Letters to the Editor

Aftereffects of Exercise

To the editor:

We read with interest the report by Cléroux and colleagues on the hemodynamic aftereffects of exercise in the February issue. They are wrong to state that no study has looked simultaneously at regional and systemic hemodynamics after exercise and also to imply that no study had controlled for the anticipatory effects on exercise. In 1989 we reported on the simultaneous measurement of systemic and forearm hemodynamics after maximal upright bicycle exercise in a random-order, controlled crossover comparison against a control period on a separate day. Our study was in normotensive subjects and, contrary to the findings of Cléroux et al., we demonstrated a clear and highly significant reduction in blood pressure and total peripheral and forearm vascular resistances. Thus, the absence of this change in Cléroux’s study is likely to reflect the weaker statistical power of their study (only nine normotensive subjects). This highlights the dangers of statistical errors in assuming that the absence of statistically significant results indicates that no change had occurred. Another possible reason for apparently different results may be the intensity of the prior exercise. We have observed (unpublished observations from our laboratory) that the peripheral vasodilatation after exercise seen in vascular beds from nonexercised limbs is dependent on the intensity of exercise. At less than 50% maximal exercise a vasodilatation is seen, whereas after greater than 50% maximal exercise a vasodilatation is seen. Thus, the study of Cléroux et al. may have serendipitously used a level of exercise that caused no net effect on forearm vascular resistance.

A second point of some interest is the suggestion that these results show a reduction in sympathetic drive after exercise that may contribute to the hypotension seen after exercise. Floras et al., using microelectrode recordings from the leg that had exercised, suggested that sympathetic withdrawal may contribute to after-exercise hypotension. Local heat and metabolite accumulation may, however, have led to local sympathetic withdrawal. In a study similar in design to that of Cléroux et al., we found that sympathetic tone to the heart (using the low frequency component of autoregulatory power spectral analysis of heart rate and blood pressure variabilities) was increased for at least 1 hour after exercise, which supports our earlier findings of persisting sympathetic tone to the heart (using the low frequency component of autoregulatory power spectral analysis of heart rate and blood pressure variabilities) was increased for at least 1 hour after exercise, which supports our earlier findings of persisting sympathetic tone to the heart (using the low frequency component of autoregulatory power spectral analysis of heart rate and blood pressure variabilities). This highlights the dangers of statistical errors in assuming that the absence of statistically significant results indicates that no change had occurred. Another possible reason for apparently different results may be the intensity of the prior exercise. We have observed (unpublished observations from our laboratory) that the peripheral vasodilatation after exercise seen in vascular beds from nonexercised limbs is dependent on the intensity of exercise. At less than 50% maximal exercise a vasodilatation is seen, whereas after greater than 50% maximal exercise a vasodilatation is seen. Thus, the study of Cléroux et al. may have serendipitously used a level of exercise that caused no net effect on forearm vascular resistance.

Thence, we commend Cléroux and colleagues for reporting on this interesting area of integrated physiology but would warn against drawing premature conclusions.

References


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The following is in response:

To the editor:

We are grateful to Drs. Coats and Piepoli for pointing out an earlier study that had unwittingly escaped our attention and for mentioning unpublished data supporting our observation 2 that forearm vascular resistance was unchanged in normotensive subjects after they had performed cycling exercise at 50% of maximal aerobic capacity. In our view, it remains all the more interesting that hypertensive subjects exercising at the same relative (percentage of maximal aerobic capacity) and absolute (Watts) intensity exhibited strikingly different postexercise hemodynamic responses.

Their comment on sympathetic nervous activity is well taken. In our article, we recognized that the evidence linking the finding of a reduced muscle sympathetic nervous activity after exercise in hypertensive subjects with our observation of reduced forearm vascular resistance and plasma norepinephrine is valid to the extent that the relations between sympathetic nervous activity and plasma norepinephrine also hold after exercise when the leg has been active and the forearm has not. This important limitation could prove them right in that it may be "misleading to extrapolate from what is happening to localized sympathetic nerve traffic in the leg to the state of sympathetic activity to other organs..." for the time being, we note that Drs. Coats and Piepoli revise their earlier conclusion that "postexercise hypotension seems associated with persistent sympathetic discharge and reduced vagal activity on the cardiovascular system" and now state that "... for cardiac sympathetic activity at least, sympathetic tone is persistently elevated rather than reduced after exercise."

In closing, we are well aware that plasma norepinephrine may be subject to criticism as an index of sympathetic nervous activity. However, the suggestion that an increased clearance of norepinephrine due to an increased renal blood flow after exercise may contribute importantly to changes in plasma levels does not find

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strong support in the literature. Indeed, plasma norepinephrine clearance does not decrease during exercise when renal blood flow is reduced or is its half-life different during recovery compared with rest. This suggests that changes in plasma levels are more likely to be due to changes in norepinephrine release. Drs. Coats and Piepoli discuss their data on sympathetic tone assessed with power spectral analysis. It may be worth mentioning that with this approach, Arai et al reported that sympathetic activity to the heart was reduced below baseline levels after a maximal exercise protocol similar to the one used by Piepoli et al, i.e., in direct contradiction with the conclusion of the latter authors, although a persistent tachycardia was observed in both studies.

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