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Neural regulation of cardiovascular function during inhibition of nitric oxide synthesis in rats

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Deficient synthesis of Nitric Oxide (NO) is an important component not only of hypertension but also of cardiac autonomic dysfunction. It is not known whether the hemodynamic and cardiac autonomic derangements associated with NO deficiency are due to interference of direct vasodilatory action of NO or involve some neuro-humoral system. In order to clarify and fill in the existing gaps, we assessed cardiac autonomic functions during acute and chronic inhibition of NO synthesis by NG-nitro-L-arginine methyl ester (L-NAME) in adult Wistar rats. Baroreflex Sensitivity (BRS) and Heart Rate Variability (HRV) were measured for assessment of cardiac autonomic functions. We further evaluated the contribution of oxidative stress, sympathetic nervous system as well as renin-angiotensin system, to the changes in neural regulation of cardiovascular system during NO inhibition. Serum Malondialdehyde (MDA) was measured as a marker of oxidative stress. "Subtractive approach" of chemical sympathectomy by 6-hydroxydopamine was used to study the role of sympathetic nervous system and extent of involvement of renin-angiotensin system was studied using angiotensin II type I receptor blocker, losartan. Our results reveal that sympathectomy completely reversed not only pressure rise but also cardiac autonomic dysfunction in chronic L-NAME treated rats whereas a partial reversal that too of only cardiac autonomic responses was observed in acute L-NAME treated rats. These results thus suggest that, it is the inhibition of direct vasorelaxant action of NO which mediates the effects seen after acute L-NAME administration on the other hand, effects of long-term inhibition of NO synthesis are primarily mediated through sympathoexcitation. Thus, the role of sympathetic nervous system in L-NAME model is time dependent and its contribution becomes important with longer NO inhibition. In addition, administration of losartan had no effect on pressor response of L-NAME irrespective of its duration, but it was effective in restoration of autonomic derangement especially after chronic L-NAME treatment.

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