

The Effect of Hypnosis on Systolic Blood Pressure

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Hypnosis is frequently used in numerous fields of complementary medical treatments. Blood pressure is an important vital sign. The changes manifest itself with hypo/hypertension. Hypertension is a common and important health problem in society. Our aim is to evaluate the effect of hypnosis on blood pressure in healthy volunteers and to make a preliminary study for treatment of hypertensive patients. Healthy twelve volunteers, six women and six men, aged between 18 - 65 years were included after getting ethical permits and consent. The room selected for hypnosis was quiet and room temperature was standart to minimize the effects on blood presure. We used rapid hypnosis technique. Measurements were made under hypnonic trans of 10-15 minutes. Volunteers were awakened by countdown method. The non-parametric Wilcoxon Signed Tanks test was used as statistics to chary comparison test. $P < 0.05$ was accepted as significant. The statistical results of all changes made by measurement of blood presure was found to be $p > 0.05$. Although $p > 0.05$ was not significant in pre-hypnosis, we observed average 4 mmHg decrease of systolic blood pressure during hypnosis. As a result of our data, it can be a preliminary study to show that longer hypnosis can be effective in treatment of people with systolic hypertension. It will be appropriate to repeat this study with more people.

KEY WORDS: Hypnosis, blood pressure, treatment

Kan basıncı önemli bir vital bulgudur. Değişikliği hipo ve hipertansiyon şeklinde kendini gösterir. Hipertansiyon toplumda yaygın ve önemli bir sağlık sorunudur. Amacımız hipnozun kan basıncı üzerine olan etkisini sağlıklı gönüllülerde değerlendirip, hipertansif kişilerin tedavisi için bir ön çalışma yapmaktır.

Sonuç hipnoz öncesi ve sonrası kan basıncındaki değerlerin sonucunda $p > 0,05$ anlamlı olmasa da sistolik kan basınçlarının ortalamalarına bakıldığında hipnoz sonrasında 4 mmHg'lık bir azalış gözlenmiştir. Veri sayısının az olması istatistiksel olarak anlamlılığını etkilemiş olabilir.

Tartışma. Elde ettiğimiz veriler sonucunda hipnozun, özellikle sistolik hipertansiyonu olan kişilerin tedavisinde etkili olabileceğini göstermesi ve daha fazla denek ile bu çalışmanın tekrarlanması açısından bir ön çalışma teşkil edebilir.

Cyclic AMP Enhances Beta Cell Network Activity in mouse pancreas

Xxxxx R₁, Yxxx G₂, **Zzzz P₁**, Wwww E₃.

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Insulin secretion from beta cells is triggered upon increase in $[Ca_2]_c$ and can be further amplified by cAMP, which has been previously described to act through PKA- or Epac2-dependent pathways. Since the precise mechanism of action is not fully understood we assessed the $[Ca_2]_c$ dynamics in beta cell populations with electro- and opto-physiological approaches combined

with the acute tissue slice technique, supported by network-based analyses. In the absence of

forskolin, substimulatory glucose concentration failed to increase $[Ca_2]_c$, while stimulatory glucose

concentration evoked a transient calcium increase followed by synchronized high frequency Ca_2 oscillations. Addition of forskolin to the substimulatory glucose concentration triggered a delayed high frequency Ca_2 oscillations. In high glucose concentration the addition of forskolin

further increased the frequency of Ca_2 oscillations. Despite a modest decrease in durations of individual oscillations, the relative active time increased by more than 50 %. Furthermore, the beta

cell functional networks become denser in the forskolin regime, suggesting a higher degree of

synchronicity. To determine which of the two aforementioned pathways was responsible for augmented Ca_2 oscillations, the same sets of experiments were performed on pancreatic slices

from mice lacking the Epac2 protein. In this case, a qualitatively very similar behaviour was observed compared with WT littermates. These results corroborate previously published data describing that phosphorylation of several targets by PKA is responsible for the cAMP-augmented

Ca₂ oscillations in pancreatic beta cells.

KEY WORDS: Insulin, cyclic AMP, pancreatic beta cells