

Risks of Overtreatment of Hypertension

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In their report "Visit Frequency for Essential Hypertension" in this issue of the *Journal*, Lichtenstein et al¹ found that the interval between visits to the hospital clinic or to the general practitioner was shorter when diastolic blood pressure was above 104 mmHg. This finding is reassuring and suggests that physicians were following patients more closely while blood pressure was in these higher ranges.

It is a matter of some concern, however, that the interval between visits lengthened progressively to reach a maximum at a diastolic pressure of 70 to 79 mmHg. The study does not include data on drug administration. It is likely that many of the patients at the lowest blood pressure levels had had their medications discontinued. Nevertheless, for those patients at lower blood pressure levels who are receiving drug therapy, it is important to monitor blood pressure and to adjust drug dosage. Considerable evidence is now available that suggests that the risk of myocardial infarction is increased when blood pressure is reduced too much by antihypertensive drug therapy.

Stewart² found that the risk of myocardial infarction was greatest in those hypertensive patients with the lowest treatment blood pressure. He followed 169 patients for 6 years. In patients who had a myocardial infarction, he noted the diastolic pressure on the last visit before the infarction. He assigned a risk of 1.0 to patients whose treatment diastolic pressure was in the range 95 to 109 mmHg. Patients in the range of 76 to 94 mmHg had a relative risk of myocardial infarction of 6.4, while those in range 110 to 140 mmHg had a relative risk of 2.7. In other words, those patients in the 76- to 94-mmHg range were at six times the risk of myocardial infarction as those with what would be classified as mild hypertension and more than twice the risk as those with what might be regarded as hypertension out of control. He suggested caution as to both the percentage reduction in pretreatment blood pressure and the actual treatment level of pressure.

The Multiple Risk Factor Intervention Trial (MRFIT) report³ suggested that intensive treatment of hypertension increased the coronary heart disease death rate. This increased death rate was noted in those subjects who had abnormal electrocardiograms (ECGs) on entry. The report suggested that these subjects might be more vulnerable because of diuretic therapy, a part of the treatment plan. There were, however, also data in the study which suggested that subjects with the lowest entry blood pressures and with normal ECGs had an increase in coronary heart disease death rate. These subjects would be likely to have lower treatment blood pressure.

Cruickshank et al⁴ reported that mortality from myocardial infarction followed a J-shaped curve in hypertensive patients receiving drug therapy. Below a diastolic pressure of 85 mmHg mortality increased, but only in patients who on entry had evidence of ischemic heart disease.

Samuelsson et al,⁵ however, in a 12-year study of hypertensive men, found that cardiovascular morbidity was lowest at a treatment diastolic pressure of 86 to 89 mmHg and increased when the diastolic pressure was below 86 mmHg. This finding was true of all subjects and was not limited to those with evidence of ischemic heart disease on entry.

It is not surprising that the heart might be vulnerable to low diastolic pressure in drug-treated hypertensive patients. Perfusion of the myocardium, especially the subendocardial myocardium, occurs only during diastole; therefore, drastic lowering of diastolic pressure might seriously impair myocardial oxygen supply.

Floras⁶ has commented on 24-hour blood pressure studies that he and his colleagues had reported earlier.⁷ Blood pressure was recorded intra-arterially in 34 hypertensive patients before and after treatment with different β -blockers. Average clinic pretreatment blood pressure was 176/108 mmHg. After 5 months of β -blocker treatment, clinic pressure averaged 151/95 mmHg. The average treatment ambulatory blood pressure was 148/78 mmHg; average treatment blood pressure when asleep was 119/60 mmHg. In 11 of the patients average hourly diastolic pressures of 50 mmHg or less were recorded during sleep. Mean diastolic pressures of 30 mmHg or less were recorded during 7 hours of sleep in some patients.

The problem of perfusion is magnified if coronary artery stenosis is present. Precise estimation of the degree of pres-

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sure drop distal to the stenosis is not possible in the clinical situation. This drop is significant, however, because pressure energy is lost as a result of a transfer of energy when normal arterial flow changes to high-velocity flow through the stenotic area. Additional energy is lost when blood expands into turbulent eddies distal to the stenosis. The degree of drop in perfusion pressure would be influenced by both the reduction in the diameter of the arterial lumen and the length of the narrowed segment of the artery. Brown et al⁸ commented on these important dynamic changes. It is clear that pressure in a stenotic coronary artery might fall to extremely low levels in a patient whose brachial artery pressure drops to 50 mmHg or even 30 mmHg during sleep.

This discussion strongly suggests that excess lowering of diastolic pressure should be avoided in caring for patients with hypertension. Two approaches to this problem are suggested. First, drug therapy for hypertension should not be initiated unless diastolic pressure is at a level at which morbidity and mortality are clearly reduced by drug treatment. This level is a matter of controversy at present, and space does not permit full discussion. A diastolic pressure of 100 mmHg or greater can be defended as the point at which drug treatment may be justified. To start drug therapy at a lower level increases the likelihood that diastolic pressure will drop too low when the blood pressure response has occurred. A study by Thomson et al⁹ reported that 90 to 104 mmHg was the level at which 92% of the physicians surveyed initiated drug therapy. Since a majority of patients with newly diagnosed hypertension fall into this blood pressure stratum, this matter is of great clinical significance.

Second, blood pressure should be monitored at suitable intervals and drug dosage should be reduced if diastolic pressure falls below 90 mmHg in the office or clinic or

below 85 mmHg at home. Further reductions in dosage should be made every 10 to 14 days as long as diastolic pressure remains below these levels.

In hypertensive patients known to have ischemic heart disease, blood pressure should be monitored often enough to give assurance that levels close to the 85- to 95-mmHg range are being maintained and that lower levels are avoided. In older patients who might have silent ischemic heart disease, the diastolic pressure at which drug therapy is initiated should probably be raised to 105 mmHg. The interval between visits should assure good control of blood pressure through appropriate adjustment of drug dosage.

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