Dissociation of Blood Pressure Level and Reactivity

To the Editor:

The recent article by Rostrup et al. in which they reported that young men with high screening blood pressure exhibit an exaggerated blood pressure response to a mental arithmetic but not to a cold pressor test is of great interest, but its interpretation is far from straightforward.

It is of particular relevance to the reactivity hypothesis, an idea first proposed more than 50 years ago which states that hyperreactive individuals are at increased risk for the development of hypertension, coronary heart disease, or both. However, the relation between hyperreactivity and hypertension remains unclear. Hyperreactivity may be considered a phasic (or transient) deviation from the norm, whereas hypertension is a tonic (or sustained) deviation. The subjects with the initial high screening pressures in Rostrup’s study were clearly hyperreactors, but not so clearly hypertensive since their resting or tonic level of blood pressure was no higher than the pressure of the group with normal screening pressure.

It has frequently been proposed that hyperreactivity precedes hypertension on the grounds that some normotensive subjects with a positive family history of hypertension are hyperreactive when compared with those subjects with a negative family history. In this context the absence of any description of a family history in Rostrup’s report is a surprising omission.

Rostrup’s results also make an interesting contrast with two other recent reports of blood pressure level and reactivity in young adults. In the first by Ravogi et al., subjects with a positive family history of hypertension had a higher tonic level of blood pressure (measured over 24 hours by ambulatory monitoring) than those with a negative family history, but they did not have higher reactivity. Rostrup’s suggestion that the tonic blood pressures of these subjects were high because of apprehension and awareness of their condition is unconvincing because they were still considered “normotensive.” Also, their pressures were higher during sleep than the pressures of the subjects with a negative family history.

Similarly, the Tecumseh study characterized a group of young subjects with modestly elevated tonic levels of blood pressure (in this case identified by home monitoring) who also had no increase of reactivity. The subjects’ parents also tended to have higher blood pressures. Another interesting contrast with Rostrup’s findings was that the hypertensive subjects in the Tecumseh study had higher resting plasma catecholamine levels than the normotensive subjects, which would be consistent with a tonic elevation of sympathetic tone. Rostrup’s subjects with hyperreactivity showed no increase of their catecholamine levels either at rest or during the stressors.

Taken together, these three important studies suggest that there is a dissociation between the tonic and phasic regulation of blood pressure and that we should be cautious in assuming that deviations in one necessarily result in deviations in the other.

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References

The following is in response:

Professor Pickering’s interpretations of our results are both relevant and interesting. Our study may illustrate a dissociation between phasic and tonic regulation of blood pressure. It is true that our subjects with cardiovascular hyperreactivity to mental stress had normal baseline blood pressure as evaluated by intraarterial recording after 30 minutes of supine rest. However, the phasic regulation of blood pressure during the cold pressor test was completely normal in the hyperreactive subjects. Thus, there seemed to be no general change in the phasic regulation of blood pressure in our subjects with hyperreactivity.

Whether certain confounding effects of awareness of hypertension directly, or more likely indirectly via the parents’ awareness and apprehension, were present in the study of Ravogi et al. is, as we see it, an open question. Details on how the subjects and their parents were informed were not presented in their report. It would also be of interest to know whether the subjects lived with their parents during the 24-hour period of the ambulatory recording and if the three groups differed in that respect. Furthermore, there are no data on how awareness of hypertension and related phenomena may affect sleep quality and thus blood pressure at night during a 24-hour ambulatory blood pressure recording. Parental hypertension was not focused in our study. This would have implemented examinations of the parents.

It is of course uncertain whether hypertension will develop in our subjects with hyperreactivity. However, the subjects were also characterized by high screening blood pressure, which may be a predictor of future hypertension. Our hypothesis is that hyperreactivity to mental stress in these subjects may be due to an increased sensitivity to catecholamines. Thus, both resting blood pressure and blood pressure reactivity were strongly correlated with plasma epinephrine in hyperreactive subjects. If these subjects become exposed to prolonged mental stress, ie, tonic activation of the sympathetic nervous system, elevated tonic levels of blood pressure would not be unexpected. However, the hyperreactivity may be a marker of an underlying trait and not itself a proven cause of later hypertension. Forthcoming data will hopefully shed further light on this possibility.

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Postexercise Hypotension

To the editor:

We read with great interest the excellent review by Kenney and Seals regarding the hypotension that follows exercise. The authors concluded that despite the publication of recent studies on this issue, several features of this response still remain obscure. We wish to add a few notes that may help in understanding this controversial area.

1. When discussing the “nature of the exercise stimulus,” the importance of the intensity of exercise on the hemodynamics and blood pressure response has been noted in our laboratory in normotensive volunteers. A less than maximal exercise induced increases in total peripheral resistance with reduced blood flow in the nonexercising limb (forearm), whereas maximal leg exercise induced opposite effects on total peripheral resistance and in the forearm. Therefore, these differential effects of different intensities of exercise may explain some of the discrepant results observed concerning the hemodynamic changes that occur after exercise in published studies.

2. The “time course of the hemodynamics” after maximal upright bicycle exercise was also recently evaluated in our laboratory (unpublished observation): total peripheral resistance was reduced for 2 hours in addition to effects on the resistances of exercising and nonexercising vascular beds. These changes were associated with falls in systolic and diastolic pressure, and all hemodynamic variables had returned to control levels by 3 hours after exercise. This suggests a 3-hour limit for the hypotensive effect of exercise. Different results are obtained when the subjects are not studied under controlled conditions, as previously reported.

3. An inhibition of sympathetic nerve activity seems to us to be an unlikely cause of the hypotension seen after exercise (at least in normotensive subjects). Together with a fall in total peripheral resistance, we showed a persistently elevated sympathetic discharge to the heart and the circulation, as detected by spectral analysis of heart rate and blood pressure variability. Changes in endogenous opioids have been proposed as a possible mechanism of the hemodynamic changes that occur after exercise, as outlined in the present review. Their activity seems to be enhanced by an elevated sympathetic tone. A situation in which there are falls in total peripheral resistance and in arterial pressure (with a result suggesting a 3-hour limit for the hypotensive effect of exercise) may be exactly the situation where opioids could play an important role in mediating the hemodynamic effects we have described after exercise.

4. In all these studies an increased cardiac output was observed after exercise, lasting for at least 1 hour, that was accounted for by increased heart rate with the stroke volume remaining unchanged after maximal exercise in normotensive subjects.

In conclusion, on the basis of the reported studies and our experiences, we believe that physical conditioning induces complex hemodynamic changes. The distribution and the nature of these changes depend on the details of the exercise. It is unclear whether these acute responses contribute to the chronic effects of training in heart disease.

References


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