Vol. 3, Suppl. to No. 3
September 1994

ISSN 1120-9879

254

HIGH BLOOD PRESSURE & Cardiovascular Prevention

XI National Congress of
the Italian Society of Hypertension

Abstracts of Oral and
Poster Presentations

Pisa, Italy - October 5-8, 1994



**The Official Journal of the
Italian Society of Hypertension**

Published by Editrice Kurtis s.r.l.
Via Luigi Zoja, 30 - 20153 Milano, Italy

HYPNOSIS AS A TOOL FOR EVALUATING THE CORTICAL COMPONENT OF HAEMODYNAMIC VARIATIONS. PILOT STUDY DURING BLOOD LETTING

Edoardo Casiglia, Augusto Rossi, A.Mazza, *G.Cavattoni, G.Colangeli, G.Ginocchio, G.DiMenza, *A.Marotti, C.Onesto, L.Pegoraro, A.C.Pessina.
Institute of Clinical Medicine, University of Padova, and *Trasfusional Center, Hospital of Padova, Italy

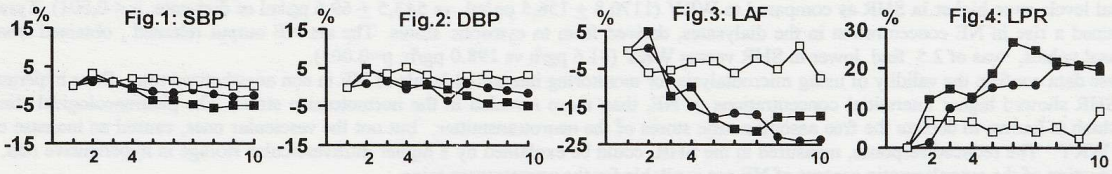
It is commonly accepted that cerebral activity influences cardiovascular functions via the sympathetic drive. This work is the first demonstration of the control exercised by «thinking» on peripheral flow and resistance.

Group A (real blood letting). In 20 consecutive healthy male blood donors aged 27±3 years, a venipuncture was performed followed by a 300 ml blood letting in 6 min. Before the beginning (needle insertion), in the middle (3 min) and at end (6 min) of this procedure, systolic and diastolic blood pressures (SBP and DBP) with a Riva-Rocci sphygmomanometer and leg arterial flow (LAF) with a strain-gauge plethysmograph were measured. Mean BP was calculated as MBP=DBP+(SBP-DBP)/3, and leg peripheral resistance (LPR) as MBP/LAF. Blood letting was followed by a 18-min recovery during which BP, LAF and LPR were measured every 3 min.

Group B (mere rituality). In 11 of the subjects from Group A, the rituality of phlebotomy was simulated after 4 weeks. Venipuncture was performed in the same room, with an identical needle, in the same position in bed, by the same doctors and nurses, and with the same timing of the blood letting. but without subtracting blood.

Group C (virtual blood letting). In 22 other healthy volunteers aged 27±2 years who underwent at least one previous blood letting in their life, blood letting was hypnotically simulated. An ideoplastic idea was induced, leading to the concrete realisation of a psychic experience having all the characteristics of a real blood letting. Virtual phlebotomy (6 min) and recovery (18 min) had the same real length of that of Groups A and B.

Results. The variations of SBP, DBP, LAF, and LPR in the three groups (real blood letting —■—, mere rituality —□—, virtual phlebotomy —●—) are shown in Fig.1, 2, 3, and 4 respectively. The BP variations were insignificantly different between the groups (F=0.64, p=0.53 for SBP; F=3.18, p=0.051 for DBP). LAF decrease and LPR increase had the same trend during both real and virtual phlebotomy (RM-ANOVA between Group A and C: F=2.16, p=0.15 for LAF; F=3.38, p=0.073 for LPR), while they were unchanged in Group B. In Figures, where the horizontal scale represents time (in min), 2 indicates the needle inserction (real in Groups A and B, virtual in Group C), and 4 the beginning of the recovery time.



Discussion. As the modifications induced by both real and hypnotically-simulated phlebotomy were quite similar, the psychic experience of the blood letting completely explains the haemodynamic changes induced by blood-letting, which are not due to the hydraulic subtraction of blood. The present results further suggest that a psychic component may be important in other situations and the method here followed seems to be the most appropriate to detect it.

ADRENERGIC RECEPTOR MECHANISMS AND ACE-INHIBITION IN SPONTANEOUSLY HYPERTENSIVE RATS

Maurizio Castellano, Damiano Rizzoni, Marina Beschi, Michael Böhm*, Enzo Porteri, Giorgio Bettoni, Massimo Salvetti, Angelo Cinelli, Enrico Agabiti-Rosei. Chair of Semeiotica e Metodologia Medica, U.O.P. Scienze Mediche, University of Brescia, Italy and * Chair of Internal Medicine, University of Köln, Germany.

In several experimental and clinical settings (e.g. in patients with heart failure) ACE inhibitors have been demonstrated to be able to modulate adrenergic nervous system hyperactivity. The spontaneously hypertensive rat (SHR) represents an experimental model of genetic hypertension characterized by increased sympathetic activity, activation of the renin-angiotensin-aldosterone system, and alterations of adrenergic receptors similar to those observed in human heart failure. The aim of this study was to evaluate the effects of chronic administration of an ACE-inhibitor, fosinopril, on noradrenergic transmission in SHR. In order to discriminate the direct effects of the drug from those mediated by blood pressure reduction, fosinopril was given at two different doses: high dose" (SHR_{HD}), 25 mg/Kg/day, and low dose" (non hypotensive), SHR_{LD}), 1 mg/Kg/day. Untreated SHR (SHR_C) and Wistar-Kyoto (WKY_C) served as controls. The following parameters have been evaluated: a) cardiac β-adrenergic receptors expression, both as gene expression (steady-state levels of mRNA, RNAase protection assay) and as membrane receptor proteins (radioligand binding); b) α-mediated vasconstriction, as evaluated by active media stress (wall tension / media thickness) developed by mesenteric small resistance arteries in response to exogenous (cumulative-dose response curve, from $3*10^{-9}$ to $1.5*10^{-5}$ M), using Mulvany's micromyographic technique. Blood pressure was significantly reduced in SHR_{HD} (164±16 mm Hg, n=20, p<0.001) but not in SHR_{LD} (190±20 mm Hg, n=20, NS) in respect to SHR_C (202± 21 mm Hg, n=16). Messenger RNA levels for the β_1 adrenergic receptor were significantly lower in SHR_C in respect to WKY_C (476±183 vs. 689±295 arbitrary densitometric units, corrected for β-actin, p<0.05). Treatment with fosinopril was associated to an increase in the levels of β_1- mRNA toward values superimposable to those observed in WKY_C, both in SHR_{HD} (654±222) and in SHR_{LD} (662±127) (p<0.05 vs SHR_C). The β_1-adrenergic receptor density was lower in SHR_C in respect to WKY_C (radioligand binding), with a small, not statistically significant increase in the treated groups. The expression of β_2-adrenergic receptor (mRNA and protein) was not significantly different between SHR_C and WKY_C, and no change was observed in any of the the treated groups.The contractile response to norepinephrine in small mesenteric arteries was similar (active media stress, ANOVA dose-response curve) in SHR_C and in WKY_C, and was significantly increased in the treated rats, both in SHR_{HD} and in SHR_{LD} (p<0.05). These results support the hypothesis that chronic treatment with ACE-inhibitors can exert an inhibitory modulation of cardiac and vascular noradrenergic neurotransmission in hypertension, as suggested by the increased gene expression of β_1-adrenergic receptor and by the increased response to exogenous norepinephrine (α-mediated vasconstriction) in treated SHR. The observation that these changes are present also in SHR_{LD} suggests that they are not related to the blood pressure reduction.