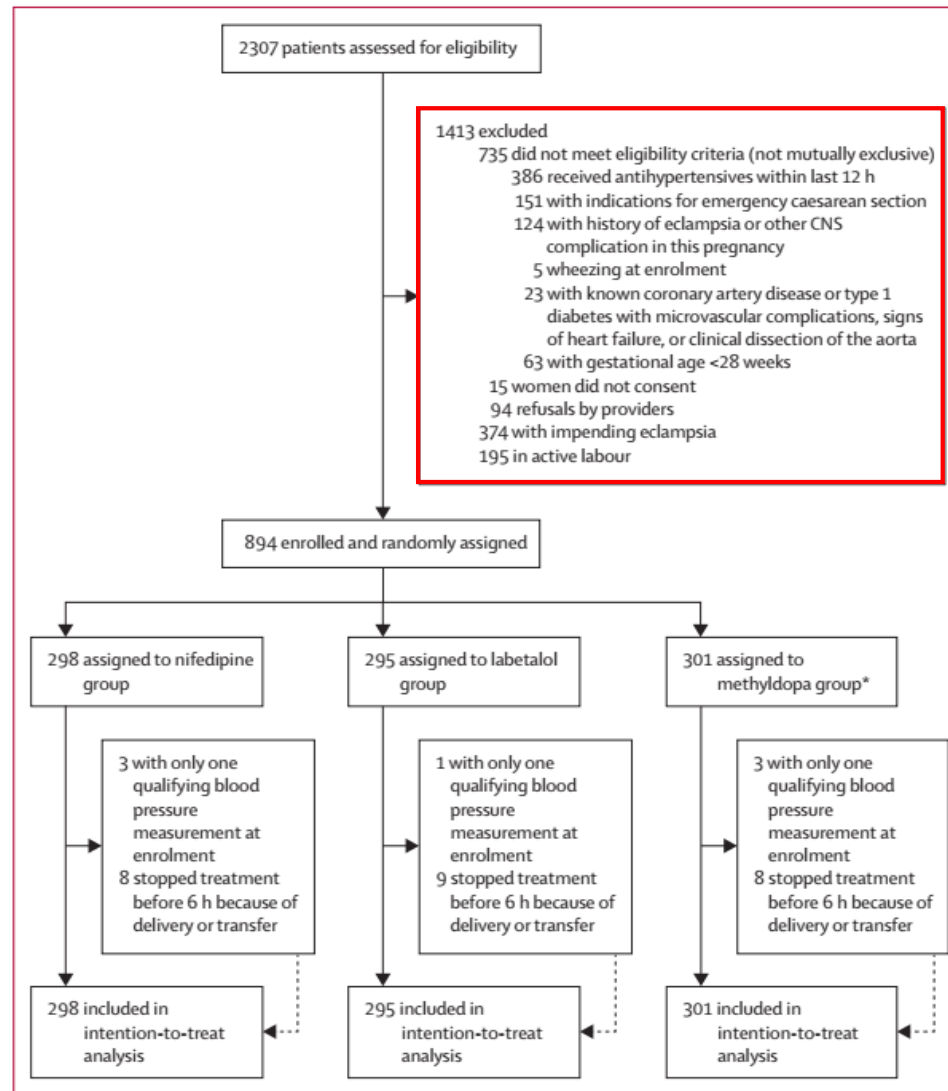
Collaboration avec Université de Washington
Et Université de Colombie Britannique

Introduction

- Médicaments étudiés surtout intraveineux.
 - Nécessité de voie d'accès veineuse.
 - Baisse de la tension parfois trop rapide.
 - Monitoring foetal requis.
- Avantages potentiels de la voie orale.
- Comparaison directe des médicaments oraux requise

Méthodes

- Étude ouverte, randomisée, groupes parallèles, multicentrique.
- Réalisée dans 2 hôpitaux publics universitaires en Inde.
- Critère d'inclusion :
 - Age gestationnel ≥ 28 semaines
 - TAS ≥ 160 mm Hg and TAD ≥ 110 mm Hg. (Méthode auscultatoire)
- Intervention:
 - Nifédipine LA 10 mg q1h (max 30 mg),
 - Labétalol 200 mg q1h (max 600 mg),
 - Méthylidopa 1000 mg (dose unique)
- 2307 femmes; 261 pts/groupe puissance 80%
- Contrôle = TAS 120-150 mm Hg et TAD 70-100 mm Hg. En moins de six heures.
- Suivi par méthodes oscillométrique Q 20 minutes
- Analyse par intention de traiter.
- Financement: PREEMPT: Fondation Bill & Melinda Gates



1:1:1

Caractéristiques démographiques

	Nifedipine (n=298)	Labetalol (n=295)	Methyldopa (n=301)
Study site			
Government Medical College	150 (50%)	151 (51%)	151 (50%)
Daga Women's Hospital	148 (50%)	144 (49%)	150 (50%)
Maternal demographics			
Maternal age, years	25.6 (4.0)	25.5 (4.2)	25.5 (4.2)
Body-mass index, kg/m ²	27.1 (4.3; 16.4–43.2)	27.4 (4.3; 17.8–43.7)	27.3 (4.3; 16.2–39.2)
Pregnancy characteristics			
Gestational age, weeks	36.5 (2.9)	36.5 (2.7)	36.7 (2.6)
Multiple pregnancy	12 (4%)	7 (2%)	9 (3%)
Fetus alive at enrolment	298 (100%)	295 (100%)	301 (100%)
Clinical measurements			
Mean systolic blood pressure, mm Hg	158 (11.1; 130–200)	158 (11.5; 130–200)	157 (11.7; 130–200)
Highest systolic blood pressure at enrolment, mm Hg*			
<160	109 (37%)	102 (35%)	109 (36%)
160–169	112 (38%)	125 (42%)	129 (43%)
≥170	77 (26%)	68 (23%)	63 (21%)
Mean diastolic blood pressure, mm Hg	109 (7.6; 87–165)	108 (7.1; 87–140)	108 (6.8; 90–130)
Highest diastolic blood pressure at enrolment, mm Hg			
<110	56 (19%)	64 (22%)	73 (24%)
110–119	202 (68%)	186 (63%)	199 (66%)
≥120	40 (13%)	45 (15%)	29 (10%)
Urinary dipstick proteinuria			
Nil or trace	108 (36%)	98 (33%)	105 (35%)
1+	93 (31%)	91 (31%)	104 (35%)
2+	69 (23%)	70 (24%)	60 (20%)
>3+	28 (9%)	36 (12%)	32 (11%)
Oxygen saturation <95%	0	1 (<1%)	0
Mean heart rate, bpm	95.7 (13.9; 55–141)	97.4 (12.6; 62–134)	97.2 (14.8; 56–142)
Source of admission			
Referred	79 (27%)	83 (28%)	100 (33%)
Outpatient department	180 (60%)	179 (61%)	173 (58%)
Walk-in	39 (13%)	33 (11%)	28 (9%)
Received magnesium sulphate in 12 h before enrolment	16 (5%)	7 (2%)	15 (5%)
Received an antihypertensive drug at least 12 h before enrolment	63 (21%)	65 (22%)	61 (20%)
Methyldopa	12 (4%)	16 (5%)	13 (4%)
Nifedipine	35 (12%)	30 (10%)	29 (10%)
Labetalol	21 (7%)	22 (8%)	20 (7%)
Plans for delivery at time of enrolment			
Planned induction of labour	118 (40%)	128 (43%)	114 (38%)
Planned expectant management	180 (60%)	167 (57%)	187 (62%)
Laboratory values, n (%)/N			
Platelet count <1 × 10 ⁹ /L	16 (6%)/287	12 (4%)/286	17 (6%)/295
Serum creatinine ≥1.0	22 (8%)/294	23 (8%)/294	20 (7%)/300
Aspartate transaminase >80 IU/L	10 (3%)/293	12 (4%)/293	20 (7%)/301
Aspartate transaminase >80 IU/L and platelet count <1 × 10 ⁹ /L	1 (<1%)/285	1 (<1%)/285	3 (1%)/295

Data are n (%), n (%)/N, mean (SD), or mean (SD; range). *Seven women did not meet blood pressure eligibility criteria at the time of enrolment: three women in the nifedipine group, one woman in the labetalol group and three women in the methyldopa group.

Table 1: Baseline characteristics

Résultats

	Nifedipine (n=298)	Labetalol (n=295)	Methyldopa (n=301)	Absolute difference, nifedipine vs labetalol (95% CI)	Absolute difference, nifedipine vs methyldopa (95% CI)	Absolute difference, labetalol vs methyldopa (95% CI)
Primary outcome*						
Achieved primary outcome	249 (84%)	228 (77%)	230 (76%)	6.3 (-0.1 to 12.6)	7.1 (0.8 to 13.5)	0.9 (-5.9 to 7.6)
Exploratory results of primary outcome						
Achieved primary outcome without needing additional antihypertensive therapy†	247 (83%)	227 (77%)	190 (63%)	6.0 (-0.4 to 12.4)	19.8 (12.9 to 26.7)	13.8 (6.5 to 21.1)
Reached the blood pressure target	254 (85%)	231 (78%)	232 (77%)	6.9 (0.7 to 13.1)	8.2 (1.9 to 14.4)	1.2 (-5.5 to 7.9)
Any adverse outcome‡	7 (2%)	4 (1%)	3 (1%)	1.0 (-1.2 to 3.2)	1.4 (-0.7 to 3.4)	0.4 (-1.4 to 2.1)
Received additional antihypertensive drugs	2 (1%)	9 (3%)	56 (19%)	-2.4 (-4.6 to -0.2)	-17.9 (-22.4 to -13.4)	-15.6 (-20.4 to -10.7)
Achieved primary outcome at 3 h	219 (74%)	212 (72%)	185 (62%)	1.6 (-5.5 to 8.8)	12.0 (4.6 to 19.5)	10.4 (2.9 to 17.9)
Received magnesium sulphate during study period	31 (10%)	40 (14%)	34 (11%)	-3.2 (-8.4 to 2.1)	-0.9 (-5.9 to 4.1)	2.3 (-3.0 to 7.6)

Nifédipine (84%) vs methyldopa (76%) p=0,03

Nifédipine (84%) vs Labétalol (77%) p=0,05

Labétalol (77%) vs methyldopa (76%) p=0,80

P=0,04

Doses et besoin de médicament de secours

Doses additionnelles

	Nifedipine (n=298)	Labetalol (n=295)	Methyldopa (n=301)	Absolute difference, nifedipine vs labetalol (95% CI)
Received a second dose of allocated medication	131 (44%)	142 (48%)	0	-4.1 (-12.1 to 3.9)*
Median time received after randomisation, min	85 (60 to 365)	130 (60 to 370)	0	-45 (-78 to -12)
Received a third dose of allocated medication	45 (15%)	64 (22%)	0	-6.6 (-12.8 to 0.4)†
Median time received after randomisation, min	175 (120 to 360)	190 (125 to 330)	0	-15 (-66 to 36)

Besoin d'un autre médicament

Received another antihypertensive drug during the study period‡	2 (1%)	9 (3%)	56 (19%)	-2.4 (-4.6 to -0.2)	-17.9 (-22.4 to -13.4)	-15.6 (-20.4 to 10.7)
Nifedipine§	0	8 (3%)	47 (16%)	-2.7 (-4.5 to -0.9)	-15.6 (-19.7 to -11.5)	-12.9 (-17.4 to -8.4)
Labetalol (orally)¶	1 (<1%)	0	10 (3%)	0.3 (-0.3 to 0.9)	-3.0 (-5.1 to -0.9)	-3.3 (-5.3 to -1.3)
Labetalol (intravenously)	0	1 (<1%)	0	-0.3 (-0.9 to 0.3)	..	0.3 (-0.3 to 0.9)
Methyldopa	1 (<1%)	0	1 (<1%)	0.3 (-0.3 to 0.9)	0.003 (-0.9 to 0.9)	-0.3 (-0.9 to 0.3)

Effets indésirables

- Fréquence non précisée
- Nifédipine:
 - céphalée et tachycardie
 - Plus bébés de petit poids avec séjour UNN; âge gestationnel idem (NS)
 - Évolution travail et accouchement similaire

Discussion

- Pas d'insu mais endpoint objectifs
 - Sauf décision de titration/ajout ?
- Fraction échantillonnale faible.
- Validité externe ?
- Validité pour le contrôle de l'hypertension post-partum ?
- Rôle médicament p.o. vs i.v. ?



Patiromer versus placebo to enable spironolactone use in patients with resistant hypertension and chronic kidney disease (AMBER): a phase 2, randomised, double-blind, placebo-controlled trial

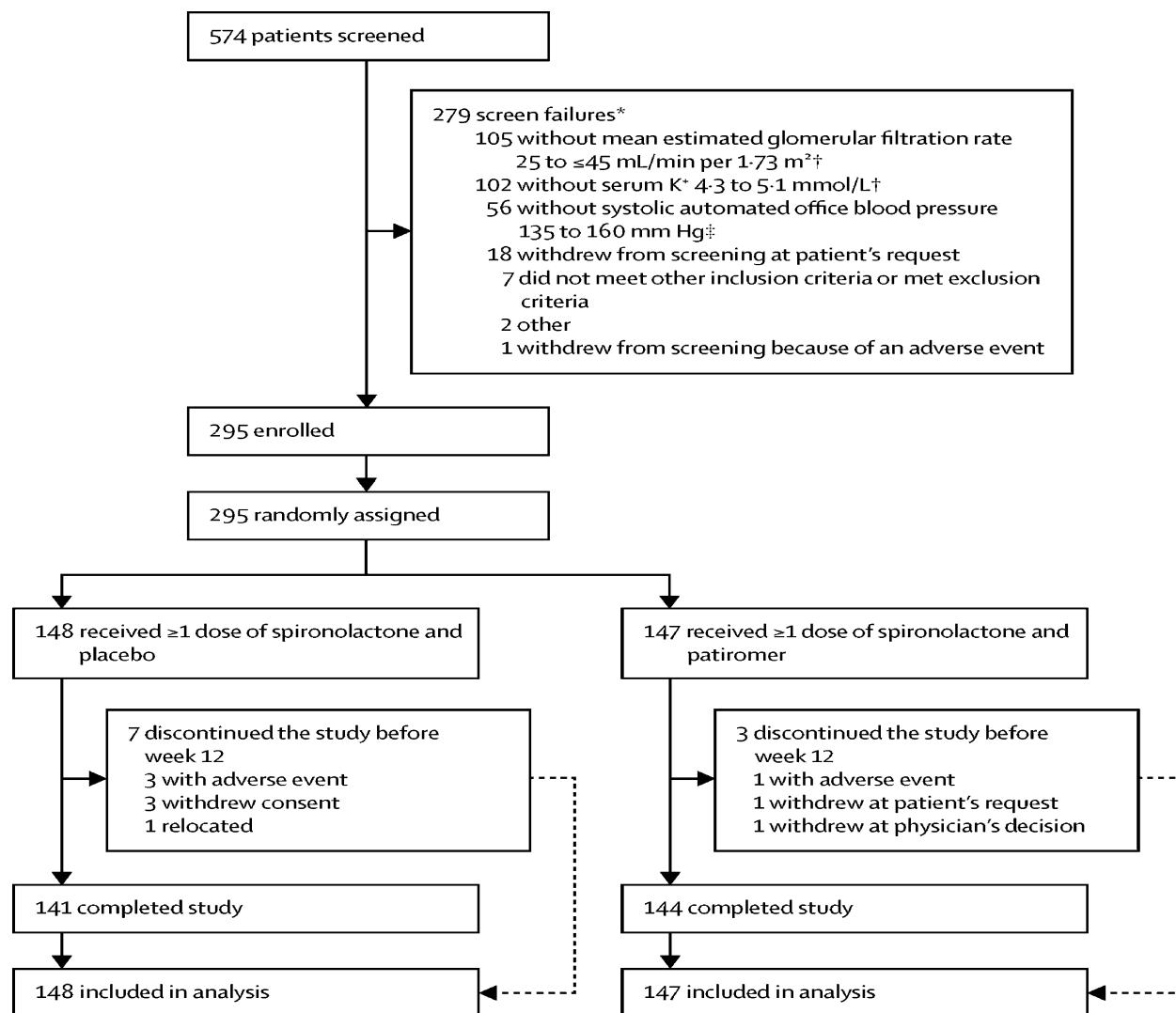
Agarwal R et al Lancet 2019; 394: 1540–50

Objectifs

- Ajout spironolactone (S) à IECA/ARA double le risque d'hyperkaliémie dans l'HTA réfractaire en insuffisance rénale
- Patiromer est un agent liant du K^+ , sans sodium
- Efficace pour prévenir l'hyperkaliémie induite par S en insuffisance cardiaque.
- Objectif primaire: Proportion de patients sous S à 12 sem
- Objectifs secondaires
 - Variation TAS à 12 sem
 - Dose cumulative de S, durée traitement au S, temps pour discontinuation

Méthode

- Essai 12 semaines chez patients avec
 - IRC (25-45 mL/min/1,73 m²)
 - K⁺ 4,3 – 5,1 mmol/L
 - HTA réfractaire: 3 Rx dont IECA/ARA+ diurétiques
- 62 centres
- A l'aveugle
- Mesure TA oscillométrique automatisée sans surveillance après repos 3-5 min puis x 3.
- Validation observance au spironolactone par dosages plasmatiques
- Spironolactone 25 mg ID ↑ 50 mg (sem 3) si TAS ≥ 120 mmHg et K⁺ < 5.1 mmol/L
- Patiromer 2 sachets le matin 3h avant et après médicaments (0-6/jours)
- Puissance statistique 90% pour différence 20% sur Objectif primaire

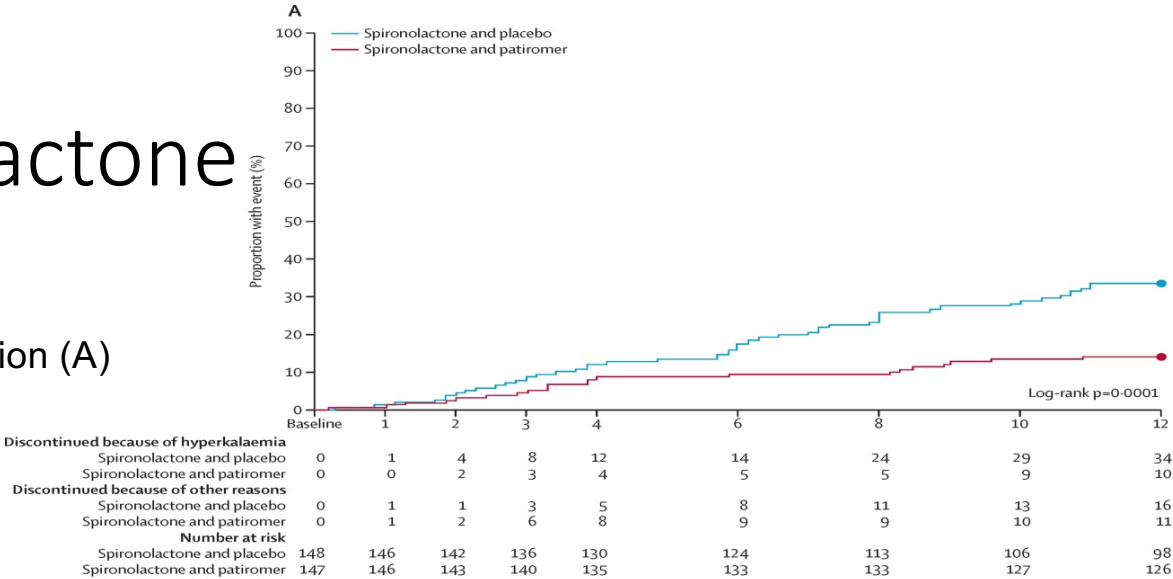


Résultats

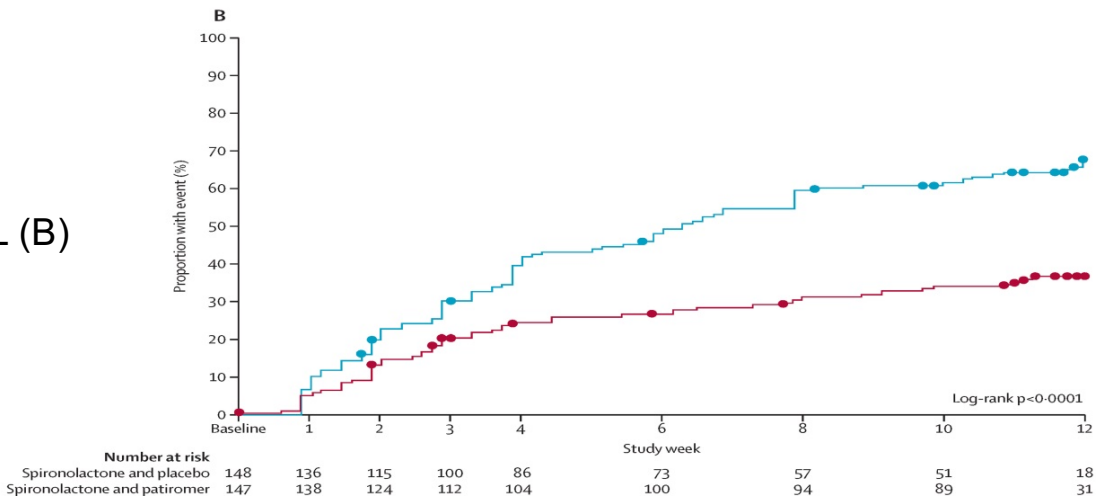
	Spironolactone and placebo (n=148)	Spironolactone and patiomer (n=147)	Difference between groups (95% CI)	p value for between-group difference
Primary endpoint				
Patients who remained on spironolactone at week 12	98 (66%)	126 (86%)	19.5% (10.0 to 29.0)	<0.0001
Secondary endpoint				
Systolic automated office blood pressure (mm Hg)	..	..	-1.0* (-4.4 to 2.4)	0.58
Baseline	144.9 (0.6)	143.3 (0.5)	..	..
Week 12	133.9 (1.4)	131.9 (1.2)	..	..
Change from baseline in systolic automated office blood pressure (mm Hg), least squares mean (95% CI)	-10.8 (-13.2 to -8.3)†	-11.7 (-14.1 to -9.3)‡	..	..
p value for change from baseline	<0.0001	<0.0001	..	..
Spironolactone dose				
Cumulative dose of spironolactone (mg)	2580.7 (95.8)	2942.3 (80.1)	384.7* (140.4 to 629.0)	0.0021
Data are n (%) or mean (SE), unless otherwise indicated. *Least squares mean. †n=141. ‡n=144.				
Table 2: Primary endpoint, secondary efficacy endpoint, and spironolactone dose				

Délai arrêt spironolactone

Time to spironolactone discontinuation (A)



Time to serum potassium ≥ 5.5 mmol/L (B)

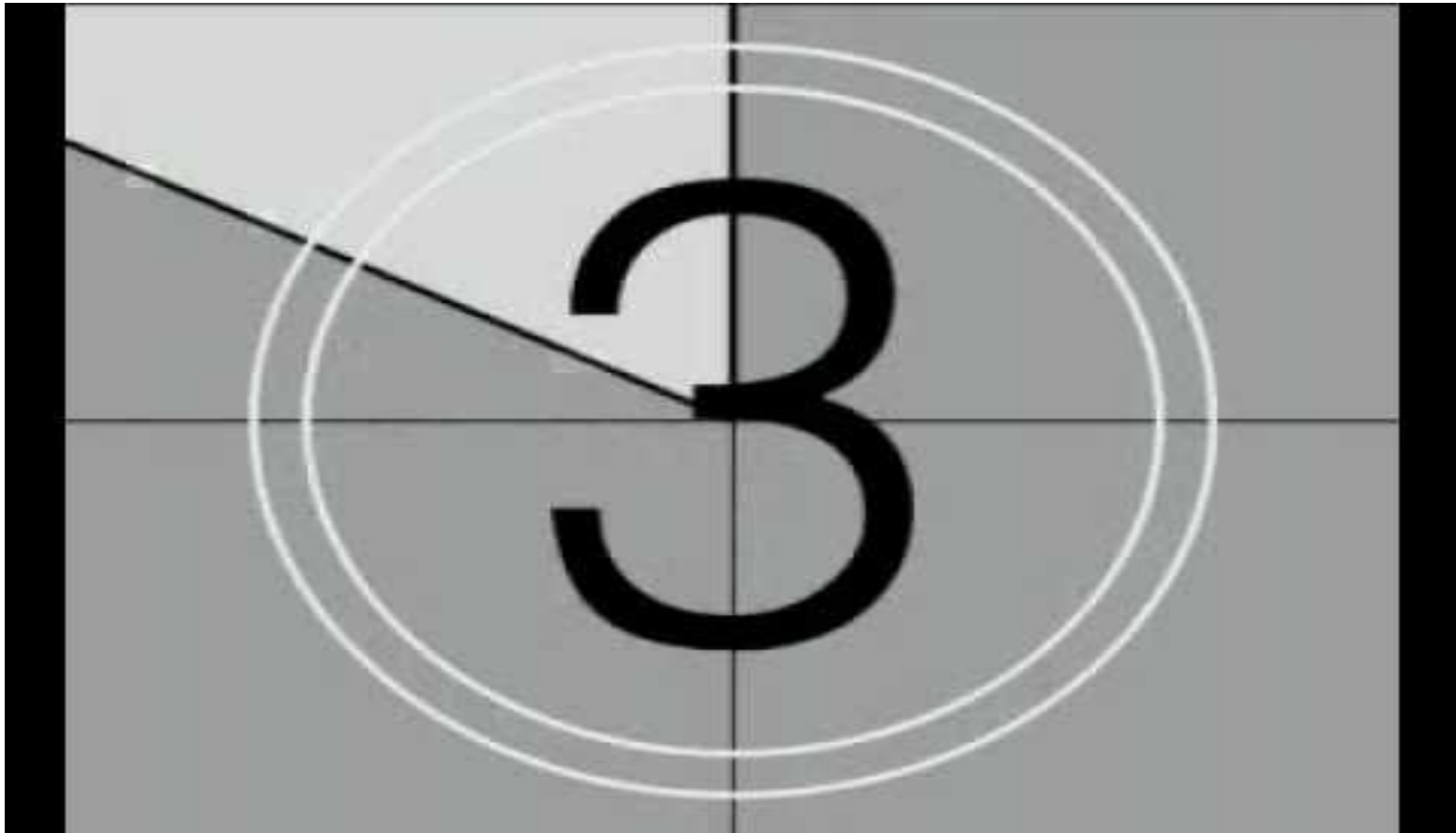


Effets indésirables

	Spironolactone and placebo (n=148)	Spironolactone and patiromer (n=147)
Adverse event summary		
Adverse events	79 (53%)	82 (56%)
Severe adverse events	3 (2%)	2 (1%)
Serious adverse events*	4 (3%)	1 (1%)
Adverse event leading to study treatment discontinuation	21 (14%)	10 (7%)
Hyperkalaemia	11 (7%)	2 (1%)
Adverse event leading to death	1 (1%)	0
Most common individual adverse events†		
Hyperkalaemia or blood potassium increased	14 (9%)	9 (6%)
Renal impairment	10 (7%)	13 (9%)
Headache	11 (7%)	9 (6%)
Diarrhoea	8 (5%)	9 (6%)
Hypotension‡	6 (4%)	9 (6%)

Discussion

- Méthode estimation DFG non décrite.
- 69% ont reçu 50 mg S
- Pas de différence significative de TAS mais 66% patients sans patiromer sous S
- Efficace
- Bien toléré
- Observance ? (3 heures avant et après les médicaments)
- Permet meilleure tolérance de S avec IECA/ARA,



Bedtime hypertension treatment improves cardiovascular risk reduction: the Hvsia Chronotherapy Trial

Hermida RC et al European Heart Journal (2019) 0, 1–12 CLINICAL RESEARCH doi:10.1093/eur



Introduction

- Variations circadiennes de la TA
- Activité accrue du SRAA durant sommeil
- TA nocturne est meilleur marqueur pronostique de MCV que TA diurne
- Risque CV accru chez les non-dipper
- Études passées d'administration HS sans contrôle avec administration AM
- Étude MAPEC a démontré que dose HS améliore TA nocturne, réduit le non-dipping, réduit événements CV (Suivi 5,6 ans)
- Objectifs: Comparer contrôle TA et réduction risque CV avec dose HS vs AM

Méthode

- Espagne, soins première ligne
- Synchronisés activité diurne, sommeil nocturne
- HTA diagnostiquée par MAPA
 - Diurne TAS ≥ 135 , TAD ≥ 85 ; nocturne TAS ≥ 120 TAD ≥ 70
 - Traité par médicaments
- Journal d'activité et heures de coucher et lever
- Exclusion: travailleur de quarts, HTA secondaire, MCAS instable, arythmies, IRC (≤ 60 mL/min), albuminurie
- Suivi médian 5 ans, minimum 1 an/participant
- Taille échantillon puissance 80% pour détecter différence morbi-mortalité de 20%

Méthode (suite)

- Anti-HTA: ARA, IECA, BCC, β -bloq, diurétique: tous pris AM vs HS
- Suivi annuel avec MAPA48h si TA contrôlée
- Analyse des événements à l'aveugle
 - IM, revascularisation coronarienne, ICC, AVC, mort CV
- Dipper définis comme baisse de $\geq 10\%$
- Analyse en ITT
- Co-variables :DB, DLP, age, sexe

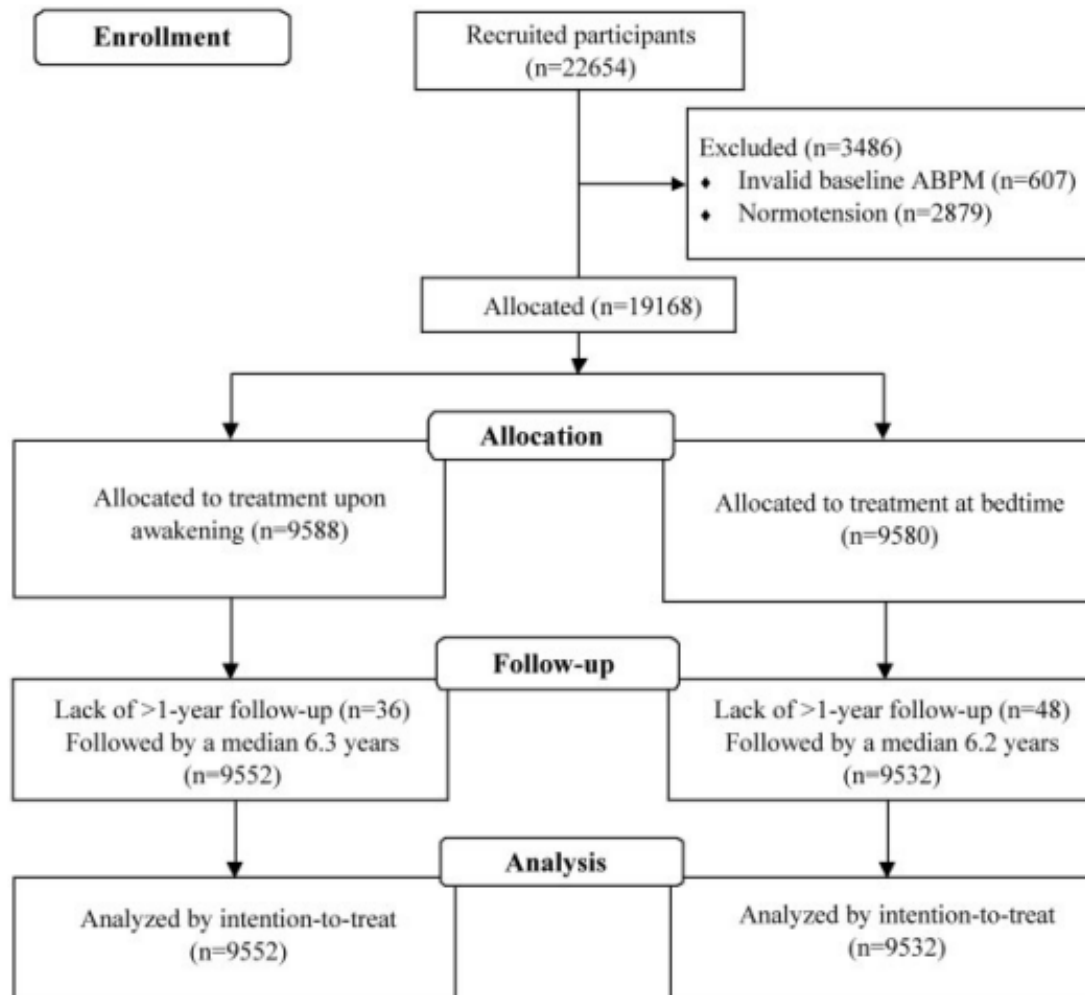


Figure 1 Flow diagram of participants in the study.

Résultats

Table 2 Final characteristics of participants categorized according to treatment-time regimen (either upon awakening or at bedtime)

Variable	Awakening	Bedtime	P between groups
Participants, n	9552	9532	
Hypertension treatment			
Number of medications	1.80 ± 0.89	1.71 ± 0.93	<0.001
ARB, %	53.1	53.1	0.995
ACEI, %	25.3	23.4	0.002
CCB, %	32.7	36.8	<0.001
β-Blocker, %	22.0	17.5	<0.001
Diuretic, %	46.5	39.5	<0.001
Clinical laboratory test values			
Glucose, mg/dL	108.1 ± 33.5	108.3 ± 31.7	0.656
Creatinine, mg/dL	1.16 ± 0.96	1.06 ± 0.90	<0.001
Uric acid, mg/dL	5.9 ± 1.6	5.8 ± 1.5	0.057
Total cholesterol, mg/dL	198.5 ± 40.3	197.7 ± 40.6	0.385
Triglycerides, mg/dL	131.7 ± 84.6	131.1 ± 80.6	0.639
HDL cholesterol, mg/dL	51.8 ± 15.8	53.0 ± 14.8	<0.001
LDL cholesterol, mg/dL	120.7 ± 36.6	118.2 ± 36.5	0.002
Haemoglobin, g/dL	14.1 ± 1.6	14.0 ± 1.6	0.270
Estimated glomerular filtration rate	75.7 ± 26.9	79.3 ± 23.5	<0.001
Albumin/creatinine (Cr) ratio, mg/gCr, median (interquartile range)	7.0 (3.7–20.0)	6.5 (3.6–18.0)	0.030
Office* and ambulatory BP			
Office SBP, mmHg	143.2 ± 20.9	140.0 ± 20.6	<0.001
Office DBP, mmHg	82.4 ± 12.3	81.4 ± 12.4	<0.001
Office PP, mmHg	60.8 ± 17.9	58.6 ± 17.9	<0.001
Office heart rate, beats/min	71.9 ± 12.5	72.4 ± 12.5	0.078
Awake SBP mean, mmHg	129.5 ± 14.7	129.2 ± 13.4	0.294
Asleep SBP mean, mmHg	118.0 ± 16.6	114.7 ± 14.6	<0.001
48 h SBP mean, mmHg	125.6 ± 14.5	124.3 ± 12.9	<0.001
Sleep-time relative SBP decline, %	8.5 ± 8.4	12.2 ± 7.7	<0.001
Awake DBP mean, mmHg	76.7 ± 10.6	76.3 ± 10.0	0.124
Asleep DBP mean, mmHg	66.1 ± 10.1	64.5 ± 9.3	<0.001
48 h DBP mean, mmHg	73.1 ± 9.9	72.2 ± 9.2	<0.001
Sleep-time relative DBP decline, %	13.3 ± 9.4	15.3 ± 8.6	<0.001
Non-dipper, %	50.3	37.5	<0.001

Values are shown as mean ± SD, unless otherwise indicated. Sleep-time relative BP decline, index of BP dipping, defined as percentage decrease in mean BP during nighttime sleep relative to mean BP during daytime activity, calculated as: [(awake BP mean - asleep BP mean)/awake BP mean] × 100. Non-dipper: individuals with sleep-time relative SBP decline <10%, using data sampled by ABPM for 48 consecutive hours.

*Values correspond to the average of at least three conventional BP measurements obtained per participant in the morning at the clinic after resting ≥10 min before initiating 48-h ABPM.

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-II receptor blocker; CCB, calcium channel blocker; DBP, diastolic blood pressure; PP, pulse pressure (SBP-DBP); SBP, systolic blood pressure.

Événements CV

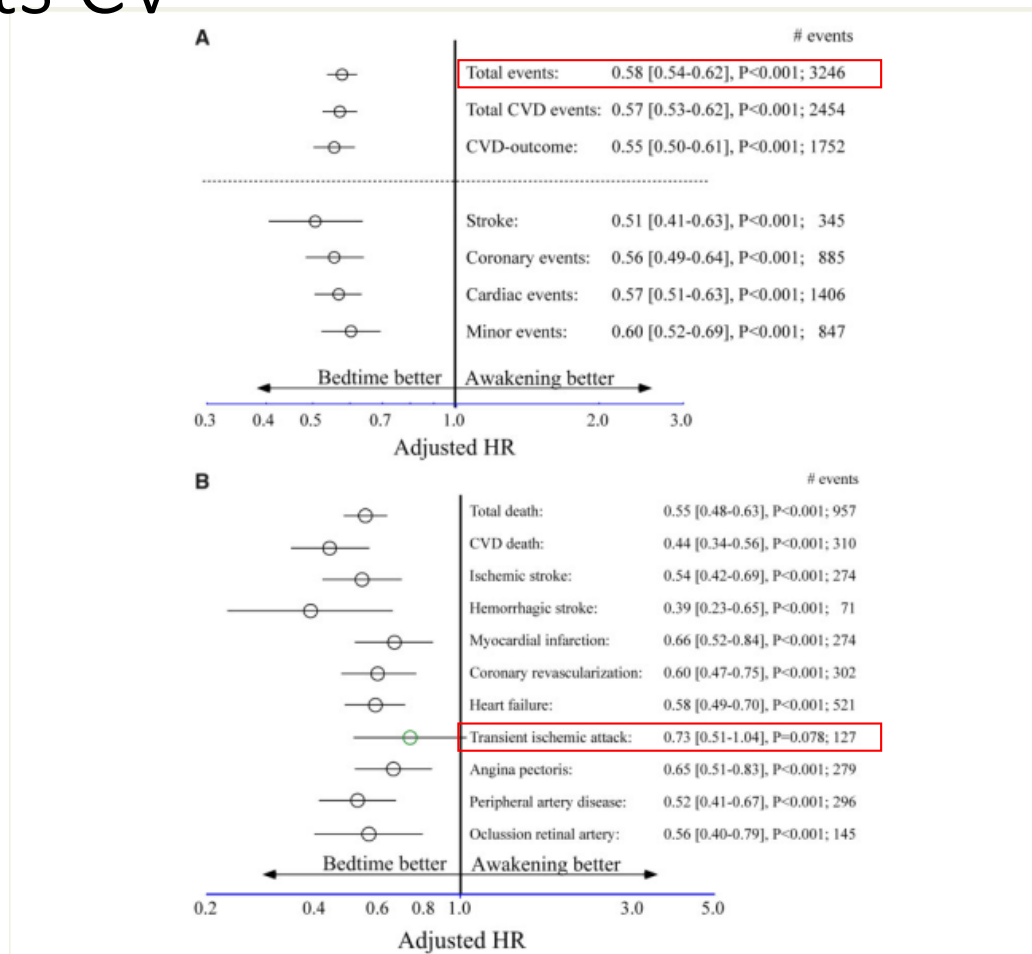


Figure 2 Adjusted hazard ratio of cardiovascular disease outcome as a function of hypertension treatment-time regimen (either upon awakening

Événements CV

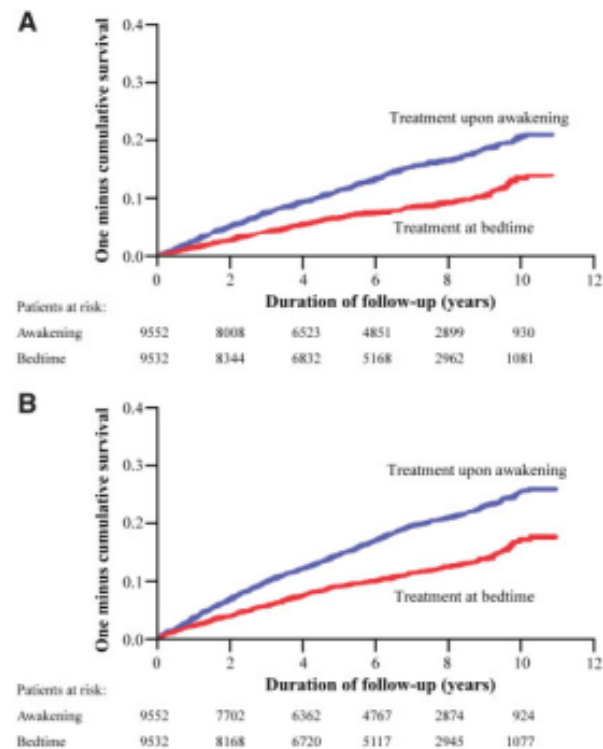


Figure 3 Kaplan–Meier cumulative hazard curves for cardiovascular disease outcome as a function of hypertension treatment-time regimen (either upon awakening or at bedtime). (A) Cardiovascular disease outcome: composite of cardiovascular disease death, myocardial infarction, coronary revascularization, heart failure, and stroke; log-rank: 140.1, $P < 0.001$. (B) Total cardiovascular disease events: composite of cardiovascular disease death, myocardial infarction, coronary revascularization, heart failure, stroke, angina pectoris, peripheral artery disease, and transient ischaemic attack; log-rank: 174.0, $P < 0.001$.

Co-variables

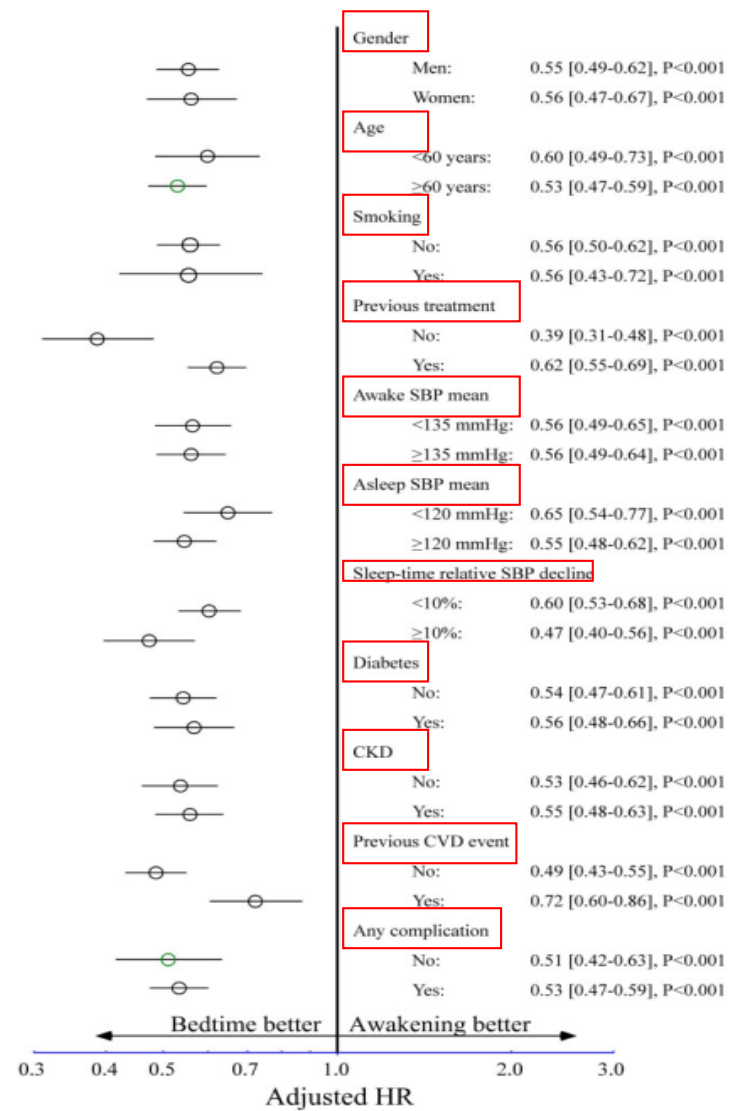


Figure 4 Adjusted HR (95% CI) of the primary cardiovascular disease outcome (cardiovascular disease death, myocardial infarction, coronary

Autres résultats

- Observance 97,2 % (Morisky)
- Pas de différence significative pour effets indésirables :
 - 6,7 (jour) vs 6 % (nuit)
- 0,3% d'hypotension nocturne (TAD < 50 mm Hg)

Discussion

- Méthodologie rigoureuse
- Résultats impressionnants
- Espagne:
 - Heures des repas et activité différent de l'Amérique
 - Pas un événement synchronisant majeur des rythmes biologiques
- Bien toléré
- Oui, oui, diurétiques au coucher !



Anti-Hypertensive Effect of Sacubitril/Valsartan: A Meta-Analysis of Randomized Controlled Trials

Renato De Vecchis^{a, b}, Silvia Soreca^a, Carmelina Ariano^a

Introduction

- Sacubitril est inhibiteur neprylisine centrale. Pas effet anti-hypertenseur en soi
 - Allonge t $\frac{1}{2}$ BNP et ANF, angiotensine II et endotheline
- Crainte neurotoxicité long terme par accumulation beta-amyloïde
- Risque TNC acceptable en ICC sévère, mais en HTA ?
- Expérience en ICC révèle risque faible de neurotoxicité.
- Place dans traitement HTA systolique isolée chez personne âgées de plus en plus étudié

Méthode

- Méta-analyse, méthode PRISMA
- Études publiées jusqu'en juin 2018
- Pts $\geq$ 55 ans
- Gr contrôles avec IECA/ARA ou BCC ou Beta-bloquant
- Variables:
 - Efficacité TAS, TAD, TAM par MAPA
 - Sécurité: Effets indésirables

5 études
 1513 patients
 Dose 100-400 mg/jour !
 Durée 4 à 52 sem:
 moy 95 ± 82 jours
 Comparateurs
 Olmesartan (3)
 Valsartan (2)

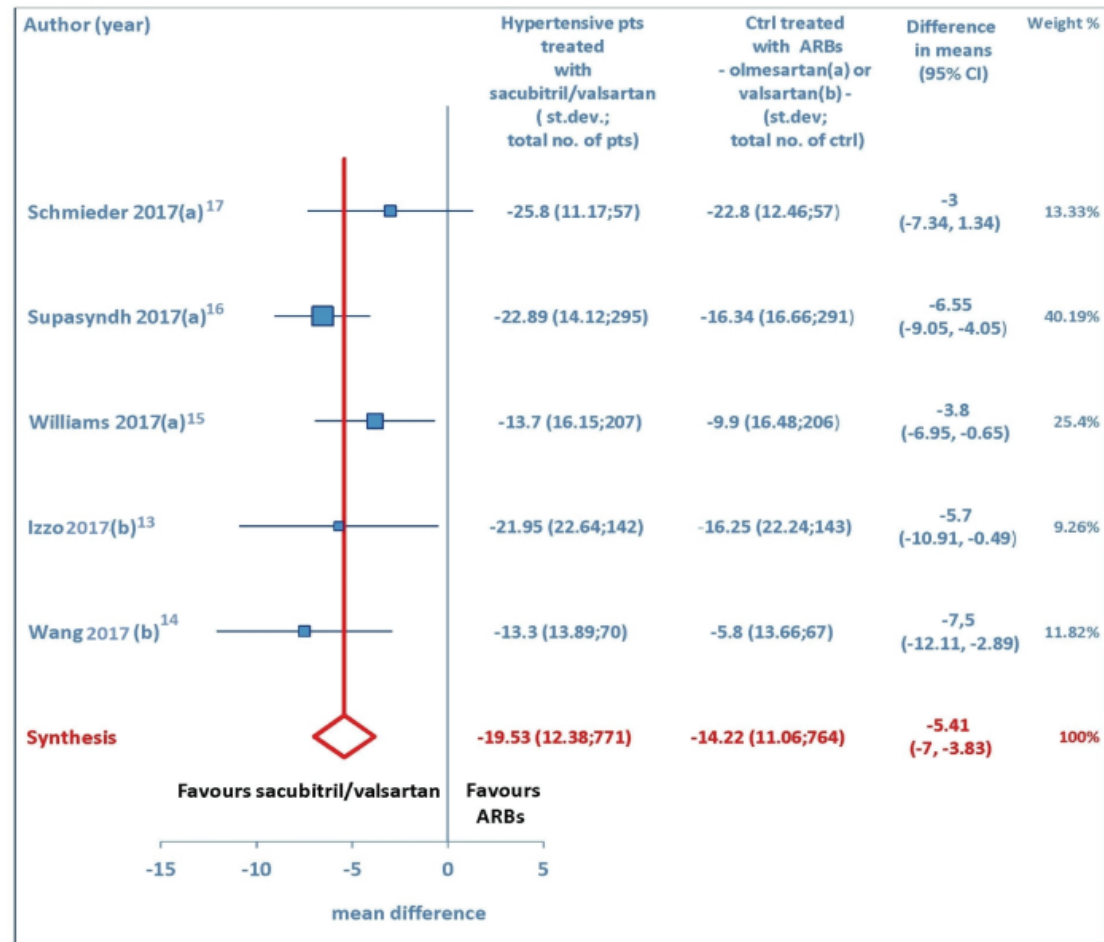


Figure 2. Mean reduction (mm Hg) in sitting systolic blood pressure (msSBP) in hypertensive patients treated with sacubitril/valsartan compared to ARB-treated controls. (a) In the control group olmesartan was used as comparator drug. (b) In the control group valsartan was used as comparator drug. pts: patients; st.dev: standard deviation; ctrl: controls; ARBs: angiotensin receptor blockers; CI: confidence interval.

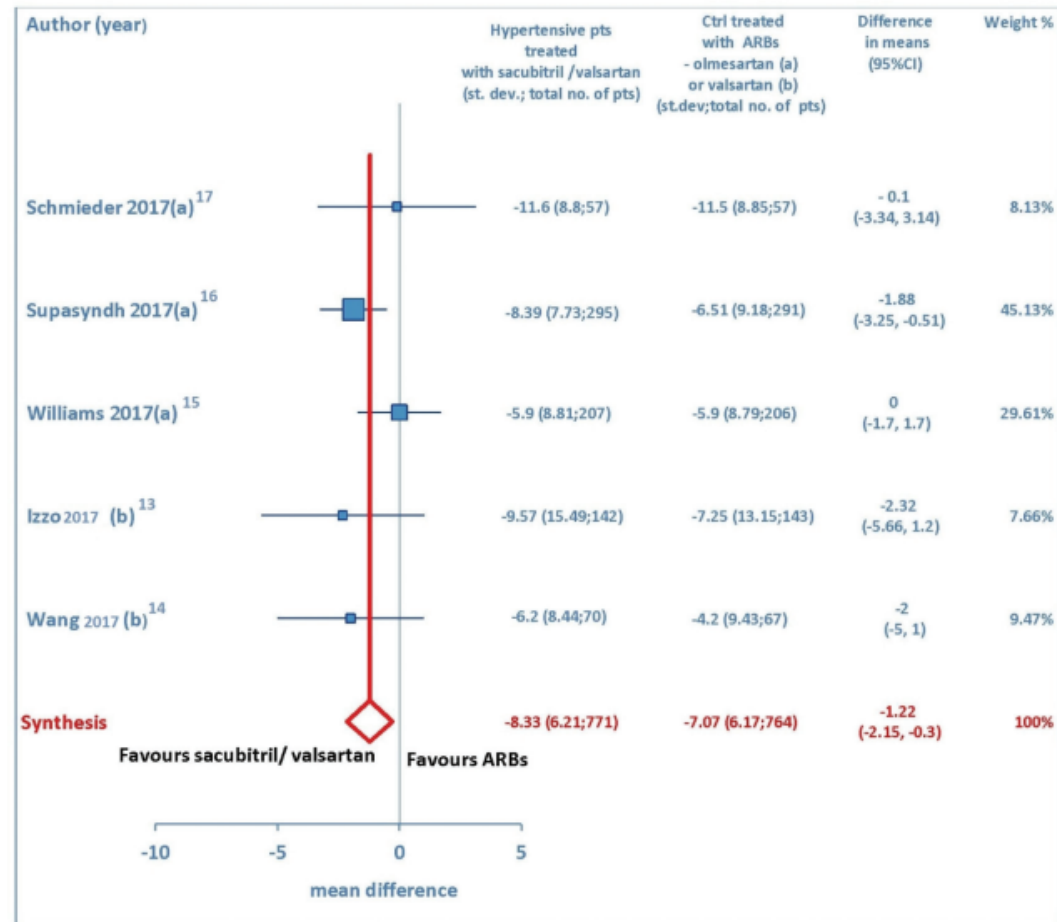


Figure 3. Mean reduction (mm Hg) in sitting diastolic blood pressure (msDBP) in hypertensive patients treated with sacubitril/ valsartan compared to ARB-treated controls. (a) In the control group olmesartan was used as comparator. (b) In the control group valsartan was used as comparator. pts: patients; st.dev: standard deviation; ctrl: controls; ARBs: angiotensin receptor blockers; CI: confidence interval.

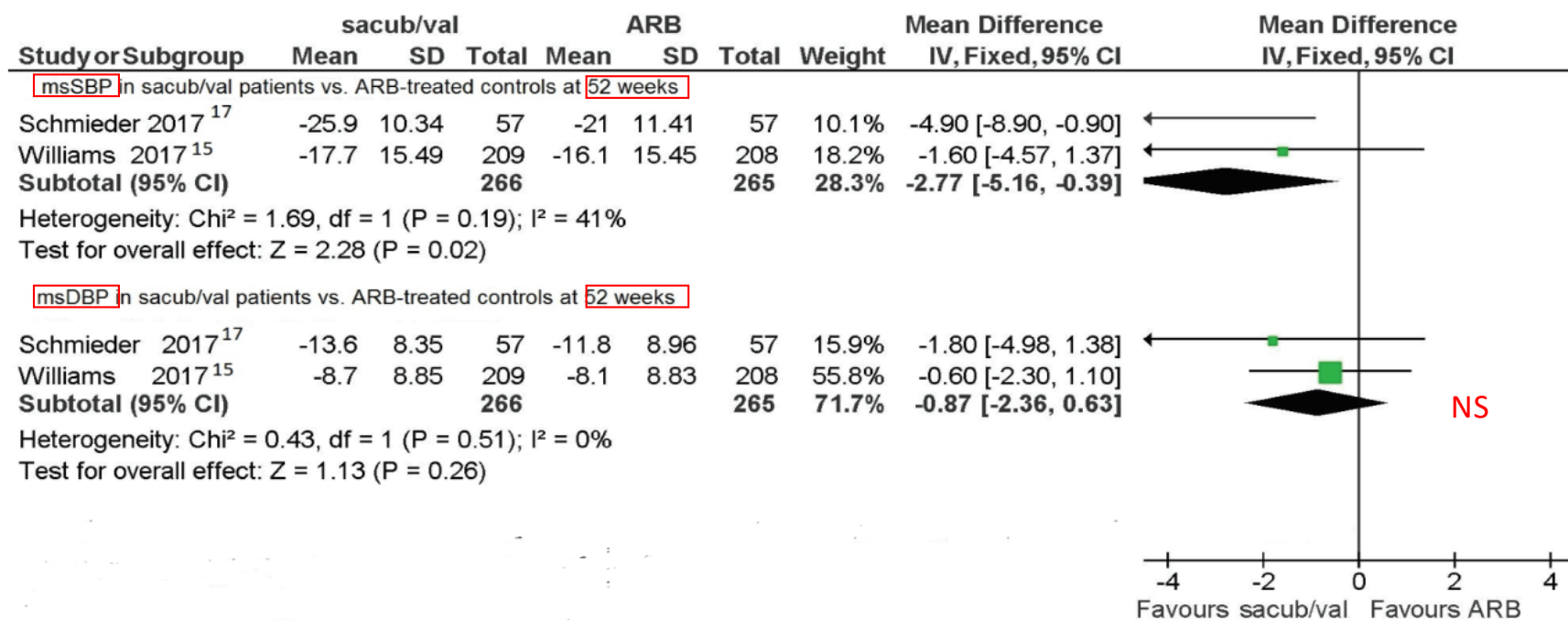


Figure 4. Mean sitting blood pressures (msSBP and msDBP) in sacubitril/valsartan-treated patients vs. ARB-treated controls.

Effets indésirables

- 37,6% sacubitril/valsartan vs 28,7% ARA
- Principaux: nasopharyngite, hyperuricémie, IVRS, étourdissements
- TNC ???

Discussion

- Dose plus élevées qu'en ICC
- Période observation courte pour exclure les effets indésirables recherchés.
- Analyses de sensibilité ne démontrent pas de biais significatifs.
- Bénéfice modeste
- Effets indésirables d'intérêt non rapportés
 - N probablement trop petit



Original Article

Long Working Hours and the Prevalence of Masked and Sustained Hypertension

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Introduction

- Effet de longues heures de travail sur TA inconsistent
 - > 48 hr/sem
- Peu réalisées avec MAPA
- Pas de données sur prévalence HTA masquées dans ce groupe
- Objectifs
 - Examiner l'association entre longues heures de travail et prévalence HTA masquée et HTA soutenue

Méthode

- 3547 col blancs
- 5 ans: 1,3 et 5
- 6733 observations
- HTA masquée: TA clinique $< 140/90$ mm Hg et MAPA $\geq 135/85$ mmHg
- HTA soutenue: TA clinique $\geq 140/90$ mm Hg et MAPA $\geq 135/85$ mmHg

Hypertension masquée

Table 3. Prevalence Ratio for Long Working Hours and Masked Hypertension

Work Hours, h/wk	Prevalence (%)	Model 1		Model 2		Model 3	
		PR	95% CI	PR	95% CI	PR	95% CI
<35	14.7	1.00	...	1.00	...	1.00	...
35–40	16.7	1.14	0.91–1.43	1.08	0.86–1.35	1.05	0.84–1.33
41–48	28.2	1.93	1.36–2.73	1.54	1.09–2.19	1.51	1.06–2.14
≥49	36.4	2.48	1.56–3.96	1.76	1.12–2.77	1.70	1.09–2.64

PR indicates prevalence ratio. Reference category: normotension; Model 1: crude; Model 2: adjusted for age, sex, education and occupation; and Model 3: Model 2+body mass index, alcohol intake, smoking, sedentary behaviors, diabetes mellitus, family history of CVD and job strain.

Hypertension soutenu

Table 4. Prevalence Ratio for Long Working Hours and Sustained Hypertension

Work Hours, h/wk	Prevalence, (%)	Model 1		Model 2		Model 3	
		PR	95% CI	PR	95% CI	PR	95% CI
<35	21.0	1.00	...	1.00	...	1.00	...
35–40	21.7	1.04	0.86–1.26	1.07	0.89–1.27	1.04	0.87–1.23
41–48	32.9	1.57	1.14–2.15	1.42	1.07–1.88	1.33	0.99–1.76
≥49	40.4	1.94	1.28–2.93	1.76	1.17–2.65	1.66	1.15–2.50

PR indicates prevalence ratio. Reference category: normotension; Model 1: crude; Model 2: adjusted for age, sex, education and occupation; and Model 3: Model 2+body mass index, alcohol intake, smoking, sedentary behaviors, diabetes mellitus, family history of CVD and job strain.

Discussion

- Validité externe cols bleus ?
- Impact de l'exercice physique et le surpoids
- Pas évaluation MAPA nocturne
- Invite à l'identification précoce de groupes de patients à risque surtout pour HTA masquée.



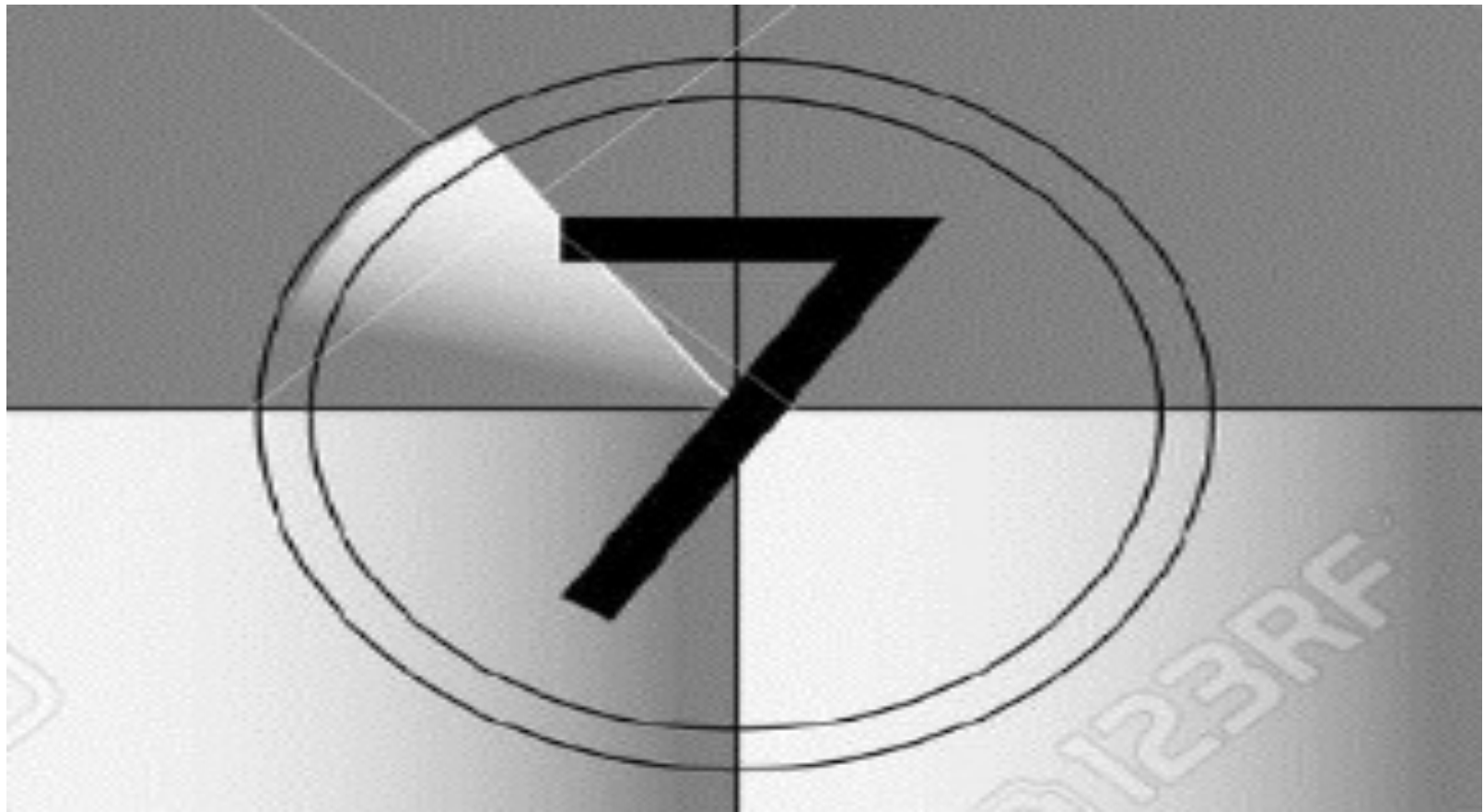
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Ronald B. Melles, M.D., and Deepak L. Bhatt, M.D., M.P.H.

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Eplerenone Versus Spironolactone in Resistant Hypertension: an Efficacy and/or Cost or Just a Men's Issue?

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Merci !