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 Nitroprussiate 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.

**METHODS AND RESULTS**

There were 20 to 160 mmHg, 20 mmHg per step). Stress (σ = ID x IP / 2WT) and strain (ε = (ID-ID0) / ID0 where ID0 is ID at 20 mmHg) were calculated. Stress-strain data were fitted to an exponential curve: σ = o0.e(ε.ET), where o0 is the stress at 20 mmHg and ET is the slope of the tangential elastic modulus versus stress.

**CONCLUSIONS**

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

**REFERENCES**

- F. DUPUIS, S. FOULQUIER, M. MAROCCHINI, M. DURAND, I. LARTAUD, J. ATKINSON, C. CAPDEVILLE-ATKINSON
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- A high salt diet (HSD) is a risk factor for stroke. However, little is known about the consequences of a HSD on the mechanical properties of cerebral vessels. The aim of the present work was to evaluate the impact of a HSD on the distensibility of the middle cerebral artery (MCA).

**METHODS**

- Normotensive male Wistar rats were given a normal diet (n=11) or HSD (n=10; 1 % NaCl in drinking water) for 1 month. Sodium balance was calculated as the difference between sodium intake and sodium excretion. MCA were mounted and pressurized (60 mmHg, i.e. 60 % of mean blood pressure, BP) in a small vessel arteriograph. Passive (inactivation of smooth muscle cells, EDTA 2 mM) internal diameter (ID) and wall thickness (WT) of MCA were measured during a stepwise increase in intraluminal pressure (IP, 20 to 160 mmHg, 20 mmHg per step). Stress (σ = ID x IP / 2WT) and strain (ε = (ID-ID0) / ID0 where ID0 is ID at 20 mmHg) were calculated. Stress-strain data were fitted to an exponential curve: σ = o0.e(ε.ET), where o0 is the stress at 20 mmHg and ET is the slope of the tangential elastic modulus versus stress.

In conclusion, a HSD induces an increase in wall stiffness of the MCA in the absence of any effect on BP. This may (partially) contribute to the cerebrovascular dysfunction linked to stroke in patients on a HSD.