LETTERS TO THE EDITOR

References

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AUTHOR’S RESPONSE:

I am pleased that Drs. Boer and Geyskes found my review article an incentive for additional study of the levels of renin substrate in essential hypertension. Their data on PRS values in patients treated with enalapril show a clear correlation between the lowering of PRS and lowering of blood pressure, thus supporting the hypothesis of a possible causal relationship. Their data on such values in patients treated with propranolol show exactly the opposite, i.e., that increasing PRS values correlate with lowering of blood pressure. There are two obvious conclusions to be drawn from these data: 1) the hypothesis of a positive correlation between blood pressure and PRS is not valid in all circumstances; and 2) the mechanism of blood pressure lowering by propranolol is quite different from that of enalapril. The finding of an actual increase in PRS with increasing doses of propranolol is quite surprising. It leads me to wonder if large increases of PRS may be found to coincide with undesirable side effects related to increased peripheral vasoconstriction, such as Raynaud’s phenomenon, intermittent claudication, and peripheral gangrene, which occur in some patients treated with propranolol.

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Correction to Graph in September-October Supplement

To THE EDITOR:

I enclose the corrected version of figure 7 of my paper in the Supplement of Hypertension concerning the Satellite to the European Milan Meeting. The error was in the second line of the illustration, which should be “... vs mean of 24 hours” and not “vs mean of 4 hours.” (Hypertension 5 (suppl III): III-5-III-13, 1983; Mancia G: Methods for Assessing Blood Pressure Values in Humans.)

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FIGURE 7 Comparisons of the average 24-hour mean arterial pressure value with the average mean arterial pressure (MAP) values obtained during different hours (upper panel) or different 4-hour periods (lower panel) of the same 24 hours. Data are shown individually for 28 subjects in which 24-hour intraarterial blood pressure recordings were made. Each recording was analyzed by a computer that sampled the blood pressure trace every 60 msec to obtain the 24-hour average MAP value. The same analysis was performed to obtain hourly or 4-hour averages starting from midnight and advancing at half-hour steps (horizontal lines). The differences between the hourly or 4-hour averages and the 24-hour averages were plotted by considering the latter as the 0 reference value. (From Di Rienzo et al., unpublished data.)
Correction to graph in September-October supplement.
G Mancia

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