I001
MANAGEMENT OF PATIENTS TREATMENT WITH HYPERTENSIVE EMERGENCIES REVIEW OF THE LITERATURE
M. ESCANDE 1
1 Centre hospitalier d’Allauch, Allauch, France
Hypertensive emergency is common but a severe clinical outcome in hypertensive patients.

The definition of the hypertensive emergency goal blood pressure is not consistent over the literature, but often involves a target organ damage in a situation of emergency.

Treatment practice in hypertensives crisis is difficult because of the lack of evidence supporting the use of one drug over another and a consensus on the posology.

After review of the medical literature, we found only four randomized trials comparing Nitroprussiinate to Uradipil or Nifedipine, or nifedipine to captopril. Those trials included only few patients, and Ib or Iib probe level without long term morbidity data.

In conclusion, the clinical practice is still far away from the Evidence-based Medicine. Clinical research must go further to prevent cerebral, cardiovascular or renal complications in hypertensive patients.

I012
TRANSFORMING GROWTH FACTOR-BETA ACTIVITY IN MACROPHAGES PROTECTS FROM ANGIOTENSIN II-INDUCED AORTIC ANEURYSM IN MICE
Y. WANG 1, H. AIT-OUFELLA 1, O. HERBIN 1, J. HUANG 1, P.-L. THARAUX 1, A. TEDGUI 1, Z. MALLAT 1
1 Inserm, Paris, France

Background — Complicated aortic aneurysm is a major cause of mortality in elderly men. The critical pathophysiological mechanisms responsible for disease development and complications remain largely unknown. Mutations in transforming growth factor (TGF)-β receptor type II are associated with familial forms of the disease, and increased angiotensin II (AngII)-dependent TGF-β activity has been directly linked to aortic aneurysm formation in a mouse model of Marfan syndrome. However, the direct role of TGF-β signaling in common forms of the disease has not been assessed.

Methods and Results — All-induced abdominal aortic aneurysm (AAA) is a validated model of aneurysm formation in mice, and is prevented, like murine Marfan syndrome, by treatment with angiotensin II receptor type 1 antagonists. Normocholesterolemic C57Bl/6 mice are resistant to All-induced AAA. Here, we show that systemic neutralization of TGF-β activity leads to unexpected and marked increase in the susceptibility of these mice to All-induced AAA (from ~10% to 92.5%, n=40). These AAA display a large spectrum of complications on echography, including thrombosis, fissuration, false channel formation and rupture, leading to a high level of mortality (65%). Unexpectedly, the disease and its complications were refractory to inhibition of IFN-γ (n=10, 100% AAA), deletion of IL-4 (n=10, 80% AAA) or deletion of T and B lymphocytes (n=22 Rag-/- mice, 77.5% AAA). Interestingly, depletion of circulating monocytes for 14 days using clodronate liposomes completely prevented AAA formation (0% AAA in clodronate group vs 60% AAA at day 14 in the group without clodronate).

Conclusions — This study identifies a major protective effect of TGF-β activity against AngII-induced AAA, through modulation of monocyte/macrophage function.