When and How Far Should We Lower Blood Pressure?

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SUMMARY  The accuracy of the diagnosis of mild or borderline hypertension is dubious. Perhaps up to one-third of such patients are wrongly categorized when more accurate measures are taken, largely because of the "defense" reaction. The recent trials of mild hypertension are discussed. Patients with a diastolic blood pressure in the range of 95 to 100 mm Hg should only begin treatment after careful assessment and consideration of other risk factors.

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KEY WORDS  • borderline hypertension • pseudohypertension • defense reaction • hypertension screening

The question of how much should we lower blood pressure resolves itself into at least two: 1) At what level of pressure should we recommend treatment? and 2) If we do recommend treatment, should we set a target pressure? If so, how hard should we strive to achieve that target?

Epidemiology gives some help with the first question. The Society of Actuaries (1979) apparently showed convincingly that there is no clear threshold pressure, but rather a smooth gradation in risk with rising pressure — even in the so-called borderline and normal ranges. (I say "apparently" because I always have some misgivings about the accuracy of the pressure measurements in insurance statistics; on the one hand, the pressures may be unduly raised because of anxiety over premium loading; on the other hand, the physicians’ bias is often to favor the patients against the insurance company, and so consciously or unconsciously record figures that are too low.)

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Accuracy of Diagnosis Essential

The most worrisome feature of the recent enthusiasm for drug treatment of mild hypertension is the great difficulty in making a secure diagnosis. We recruited for a trial 59 untreated subjects with mild to moderate hypertension (pressure greater than 140/90 averaged over three clinic visits, after 5 minutes of rest). The mean (diastolic + ½ pulse pressure) pressure was 119 ± 17.8 mm Hg (sd). The study began with a 24-hour ambulatory recording of intraarterial pressure. We found considerable differences between the "resting" cuff pressures taken in the clinic and the ambulatory direct readings from the artery. In 32 of 59 patients the intraarterial mean pressures were lower by more than 10 mm Hg (simultaneous comparison of cuff and intraarterial pressures gives very close agree-
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ment). Twenty of the subjects classified as hypertensive had intraarterial pressures less than 140/90 mm Hg during waking hours. Using cuff pressures alone would therefore have led to possibly inappropriate treatment (? lifelong) in 1/3 of these subjects. Of course, it may be argued that all our knowledge of the benefit of treatment, and of prognosis, are based on cuff measurement. My reply would be that the correlation between blood pressure and future risk would be even stronger if we could remove the "noise" of these false cuff readings.

Indeed, Sokolow and his colleagues used an early model of the Remmler portable cuff recorder to show that prognosis did indeed correlate better with repeated ambulatory cuff recordings than casual clinic cuff estimates. Pickering et al. have used a similar method to show that, for normotensive subjects and for established hypertensives, clinic and ambulatory pressures agree well but borderline hypertensive patients overreacted to the physician's office with falsely high values. They too have found better correlation between echocardiographic measurements of left ventricular wall thickness and blood pressures taken at work.

This important effect of the defense or alerting reaction may have been responsible for the Australian finding that about half their placebo group had fallen to normal pressures by 5 years: the greatest fall occurred over the first 4 months after the screening visits but continued to fall slowly at least up to the fourth year.

It is clear then that the very greatest caution should be exercised in labeling a person as hypertensive when the readings fall in the borderline range. I much prefer to supplement repeated clinic cuff readings with one or more direct ambulatory records where objective evidence is required (e.g., insurance or aviation license examination) or with home cuff measurements where less objective measures will suffice.

Other Risk Factors

It is clear from the Framingham data that high blood pressure has a much greater impact on risk when serum cholesterol is high and the subject smokes cigarettes. It is negligent to prescribe drugs but not to curb cigarette smoking. The much maligned MRFIT trial showed that it was possible to persuade half the intervention subjects to quit smoking and this had the most powerful effect on the outcome of all three interventions (the two other interventions were blood pressure and cholesterol reduction). These well-known factors are often neglected in the debate over what levels of pressure at which to start treatment. A 1980 U.S. Public Health Survey showed that only one-third of the treated patients had been advised by their doctors to stop smoking.

Thus, these other factors should be considered when evaluating whether to start treatment. Although the Framingham data have shown that systolic blood pressure (SBP) is just as good if not better a predictor of future morbidity, trials still continue to use phase V diastolic pressure as the yardstick. A recent paper from London's St. Thomas Hospital suggests that three visits (timing is not crucial) will adequately characterize the pressure.

The Australian trial suggests no benefit for treatment of patients where the average of three diastolic pressures is less than 95 mm Hg. Freis has some reservations about that trial's claim that those with a DBP 95–99 mm Hg benefited from the treatment. I agree with the statement in the second Australian report suggesting that, for those patients whose third reading is below 95 mm Hg, there is much to be said for further observation, plus nondrug treatment by weight reduction, exercise, and perhaps sodium restriction. My present practice is, therefore, to assess borderline pressures carefully and treat them if the DBP is above 95 mm Hg, particularly if other risk factors are present.

If side-effects are produced, then I am prepared to withdraw treatment and observe further if the average DBP is below 100 mm Hg.

Can We Achieve the Target?

Our hypertension clinic is part of the Hammersmith Hospital's computer-assisted follow-up system. This system has the tiresome but useful facility of pointing out inadequate treatment pressures. It is clear that a substantial proportion of patients do not achieve the goal of, say, 140/90 mm Hg or less, either through genuine resistance, or more common, through poor compliance. Does this matter? Encouragingly, Taguchi and Freis suggest that poor control is better than none at all. Dixon and Johnson have suggested that some patients (particularly those with mild hypertension) may never reach their target pressures. They suggest the use of a response line in which the fall in DBP is plotted against the pretreatment pressure. This results in a linear relation when trial data are so examined. This has some advantage for comparison of different treatments.

The degree to which I strive to achieve "normality" is very much influenced by other risk factors such as age, sex, obesity, cholesterol, glucose tolerance, and so on.

References

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