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Hypnotic Susceptibility and Hypnosis Modulate the Endothelial Response to Acute Stress

E.L. Santarcangelo*, Z. Jambrick** L. Sebastiani*, B. Ghelarducci*, E. Picano**

* Dept. of Physiology and Biochemistry. University of Pisa, Pisa, Italy
** National Council of Research, Institute of Clinical Physiology. Pisa, Italy

Neutral hypnosis is commonly used as a relaxation technique. Even if many of its physiological correlates as well as the main neuropsychological characteristics of hypnotizable individuals have been described, it is still unclear whether the hypnotic state (state theory) or the cognitive capabilities of awake hypnotizable individuals *per se* (trait theory) are responsible of the relaxation effects induced by hypnosis [1]. Indeed, specific suggestions have been proven to induce cognitive emotional experiences, associated to specific physiological patterns, which can be different in relation to hypnotizability and/or hypnosis. In recent studies [2] it has been demonstrated that awake hypnotizable subjects, at difference with non hypnotizable ones, were able to buffer the autonomic effects of a moderately unpleasant cognitive stimulation. When hypnotized, the same individuals could not suppress the autonomic output anymore, but showed the EEG correlates of a balance between relaxation and suggestion-dependent stress [3]. Thus, we hypothesized that highly hypnotizable subjects may be protected from the effects of stressful situations. To test this hypothesis, we evaluted whether hypnotizability and/or hypnosis modulates the effects of acute mental stress on peripheral vascular endothelial function.

In healthy subjects, the endothelial function that can be evaluated non invasively as post-ischaemic flow-mediated vasodilation (% FMD) by brachial artery ultrasound, is transiently reduced by acute mental stress [4]. This FMD dysfunction, which is known to outlast the stress with a peak after 10-90 minutes, is likely due to the accumulation of endogenous catecholamine and endothelin-1 and has been considered a key pathophysiological variable in triggering cardiovascular events. 18 healthy volunteers (age 19-35) with no cardiovascular risk factors or history of regular practice of relaxation techniques, were selected according to their hypnotic susceptibility through the Standford Hypnotic Susceptibility Scale, form C [5] and divided into two groups: high (Highs, score 9-12/12) and low hypnotizable individuals (Lows, score 0). The experimental session consisted of 4 periods, lasting 10 min each 1) a baseline, open eyes; 2) a simple relaxation period, eyes closed; 3) a period with a mental stress (2 minutes, at the beginning), eyes closed; 4) a later phase after mental stress, open eyes. To evaluate the possible modulation of mental stress effects by the hypnotic state, in a different day, Highs underwent another session when a a standard hypnotic induction was administered; thus, the second period was not a simple relaxation but a neutral hypnosis. Mental stress consisted in serial subtraction and multiplication. In order to induce a higher level of stress subjects were deceptively told that the result of the test would have been used to evaluate their logical-attentive capabilities. In each period, heart rate and post-ischaemic FMD of the brachial artery were measured. At the end of the session, subjects underwent a structured interview on pain and distress due to the forearm cuff occlusion, stress during mental calculation and motivation toward a good performance.

At the end of the awake session, both groups reported similar levels of stress, pain and motivation, but Highs reported a greater distress related to the arm occlusion. In addition, ANOVA revealed, in both groups, similar HR decrements during the simple relaxation period and increments during mental stress. This indicated that relaxation and stress effects on HR are not modulated by the hypnotic trait. Highs did not show any significant modification in FMD neither during mental stress nor in the other periods. On the contrary, in Lows FMD decreased throughout the whole session with significant reductions during mental stress and in the following post stress period. These results indicate that FMD was affected by mental stress only in Lows.

In the hypnotic session, Highs reported levels of distress due to the inflation lower than in the awake session. During neutral hypnosis a significant decrease of HR associated to a decrement in FMD, with respect to the precedent baseline period, was found. This effect can be due to the physiological state of higher arousal induced in hypnotizable subjects soon after the hypnotic induction. During mental calculation, HR increased, but its values did not exceed the initial baseline ones. In addition, similarly to the results obtained during wakefulness, no changes in FMD were induced by mental stress. A pre-eminent effect of hypnotic relaxation on calculation-induced stress could explain the lack of endothelial dysfunction in hypnotized susceptible subjects during mental stress.

The present findings (Fig.1) indicate that endothelial function is more influenced than heart rate by the cognitive characteristics of subjects and, also, that it can be considered a good indicator of the different hypnotizability-related vascular reactivity to stress. In conclusion, hypnotizability could prevent the toxic effect of acute

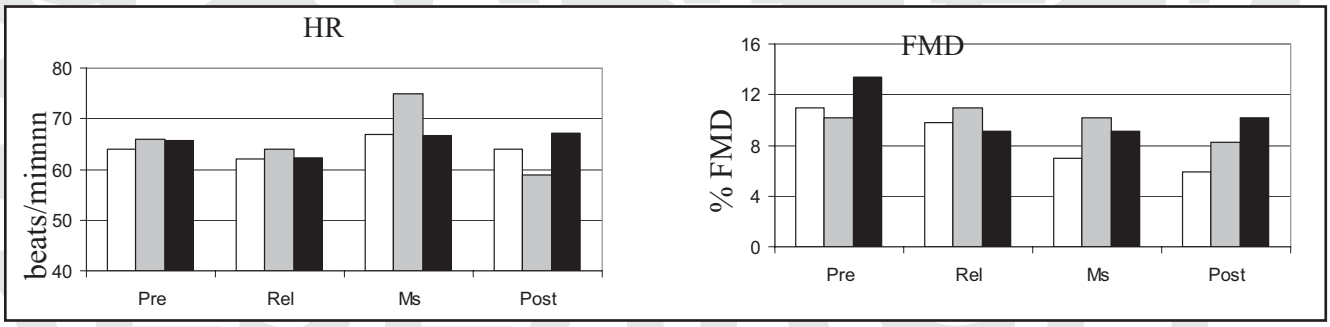

Fig. 1.

mental stress, thus contrasting its long-term effects on cardiovascular system.

References

[1] Crawford H.J., 1994. Brain dynamics and hypnosis: attentional and disattentional processes. Int. J. Clin. Exp. Hypn., 42: 204-32.
[2] Sebastiani L., Simoni A., Gemignani A., Ghelarducci B., Santarcangelo E.L., 2003. Autonomic and EEG correlates of emotional imagery in subjects with different hypnotic susceptibility. Brain Res. Bull., 60: 151-160.
[3] Sebastiani L., Simoni A., Gemignani A., Ghelarducci B., Santarcangelo E.L., 2003. Human hypnosis: autonomic and electroencephalographic correlates of a guided multimodal cognitive-emotional imagery. Neurosci. Lett., 338: 41-44.
[4] Ghiadoni L, Donald AE, Cropley M., Mullen M.J., Oakley G., Taylor M., O'Connor G., Betteridge J., Klein N., Steptoe A., Deanfield J.E., 2000. Mental stress induces transient endothelial dysfunction in humans. Circulation., 102: 2473-2478.
[5] Weitzenhoffer A.M., Hilgard E. R., 1962. Stanford Hypnotic Susceptibility Scale, form C. Consulting Psychologist Press, Palo Alto, CA.