In patients (pts) with vasovagal syncope, counsels about driving remain a challenge. In fact, for most authors, vasovagal syncope does not represent a contra-indication for driving as it seldom occurs during this activity. However, very few data substantiate this attitude.

Methods:
In order to have some epidemiological data, we reviewed the history of the last 230 consecutive pts referred to our institution for a tilt test. Tilt test was performed for 45 min at an angle of 60°. If the test remained negative a drug challenge was initiated: isoproterenol 3 μg/min for the first 135 pts and nitrates 300 μg sublingually for the 95 remaining pts.

Results:
Among these 230 pts, 90 had a positive response during the test (syncope or at least severe pre-syncope associated with fall in blood pressure with or without bradycardia or asystole). Twenty four out of these 99 pts (24 %) had at least one syncope in a sitting position and 7 (7 %) while they were driving (loss of the car control in all cases without severe consequences). After a mean follow-up of 16 months (1-44 months) only 1 pt continued to have syncope (one syncope during 39 months). This pt reduced dramatically his driving time. The 6 remaining pts had no recurrence of syncope and continued to drive as frequently as they did before.

Conclusion:
Occurrence of vasovagal syncope during driving is not an exceptional event (although a reference center, this occurrence is probably biased in our series). However recurrences in similar circumstances, as in other series, have not been observed in our study group.

In our department tilt training is considered for patients with neurocardiogenic syncope (S). They are tilted daily (60° inclination), until S, or until a maximum of 45 minutes. Patients are discharged from the hospital after 2 consecutive negative tilt sessions. They are instructed to use a drug challenge (-I-I-r) positivity type and simultaneous EEG and transcranial Doppler.

Aim:
Table testing (-I-I-r) positivity type and simultaneous EEG and transcranial Doppler.

Method:
24 patients (pts) (12 F, age 15-74) with history of recurrent syncope and/or presyncope episodes underwent TTT with continuous blood pressure (BP) recording by Chmeda Finapres System; simultaneously EEG in all pts and transcranial Doppler sonography, performed only in two pts.

Results:
EEG: 12 pts (50 %) were positive, 8 for syncope and 8 for presyncope. In the syncope positive group: 5 were type 1A, 1 type 2B and 2 type “mixed”. In the presyncope positive group: 4 were type 1 “mixed”, 1 type 2B and 2 type 3. Two very different EEG patterns were identified: in 2A and 2B, during the prodromes, slowing and reduction in amplitude of electrical activity; in type “mixed” and in type 3, instead, EEG was unmodified with regard to the basal. During syncope in 2A and 2B, pseudorhythmic and then polymorphic delta activity was recorded, followed by “flat” EEG, in type “mixed”, instead, there was no disappearance of electrical activity, but only theta activity and then polymorphic delta. In type 3 there was no modification during presyncope. In post-syncope phase, in 2A and 2B there was slowed down activity and reduced amplitude similar to the prodromic phase, in 1 “mixed”, EEG returned to normal; respectively in type 3 EEG was unmodified on respect to the basal, as in prodromic and pre-syncope phase. Transcranial Doppler sonography, performed only in two positive pts, 1 type 2A and 1 type 1 “mixed”, demonstrated in both, at the onset of prodromic symptoms, a clear reduction of diastolic velocity in the middle cerebral artery, with increase of Pourcelet’s vascular resistance index (RI = systolic / diastolic / systolic velocity). These results suggest a significant decrease in cerebral blood flow just before the syncope phase. Conclusion: in 2A and 2B, during the prodromes, EEG alterations in the absence of bradycardia or hypotension were documented, permitting the hypothesis of cerebral vasoconstriction as the cause. EEG alterations in the post syncope phase recorded with heart rate and BP back to normal, can only be explained by cerebral vasoconstriction mechanism. It is possible that the most severe EEG alterations of the syncope phase in 2A and 2B, on respect to those in 1 “mixed”, are due to presence of cerebral vasoconstriction and bradycardia / asystole / hypotension.