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. (Hypertension 5 (supp III): III-14-III-16, 1983)

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.)

Nevertheless, the Framingham data support the same concept of the lower the better — provided there is enough pressure to stop postural syncope.

Freis has recently pointed out that such epidemiological data on untreated populations cannot be used as sound predictors for the effects of treatment. The Australian trial showed that lowering blood pressure with drugs did not then lower the risk to that of an untreated subject with the same pressure. Furthermore, the same Australian trial showed that lowering diastolic pressure to 90 mm Hg was no more beneficial than lowering it to 90 mm Hg (we do not have any data on the correlation or otherwise between risk reduction and blood pressure reduction in the HDFP trial). This lack of expected benefit might, of course, be due to lack of regression of prior damage to heart and/or blood vessels. It might equally be due to adverse effects of treatment. Thiazide diuretics might be harmful, perhaps from arrhythmia due to lowered potassium. The MRFIT study showed increased mortality in the retrospectively identified subjects with ECG abnormality at entry. Some of the questions on the risks of thiazides (first line treatment in the U.S.) and of beta adrenoceptor blocking agents (fast becoming first line treatment in the U.K. and Scandinavia) should be answered by the MRC trial of mild hypertension (still to report).

Stewart has produced uncontrolled evidence to suggest that lowering blood pressure to less than 90 mm Hg might compromise coronary blood flow when coronary disease is already present.

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-
They too have found better correlation between echo-
during waking hours. Using cuff pressures alone
ambulatory cuff recordings than casual clinic cuff esti-
that prognosis did indeed correlate better with repeated
acted to the physicians office with falsely high values.
agree well but borderline hypertensive patients overre-
show that, for normotensive subjects and for estab-
benefit of treatment, and of prognosis, are based on
cuff measurement. My reply would be that the correla-
tion between blood pressure and future risk would be
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 esti-
mates. Pickering et al. have used a similar method to
show that, for normotensive subjects and for estab-
lished hypertensives, clinic and ambulatory pressures
agree well but borderline hypertensive patients overre-
tacted to the physicians office with falsely high values.
They too have found better correlation between echo-
cardiographic measurements of left ventricular wall
thickness and blood pressures taken at work.
This important effect of the defense or alerting reac-
tion 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 either
our 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 power-
ful effect on the outcome of all three interventions (the
two other interventions were blood pressure and cho-
sterol 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 character-
ize the pressure.
The Australian trial suggests no benefit for treat-
ment 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 rest-
raction. 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, Tagu-
chi and Freis suggest that poor control is better than
none at all. Dixon and Johnson have suggested that
some patients (particularly those with mild hyperten-
sion) 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 exam-
ined. This has some advantage for comparison of dif-
fent 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.

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