syncope. HUT was performed according to the Italian protocol, including NTG provocation in 25 patients. During follow-up lasting 1-2 years in 6 patients syncope recurred (group I) and in 26 patients did not (group II).
Results: the delayed rise in aldosterone concentration was established in 7 patients including 4 patients in group I and 3 patients in group II. Plasma concentration of aldosterone (in pg/ml) in studied periods are shown in the table:

	Delayed ALDO increase	Normal ALDO increase	p
ALDO I	87±34	99±86	NS
ALDO II	98±36	226±171	<0,05
ALDO III	187±125	254±119	NS

There was no correlation between delayed increase of aldosterone and NTG provocation during HUT. The delayed increase of aldosterone observed in 22% of all patients was related to syncope recurrence during follow-up, but not to the phase of tilt in which syncope occurred.
Conclusions: 1. The delayed increase of aldosterone concentration during HUT-induced syncope is a risk factor of syncope recurrence during follow-up.
2. The delayed increase of aldosterone could be the marker of disturbed activation of RAA system predisposing the patients to vasovagal syncope.

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Predictive models of neurovegetative and rhythmic causes of syncope: fact or fantasy?

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Background: contradictory results have been reported concerning the diagnostic yield of history in unexplained syncope (un-Sy). Our study is aimed at developing a predictive model of syncope (Sy) causes in patients (pts) referred to a Sy clinic.
Methods: 317 consecutive pts with un-Sy referred to our Sy clinic underwent a targeted work-up including a 12-lead ECG, physical examination, detailed history with screening for 22 warning symptoms (WS) followed by carotid sinus massage, head-up tilt test and electrophysiogical study when indicated. A putative cause was identified in 79% of the pts grouped into: neurovegetative (NV) in 41% (vasovagal 24%, psychogenic 17%), rhythmic (RHY) in 27% (brady 20%, tachy 7%), hypotensive in 8%, situational in 3% and unexplained in 21%.
Results: of the 22 WS, only 11 were significantly related to outcome. Logistic analysis identified age and number of significant WS as the only significant factors predictive of RHY causes. Combining these factors in a RHY model classified 166/317 (52%) pts as a RHY syncope, with a sensitivity of 91%, a specificity of 62%, a positive predictive value (PPV) of 46% and a negative predictive value (NPV) of 95%. Similarly, logistic analysis identified age, number of significant WS and duration of P wave as the only predictive factors of NV causes. The three parameters, combined in a NV model, classified all 151 remaining pts as a NV syncope, with a overall sensitivity of 85%, a specificity of 77%, a PPV of 72% and a NPV of 89%.
In conclusion, a two-stage rule based on a limited number of clinical and paraclinical parameters allows to identify most rhythmic and neurovegetative causes of syncope. Importantly, this approach might help define the best investigation and cost-effective strategy in patients with unexplained syncope.

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The bradycardia-hypotension reaction during head-up tilt test: clinical and hemodynamic characteristics related to blood pressure and heart rate behavior during the early stage of the test

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An abrupt bradicardia-hypotension reaction (B-H) during tilt test (TT) may occur after different hemodynamic patterns during the early stages of the test.
In order to evaluate the clinical and hemodynamic characteristics related with these initial patterns, 138 pts with ≥1 syncope or multiple presyncope episodes and with B-H reaction (age 38.1±21.9 years, 50% male) were evaluated. The early pre-established patterns of BP and HR were: orthostatic hypotension (OH): drop in systolic BP ≥ 20 mmHg (SOH) or ≥10 mmHg drop in diastolic BP (DOH) within the first 5 minutes of orthostatism; Postural tachycardia (PT): HR increase ≥ 30 bpm within the first 5 minutes orthostatism and HR > 120 bpm in the absence of hypotension, Progressive decrease (PD): slow and continous fall in BP until the end of the TT; Normal Pattern (NP): none of the above abnormalities is present.
Results (see table):

	NP	SOH	DOH	PD	PT
Age *	37 ± 21	58 ± 18	50 ± 25	36 ± 22	22 ± 11
C0-M (%)	45	87	50	50	25
HT (%) *	14	50	42	14	0
Drugs (%) *	15	44	25	21	8
Prodroms (%)	89	60	66	100	75
Episodes (median)	3	4	3	3.5	2
Time to B-H	25 ± 13	21 ± 14	20 ± 14	20 ± 12	19 ± 14
Early (+) resp (%)	15	12	17	21	25
SBP (B) *	114 ± 19	145 ± 29	134 ± 25	123 ± 20	114 ± 15
DBP (B) *	69 ± 14	81 ± 16	83 ± 12	72 ± 12	70 ± 11
HR (B)	69 ± 11	74 ± 16	73 ± 8	64 ± 8	75 ± 10
Mx HR	94 ± 17	85 ± 19	94 ± 17	91 ± 17	130 ± 10

Co-M: co-morbidity; HT: hypertension; Early (+): Positive response within the first 10 minutes; SBP (B): baseline systolic BP; DBP(B): baseline diastolic BP; HR (B): Baseline HR; Mx HR: Maximal HR.*= ANOVA $p < 0.05$

Conclusion: the distribution of time elapsed until the B-H reaction was similar for all patterns. The B-H reaction may occur in different clinical settings and may be preceded by any pattern. The patients with an initial orthostatic hypotension are older, have most co-morbid factors, a higher baseline BP and a slower maximal HR than the other groups.

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Cardiovascular effects and risk of syncope related to donepezil in Alzheimer's disease

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Purpose: When unexplained, syncope may be attributed to bradycardia caused by cholinesterase inhibitors (CI) in patients with Alzheimer's disease (AD). We studied prospectively the clinical events and cardiovascular changes occurring during treatment with donepezil in 30 consecutive patients with AD.
Methods: Clinical characteristics, blood pressure, heart rate and electrocardiogram were recorded before (baseline), and during treatment with donepezil, 5 mg/day for 1 month, 10 mg/day for 1 month, and 10 mg/day for 6 more months. We compared the baseline observations with those made at 1, 2 and 8 months. We also examined the effects of negatively