

Διαχείριση της 'LABILE' υπέρτασης



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188

93

140

93

120

90

120

80

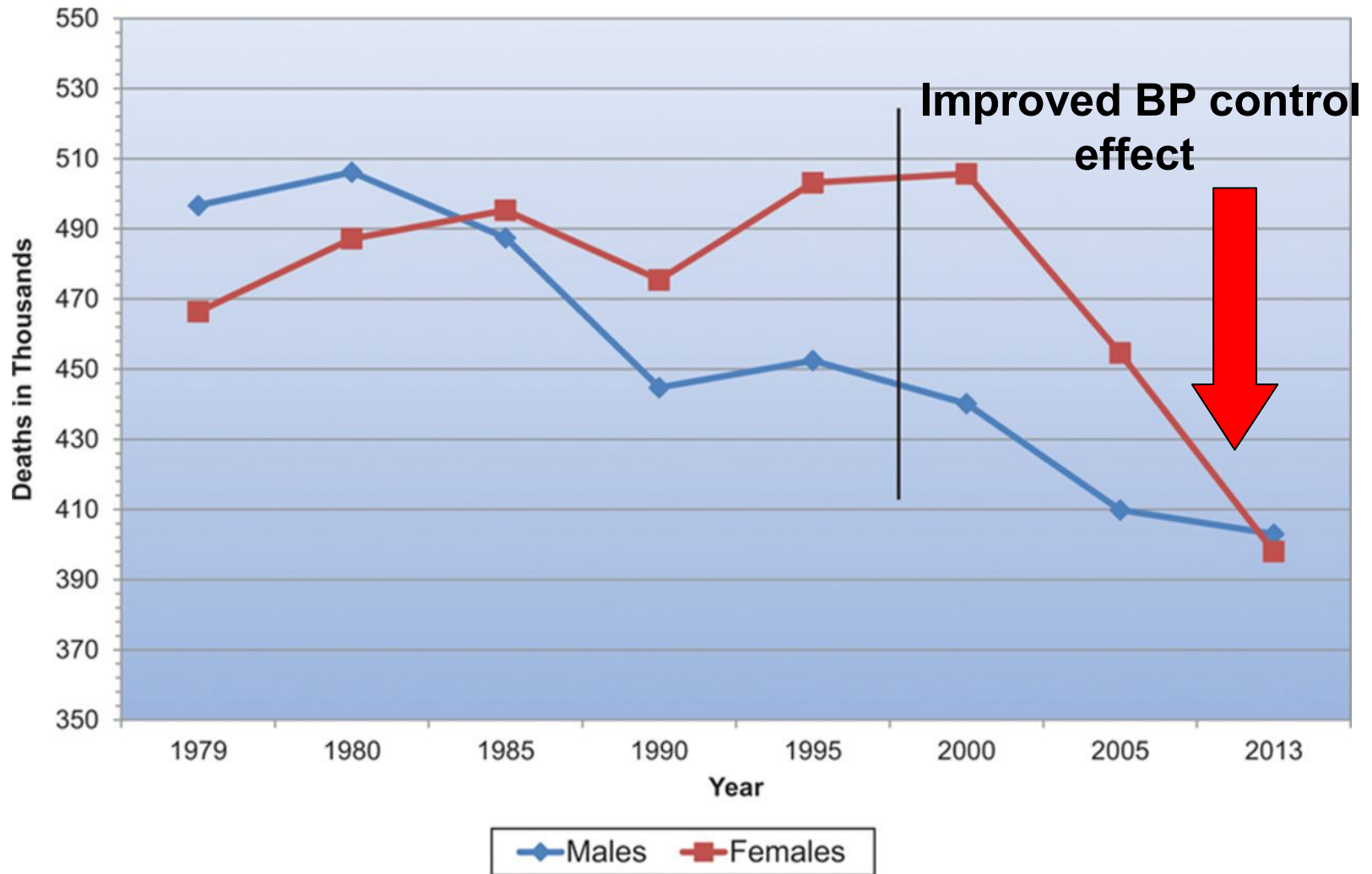
177

102

bonafide

bonafide

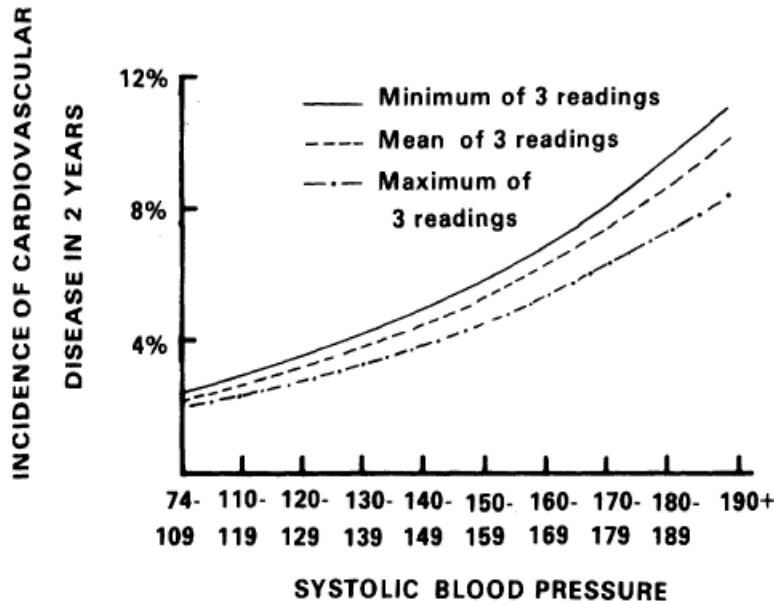
Cardiovascular disease mortality trends for males and females (United States: 1979–2013).



Labile Hypertension: A Faulty Concept?

The Framingham Study

W. B. KANNEL, M.D., PAUL SORLIE, M.S., AND TAVIA GORDON



Labile BP elevation has less clinical significance than **“fixed Hypertension”**

The mean, minimum and maximum of 3 pressures measured during an examination were equally efficient predictors of CVD.

Review Paper

The Clinical Spectrum of Labile Hypertension: A Management Dilemma

Samuel J. Mann, MD

Ironically ,in the 1960s through the 1980s,the term labile hypertension was defined between normotension and sustained hypertension ,with readings varying above and below the cutoff points of 140/90 mmHg.

Journal of Hypertension 2009

Blood Pressure Lability as a Clinical Dilemma

The alerting phenomenon (white coat hypertension)

Labile hypertension (including preprocedural hypertension)

Paroxysmal hypertension

Normal lability in patients with vulnerable underlying conditions

Cerebral aneurysm

Chronic aortic dissection

Amyloid angiopathy

Marfan syndrome

Angina

The alerting Phenomenon

The alerting phenomenon is the tendency of BP to rise at the time of measurement, but NOT always due consciously anxiety over the measurement - WCH.

Studies shows that pts with WCH do not have abnormal lability outside of physicians office. WCH is associated with lower CV risk than is sustained hypertension but the organ damage and CV risc is greater than in normontesive pts.

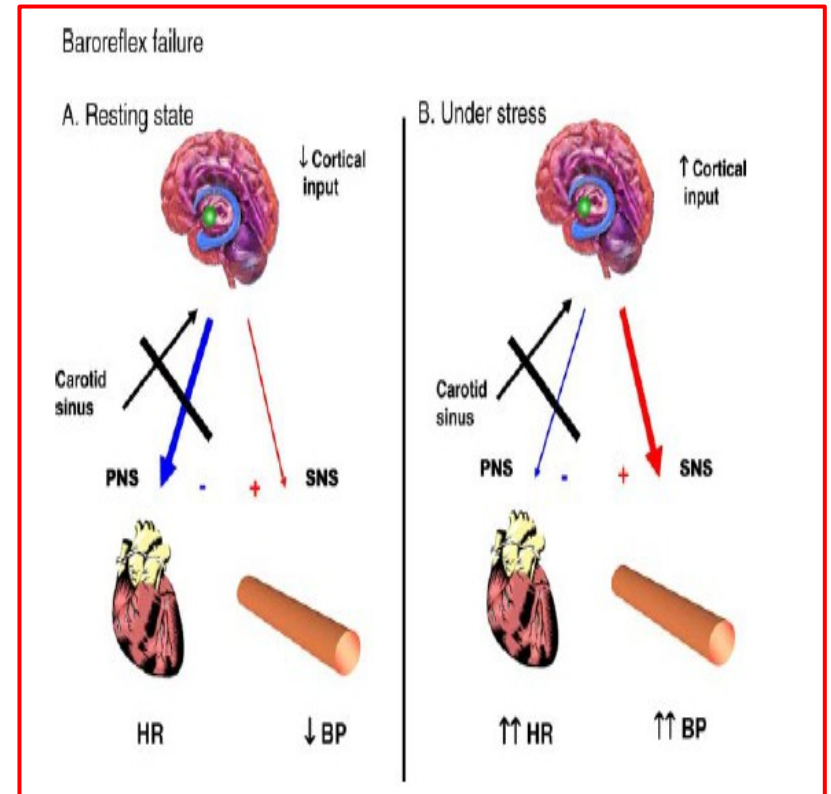
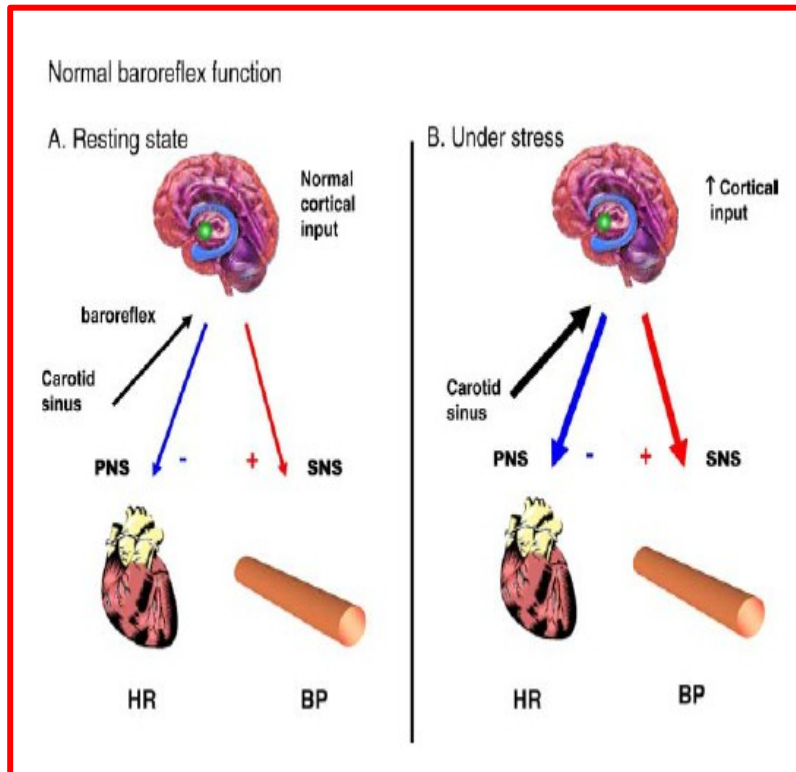
Labile Hypertension

The rise of BP is due: Anxiety, emotional distress and are likely mediated by sympathetic activation.

Palpitations, headache or flushing but usually are asymptomatic.

The BP falls spontaneously without intervention.

Baroreflex function




Labile Hypertension

- Labile hypertension may be due to hyperthyroidism, renovascular hypertension, seizure disorder, migraine, alcohol withdrawal, carcinoid syndrome, panic disorder, baroreflex failure, or drugs such as cocaine, amphetamines, or clozapine.
- Baroreflex failure should be considered in the differential diagnosis of labile hypertension, especially in subjects with a history of neck injury or neck irradiation.
- Treatment of baroreflex failure is aimed at reducing blood pressure surges and avoiding hypotensive episodes.

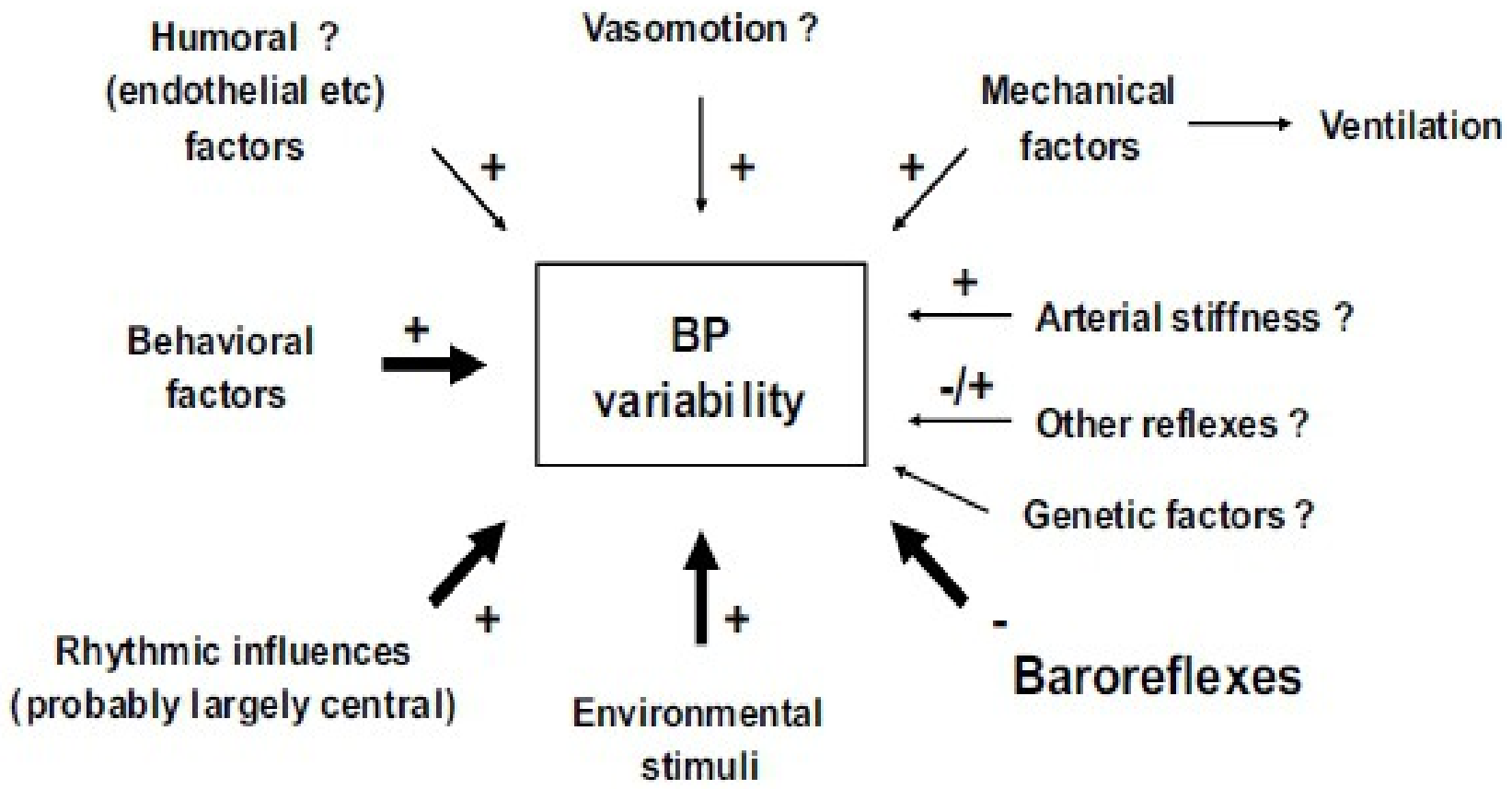
Labile vs paroxysmal HT

Differing Manifestations of Labile vs Paroxysmal Hypertension

LABILE HYPERTENSION	PAROXYSMAL HYPERTENSION (PSEUDOPHEOCHROMOCYTOMA)
<p>Can be asymptomatic or symptomatic</p> <p>Usually triggered by a stressor and accompanied by emotional distress experienced by the patient</p> <p>Patient usually links the blood pressure elevation to emotional distress</p>	<p>Markedly symptomatic</p> <p>Usually appears out of the blue without antecedent emotional distress</p> <p>Patient usually insists that the blood pressure elevation is not related to emotional distress</p>



Factors involved BP variability



Treatment Labile Hypertension

Treatment of BP Variability and BP Reactivity

Alerting Phenomenon

Preprocedural BP

Paroxysmal Hypertension

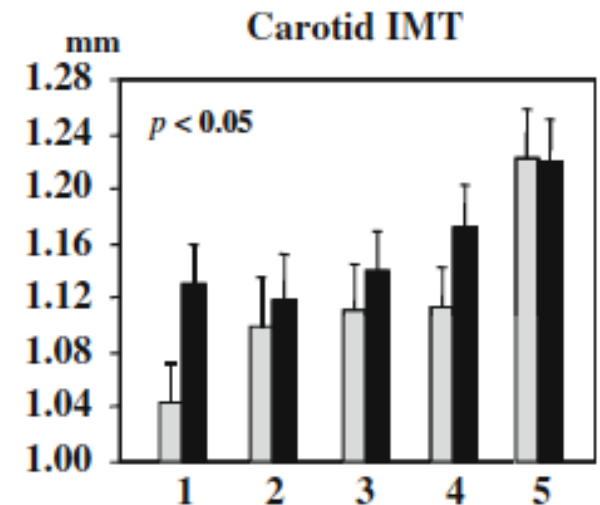
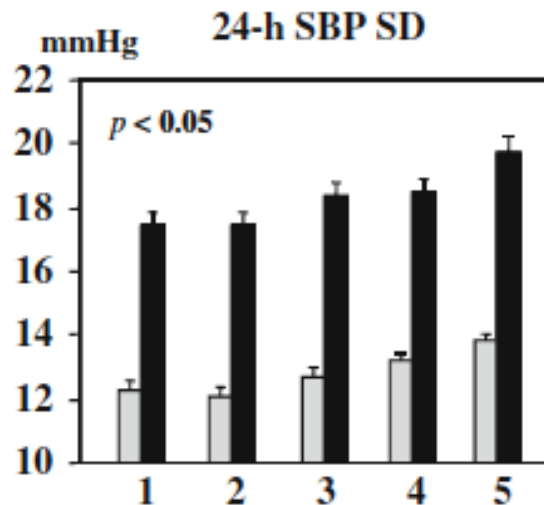
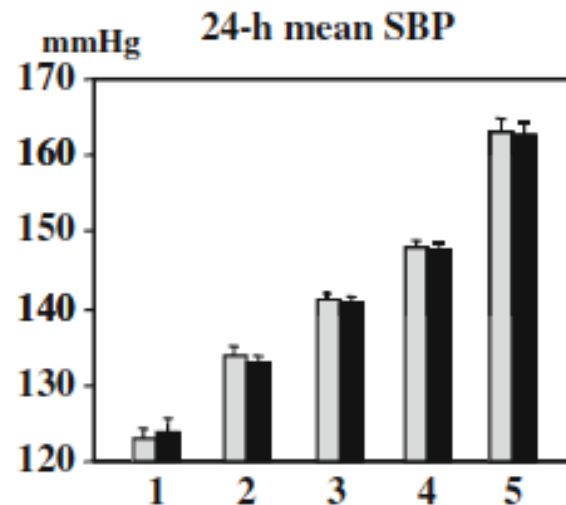
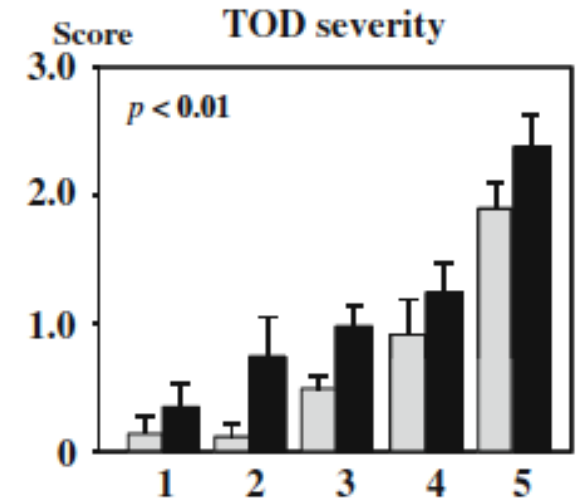
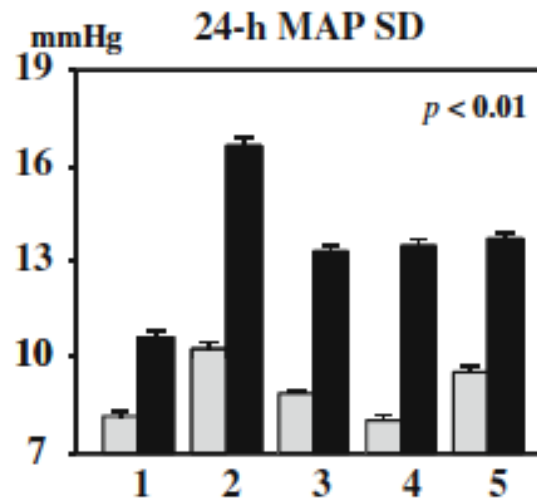
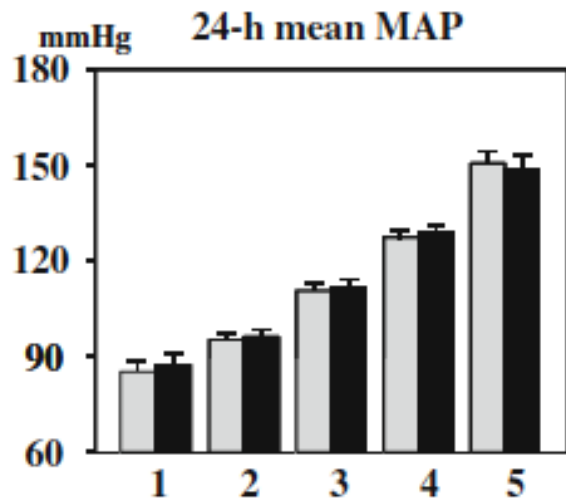
Acute management of Hypertensive paroxysms

Treatment pts with vulnerable underlying condition

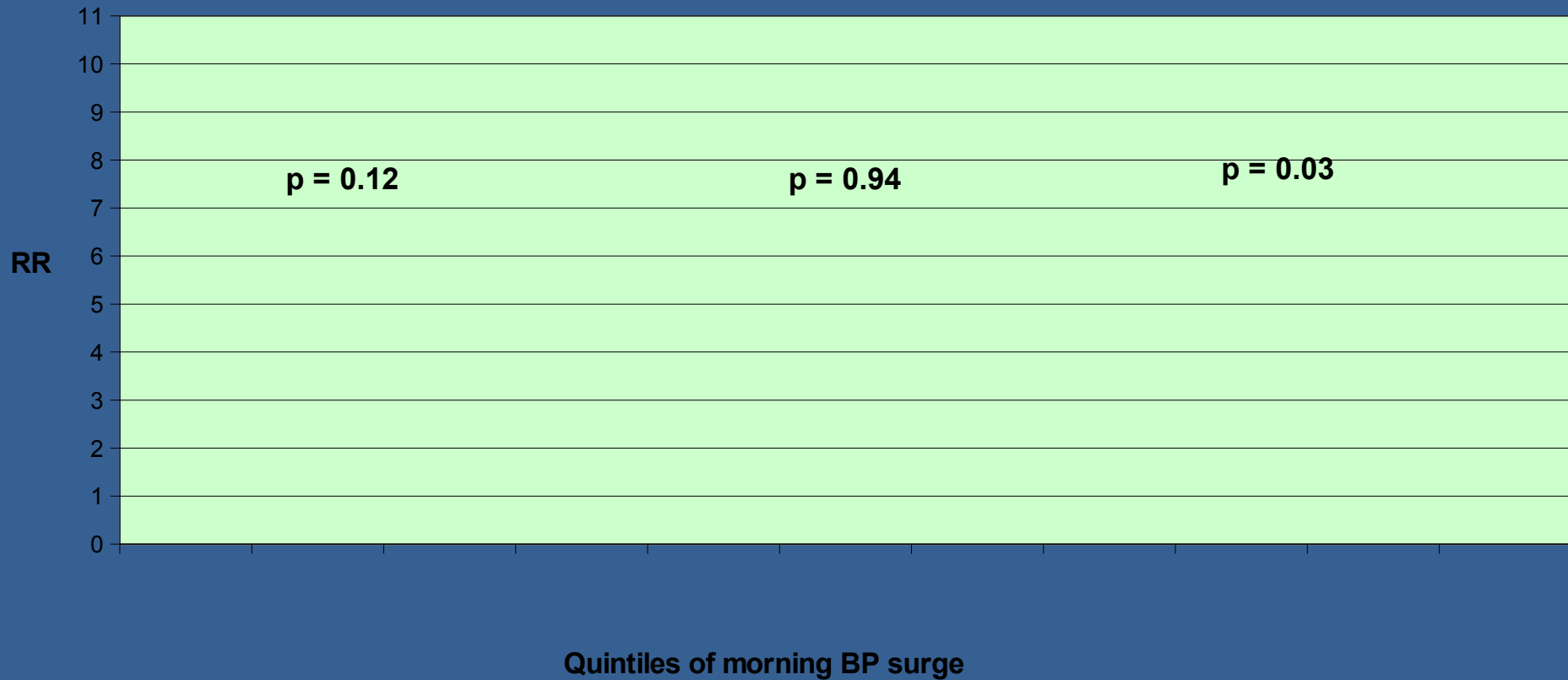
Clinical Relevance of 24h BP Variability

Study	Design	Endpoint
● Parati, 1987	Cross-sectional	TOD score
● Palatini, 1992	Cross-sectional	TOD score
● Mancia, Parati, 2001	Cross-sectional	Carotid IMT
● Liu, 2003	Longitudinal (rats)	Cardiac /renal damage
● Frattola, Parati, 1993	Longitudinal	LV mass (echo)
● Sander, 2000	Longitudinal	Carotid IMT / CV events
● Dawson, 2000	Longitudinal	Dead / dependency (after acute stroke)
● Kikuya, 2000	Longitudinal	CV mortality
● Pringle, Parati, 2003	Longitudinal	Stroke
● Mena, 2005	Longitudinal	CV events
● Mancia, 2007	Longitudinal	CV mortality
● Tatasciore, Parati, 2007	Cross-sectional	Carotid IMT, LVMI
● Parati, 2009	Longitudinal	CV events
● Hansen, 2010	Longitudinal	Only DBP for CV events / stroke

Relationship between 24-h BP variability and organ damage in hypertension.

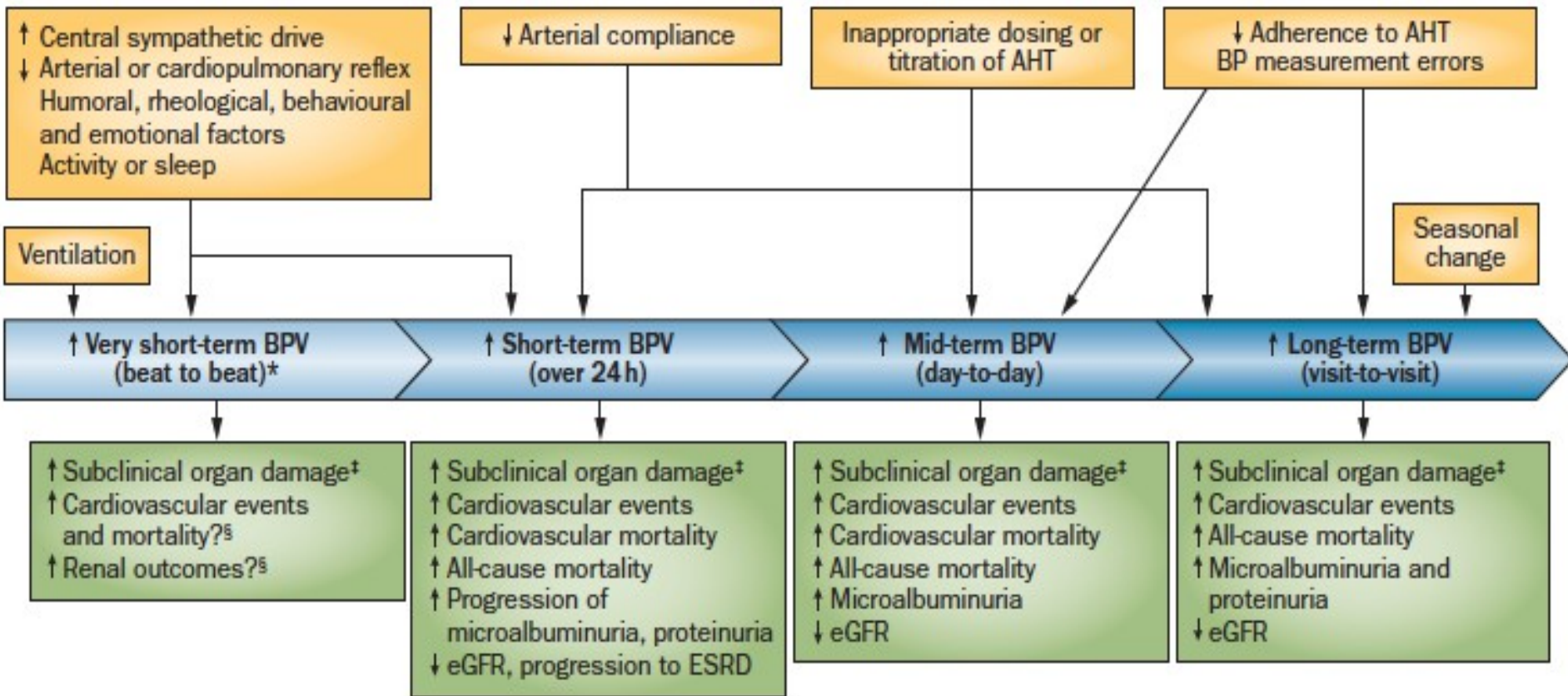


Relation Between Risk of Stroke and Morning BP Surge In The Ohasama Study



N = 1430

BPV: Types and Prognostic Significance



Question to be answer.....

- Average BP levels and/or BPV? Both
- Short or Long Term BPV? Mainly short
- Which measure of BPV to choose? SD
- Relationships with physiological variables (e.g. endothelial function, SpO2, arterial stiffness)? Some correlations exist ?
- BPV in Risk Stratification?

Question to be answer.....

- Is a drug-induced reduction in BPV accompanied by a reduction in event rate?
- Do different drug classes have a different effect on BPV and on outcome?
Probably CCBs and long-acting ARBs?
- Is there enough evidence to consider BPV as a new target for treatment?

Question to be answer.....

- 24 h BP values more closely related to TOD and future events than office readings
- **Higher 24 h BP Variability = Higher CV risk**
- Long lasting CCBs seem to score best in reducing short term and long term BPV
- Prospective outcome studies needed to confirm that treatment-induced reduction in BPV improves outcome.

Allerting phenomenon -WCH

- The treatment of WCH ,It would seem unneccesary ,and perhaps harmful
- Treatment should be aimed at reducing home BP,IF it is elevated using the usual pharmacologikal agents

Preprocedural BP elevation

- Prophylactic BP elevation

Oral α -blocker plus β -blocker for 2-3 days
and or anxiolytic agent

Acute management IV labetalol and or
anxiolytic agent

Severe labile hypertension

- Acute management

Indications for acute management not established

Consider IV labetalol if severe or symptomatic elevation

Chronic management

Oral α -blocker plus β -blocker + other agents if resting BP is elevated

Runaway anxiety about BP readings

Acute management NO

Chronic α -blocker plus β -blocker

Paroxysmal hypertension

- Acute management of paroxysms:
Severe paroxysm e.g BP >220mmHg or diastolic BP>130mmHg
IV Labetalol or nitroprusside
- Milder paroxysms:
Central α -agonist e.g Clonidine and/or anxiolytic or α -blocker plus
b-blocker with fairly rapid onset e.g labetolol or metoprolol
plus prazosin
- Chronic preventive management:
Less intensive regimen: α -blocker plus b-blocker
Antidepressant agent -selective serotonin reuptake inhibitor or
tricyclic agents.

Normal lability in pts with vulnerable underlying conditions

- Oral α -blocker + β -blocker combined with other agents to achieve lowest acceptable resting BP.

Take home message

- Although the management of labile forms of hypertension is frequently encountered clinical dilemma specific criteria or clinical trials **do not exist** .
- Studies to assess the effects of labile hypertension and outcome of treatment are needed
- The clinical spectrum of this problem and a treatment approach based on published reports and physiologic principles has been presented.