

Does This Patient Have Hypertension?

How to Measure Blood Pressure

Richard A. Reeves, MD, FRCPC

CLINICAL SCENARIO

Is This Patient's Blood Pressure Really Elevated?

A 46-year-old man who has recently moved to your neighborhood presents with a painful ankle sprain. Before he leaves, you decide to check his blood pressure (BP) and obtain an initial reading of 164/102 mm Hg. He denies having high BP previously.

WHY IS ACCURATE BP MEASUREMENT IMPORTANT?

Elevated arterial BP, or hypertension, is important because it is common, it is clinically silent, it leads to cardiovascular disease (CVD), and it decreases life expectancy. Because surveys find that approximately 20%¹⁻³ of North American adults have an elevated BP (systolic BP ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg) or are taking antihypertensive medication, physicians are advised to check all patients periodically for BP elevation.³⁻⁷ On the other hand, overestimation of BP can erroneously label people as hypertensive and potentially result in unnecessary dietary restrictions, exposure to potential side effects from drug treatment, medication expense, and adverse socioeconomic effects.^{8,9} Fortunately, measuring BP is an easy and safe diagnostic procedure that, when followed by appropriate an-

ti-hypertensive drug treatment, can lead to reduced CVD and mortality.^{10,11}

STANDARDS FOR MEASURING BP

The "gold" standard for instantaneous BP measurement is the intra-arterial or direct BP (determined by a rigid-walled catheter). The standard for clinical practice is the so-called casual cuff or indirect BP.

Guidelines for Diagnosing Hypertension

Cardiovascular disease risk rises monotonically with BP, revealing no cut point below which risk is minimal and above which CVD will definitely occur. Terms used to indicate the degree of BP elevation now emphasize the importance of what was previously termed mild hypertension and the long-recognized greater predictive value of elevated systolic BP¹² (Table 1). Risk for future CVD is predicted by even a single careful BP reading.¹³⁻¹⁵ However, BP is rather variable and often decreases with observation so that, in accord with statistical expectations, risk relates more closely to mean BP over several visits¹³ (although brief, severe BP elevation can also be catastrophic, eg, with cocaine overdose). Therefore, we could define the "treatable BP level" as that mean clinical BP above which treatment has been shown in randomized controlled trials to do more good than harm. The largest of these trials used drug treatment vs placebo after finding a consistent or average entry BP from two to three visits of greater than or equal to 160 mm Hg systolic (tested only in the elderly) with or without diastolic BP elevation,¹⁶ or greater than or equal to

90 mm Hg diastolic (tested in the young and in the elderly).¹¹ In the future, individualized assessments of absolute risk incorporating other relevant information, such as age, sex, concomitant risk factors, and coexisting target organ damage, along with the patient's tolerance for risk and history of drug side effects may replace arbitrary cut points in determining when BP elevation becomes treatable.⁷ At present, a diagnosis of hypertension reflects a consensus regarding the office BP level above which CVD risk worsens importantly, about 140/90 mm Hg.

A detailed conceptual analysis of hypertension is beyond the scope of this article, but has been addressed thoughtfully by Jennings and Netsky.¹⁷

How to Measure Clinical BP

Meticulous technique in indirect auscultatory BP measurement is mandatory for research, for diagnosis, and for optimal clinical care of hypertensive patients. Published procedural guidelines show general uniformity.^{3-5,18-20} The accuracy and reliability of BP measurement will be increased by following the widely accepted procedures recommended by the American Heart Association (Table 2, Figure 1). Blood pressure is customarily measured after obtaining the medical history as part of the "vital sign" determination at the beginning of the physical examination. At each visit, two or more readings should be obtained and averaged from the same

From the Division of Clinical Pharmacology, Department of Medicine, and the Clinical Epidemiology Unit, Sunnybrook Health Sciences Centre, University of Toronto (Ontario). Dr Reeves is now with Bristol-Myers Squibb Pharmaceutical Research Institute, Princeton, NJ.

Reprint requests to Bristol-Myers Squibb Pharmaceutical Research Institute, PO Box 4000, Princeton, NJ 08543 (Dr Reeves).

The Rational Clinical Examination section editors: David L. Simel, MD, MHS, Durham Veterans Affairs Medical Center and Duke University Medical Center, Durham, NC; Drummond Rennie, MD, Deputy Editor (West), JAMA.

Table 1.—Classification of Blood Pressure for Adults Aged 18 Years and Older*

Category	Systolic, mm Hg	Diastolic, mm Hg
Normal	<130	<85
High normal	130-139	85-89
Hypertension†		
Stage 1 (mild)	140-159	90-99
Stage 2 (moderate)	160-179	100-109
Stage 3 (severe)	180-209	110-119
Stage 4 (very severe)	≥210	≥120

*Adapted from the fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure.³

†Based on the average of two or more readings taken at each of two or more visits after an initial screening.

arm with the subject supine or seated. As a practical approach to variability, taking additional readings until a stable level is reached has been suggested when the first two differ by more than 5 mm Hg diastolic.²⁰ Blood pressure in both arms should be measured at the first visit, and the arm with the higher pressure should be used thereafter.¹⁸

Careful technique guarantees maximum accuracy. We have compiled information from a number of sources regarding factors that increase, decrease, or have no effect on BP²¹⁻⁷⁴ (Table 3). However, if all serious errors that can underestimate BP are avoided, finding the BP in any setting, position, or time to be within the normal range makes a more careful measurement at that visit unlikely to be high. Assuming BP is checked on a routine basis in all adults, the efficient practitioner can reasonably reserve the "proper" method for the 10% to 20% of patients who have known or newly detected elevated BP (as in our clinical scenario), cardiovascular target organ damage, or other risk factors or who are receiving antihypertensive therapy.

Variation in BP Measurement

Sources of clinical variability include the patient, equipment, examiner, and procedure. For BP, a major proportion of random fluctuation over time arises from the examinee. Intra-arterial monitoring clearly reveals that systolic and diastolic BP differ with every heartbeat and with the respiratory cycle.⁴⁵ Blood pressure also varies minute-to-minute, with an SD of about 4 mm Hg systolic and 2 to 3 mm Hg diastolic,^{59,75} as well as over hours^{76,77}; short-term variability in systolic BP is increased with impaired baroreflexes.^{77,78} Day-to-day variation is even greater. With two or more cuff readings at each visit, the SD between visits is approximately 5 to 12 mm Hg systolic and 6 to 8 mm Hg diastolic.^{13,59,60,79,80} This variability explains why two BP measurements in a patient often differ, but it also suggests that a repeat visit's measurements could be as

Table 2.—Technique for Measuring Blood Pressure*

The intent and purpose of the measurement should be explained to the subject in a reassuring manner and every effort made to put the subject at ease. [Include a 5-minute rest before the first measurement.]

The sequential steps for measuring the blood pressure in the upper extremity, as for routine screening and monitoring purposes, should include the following:

1. Have paper and pen at hand for immediate recording of the pressure.
2. Seat the subject in a quiet, calm environment [with feet flat on the floor, back against the chair] with his or her bared arm resting on a standard table or other support so the midpoint of the upper arm is at the level of the heart.
3. Estimate by inspection or measure with a tape the circumference of the bare upper arm at the midpoint between the acromion and olecranon process and select an appropriately sized cuff. The bladder inside the cuff should encircle 80% of the arm in adults and 100% of the arm in children less than 13 years old. If in doubt, use a larger cuff. If the available cuff is too small, this should be noted.
4. Palpate the brachial artery and place the cuff so that the midline of the bladder is over the arterial pulsation, then wrap and secure the cuff snugly around the subject's bare upper arm. Avoid rolling up the sleeve in such a manner that it forms a tight tourniquet around the upper arm. Loose application of the cuff results in overestimation of the pressure. The lower edge of the cuff should be 1 in (2 cm) above the antecubital fossa where the head of the stethoscope is to be placed.
5. Place the manometer so the center of the mercury column or aneroid dial is at eye level [except for tilted-column floor models] and easily visible to the observer and the tubing from the cuff is unobstructed.
6. Inflate the cuff rapidly to 70 mm Hg, and increase by 10 mm Hg increments while palpating the radial pulse. Note the level of pressure at which the pulse disappears and subsequently reappears during deflation. This procedure, the palpatory method, provides a necessary preliminary approximation of the systolic blood pressure to ensure an adequate level of inflation when the actual, auscultatory measurement is made. The palpatory method is particularly useful to avoid underinflation of the cuff in patients with an auscultatory gap and overinflation in those with very low blood pressure.
7. Place the earpieces of the stethoscope into the ear canals, angled forward to fit snugly. Switch the stethoscope head to the low-frequency position (bell). The setting can be confirmed by listening as the stethoscope head [ie, the bell orifice] is tapped gently.
8. Place the head of the stethoscope over the brachial artery pulsation, just above and medial to the antecubital fossa but below the edge of the cuff, and hold it firmly [but not too tightly²¹] in place, making sure that the head makes contact with the skin around its entire circumference. Wedging the head of the stethoscope under the edge of the cuff may free up one hand but results in considerable extraneous noise [and is nearly impossible with the bell in any event].
9. Inflate the bladder rapidly and steadily to a pressure 20 to 30 mm Hg above the level previously determined by palpation, then partially unscrew (open) the valve and deflate the bladder at 2 mm [Hg/sec] while listening for the appearance of the Korotkoff sounds.
10. As the pressure in the bladder falls, note the level of the pressure on the manometer at the first appearance of repetitive sounds (Phase I) and at the muffling of these sounds (Phase IV) and when they disappear (Phase V). During the period the Korotkoff sounds are audible, the rate of deflation should be no more than 2 mm per pulse beat, thereby compensating for both rapid and slow heart rates.
11. After the Korotkoff sound is heard, the cuff should be deflated slowly for at least another 10 mm Hg, to ensure that no further sounds are audible, and then rapidly and completely deflated, and the subject should be allowed to rest for at least 30 seconds.
12. The systolic (Phase I) and diastolic (Phase V) pressures should be immediately recorded, rounded off (upwards) to the nearest 2 mm Hg. In children, and when sounds are heard nearly to a level of 0 mm Hg, the Phase IV pressure should also be recorded [example: 108/64/56 mm Hg]. All values should be recorded together with the name of the subject, the date and time of the measurement, the arm on which the measurement was made, the subject's position, and the cuff size (when a nonstandard size is used).
13. The measurement should be repeated after at least 30 seconds, and the two readings averaged. In clinical situations, additional measurements can be made in the same or opposite arm, in the same or an alternative position.

*Reproduced with permission.¹⁸ Copyright 1993 American Heart Association. Bracketed comments added by the author.

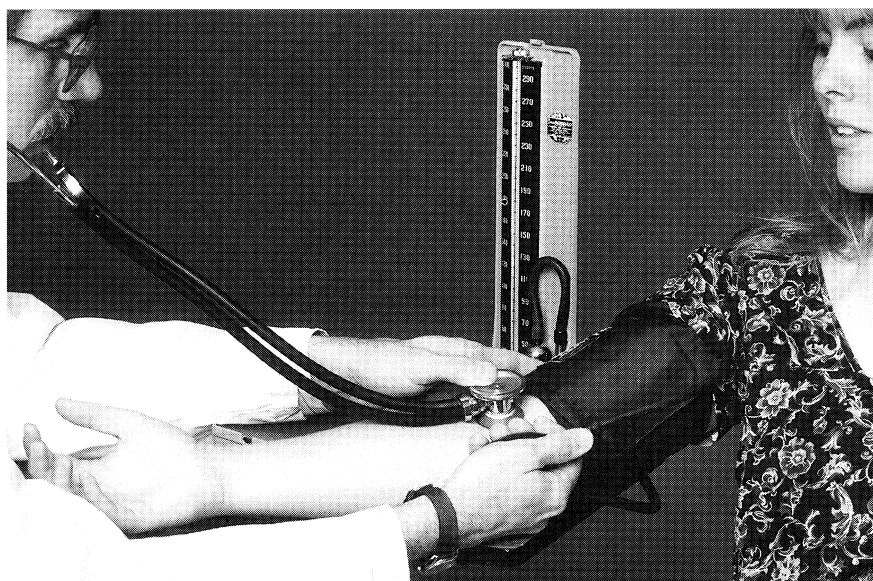


Figure 1.—Clinical measurement of indirect blood pressure.

Downloaded from www.jama.com at CALIF PACIFIC MEDICAL CTR, on August 8, 2007

much as 15/12 mm Hg higher or lower than today's result about 5% of the time. The greater magnitude of the between-visits within-visit variability is the reason why more visits are recommended to achieve greater diagnostic precision rather than more replications at a visit. In reality, the return BP reading in our clinical scenario will likely be lower, possibly much lower, because of our patient's present distress, unfamiliarity with the physician and the physician's office procedures, and "regression to the mean" (discussed herein).

Arrhythmias, particularly atrial fibrillation, cause beat-to-beat cardiac output to vary substantially and increase interobserver variation in measured BP.⁸¹ With atrial fibrillation, probably the best one can do is to deflate the cuff slowly while attempting to ascertain when most of the contractions are resulting in audible Korotkoff sounds (the approximate systolic BP) and when the sounds have all but ceased yet still occur infrequently (the approximate diastolic BP), or one can average several readings.^{18,20} Because Korotkoff sounds generated by occasional premature beats (and the subsequent beat) are unrepresentative of the day's mean BP level, they should be ignored.²²

Examiners can introduce random errors. Under ideal conditions, simultaneous BP readings by independent observers typically correlate above $r=0.95$ with mean absolute differences of less than 2 mm Hg systolic and less than 1 mm Hg diastolic.⁸² However, even in research settings, careful BP readings obtained just a few minutes apart show distressingly high variation (eg, SD of 7 mm Hg systolic and 5 mm Hg diastolic^{13,54,61}). In routine medical practice, physicians and nurses often measure BP far less carefully: differences of 10/8 mm Hg are common.^{83,84} White et al⁸⁵ performed intra-arterial BP recording in 48 hypertensive patients and found the humbling result that two auscultatory automatic monitors showed less overall discrepancy and fewer widely discrepant readings than did experienced clinicians using the standard method.

Environmental problems (eg, noise from construction work next door) or deficient equipment (eg, an inadequately damped, "bouncy" mercury column, remedied by tightening the knurled nut at the column's top²²) may also be expected to decrease precision.

Accuracy of BP Measurement

Accuracy, or validity, refers to agreement with the truth and requires not only precision but also freedom from systematic error (ie, bias). In clinical BP

Table 3.—Factors Affecting the Immediate Accuracy of Office BP*

Factor	Magnitude, SBP/DBP, mm Hg	Reference
Increases Recorded BP		
Examinee		
Soft Korotkoff sounds	DBP	Assumed
Missed auscultatory gap	DBP (rare, huge)	22
Pseudohypertension	2 to 98/3 to 49	23-25
"White coat" reaction		
To physician	11 to 28/3 to 15	26-30
To nonphysician	1 to 12/2 to 7	27, 31, 32
Paretic arm (due to stroke)	2/5	33
Pain, anxiety	May be large	22
Acute smoking	6/5	34
Acute caffeine	11/5	35
Acute ethanol ingestion	8/8	36
Distended bladder	15/10	37
Talking; signing	7/8	38; 39
Setting, equipment		
Environmental noise	DBP	Assumed
Leaky bulb valve	≥2 DBP	40
Blocked manometer vents	2 to 10	41
Cold hands or stethoscope	Not stated	22
Examiner		
Expectation bias	Probably <10	In theory
Impaired hearing	DBP	22
Examination		
Cuff too narrow	-8 to +10/2 to 8	42-44
Cuff not centered	4/3	45
Cuff over clothing	5 to 50	46
Elbow too low	6	47
Cuff too loose	Not stated	48
Too short rest period	Varied estimates	22
Back unsupported	6 to 10	49, 50
Arm unsupported	1 to 7/5 to 11	51
Too slow deflation	-1 to +2/5 to 6	52, 53
Too fast deflation	DBP only	52, 53
Parallax error	2 to 4	By author
Using phase IV (adult)	6 DBP	45
Too rapid remeasure	1/1	52, 54
Cold season (vs warm)	6/3 to 10	55-57

(continued)

measurement, we look through a series of dark glasses, further considered herein: (1) the indirect BP may not reflect the concurrent intra-arterial BP; (2) the cuff technique may be incorrectly performed; and (3) a perfectly executed indirect (or even direct) BP reading at a particular moment may not represent the patient's average clinic BP nor the average BP throughout the day's activities, as addressed in the section on ambulatory BP monitoring. Finally, to interpret even a perfect BP reading requires consideration of the whole patient because factors other than BP strongly influence the risk for CVD events.

Indirect BP vs Direct BP.—Indirect auscultatory BP correlates well with the simultaneous intra-arterial value ($r=0.94$ to 0.98).⁸⁶ However, the Korotkoff phase I sounds do not appear until 15 to 4 mm Hg below the direct systolic BP, whereas at phase V, the sounds disap-

pear 3 to 6 mm Hg above the true diastolic BP in adults.^{45,85,86}

If these technical differences applied equally to all patients, they would be merely academic; clinical importance arises when an individual patient possesses an unusual discrepancy. Such patients are often elderly (where false elevation is termed "pseudohypertension"^{23,24,87-89}) or obese,⁴⁶ but otherwise unexplained extreme false elevations in cuff BP may also occur.⁹⁰ Pseudohypertension might seem at first glance to be a variant of normal BP. However, most patients actually have chronic hypertension,⁹¹ on which is superimposed a further false BP elevation. Although it has been claimed that pseudohypertension can be suspected in an older person if "Osler's sign" (while feeling the radial pulse, occlude the brachial artery by cuff inflation or by direct pressure using the other thumb; if the radial artery remains

Table 3.—Factors Affecting the Immediate Accuracy of Office BP* (cont)

Factor	Magnitude, SBP/DBP, mm Hg	Reference
Decreases BP		
Examinee		
Soft Korotkoff sounds	SBP	Assumed
Recent meal	−1 to 1/1 to 4	58
Missed auscultatory gap	10 to 50 SBP	45
High stroke volume	Phase V can = 0	45
Habituation	0 to 7/2 to 12	59-61
Shock (additional pseudohypotension)	33 SBP	62
Setting, equipment		
Noisy environs	SBP	Assumed
Faulty aneroid device	Can be >10	63
Low mercury level	Varies	22
Leaky bulb	≥2 SBP	40
Examiner		
Reading to next lowest 5 or 10 mm Hg, or expectation bias	Probably ≤10	64
Impaired hearing	SBP only	22
Examination		
Noisy environs	SBP	Assumed
Left vs right arm	1/1	65
Resting for too long (25 min)	10/0	66
Elbow too high	5/5	47
Too rapid deflation	SBP only	40
Excess bell pressure	≥9 DBP	21
Parallax error (aneroid)	2 to 4	By author
No Effect on BP		
Examinee		
Menstrual phase	...	67, 68
Chronic caffeine ingestion	...	69
Phenylephrine nasal spray	...	70
Cuff self-inflation	...	71
Examinee and examiner		
Discordance in sex or race	...	72, 73
Examination		
Thin shirtsleeve under cuff	...	74
Bell vs diaphragm	...	49
Cuff inflation per se	...	29
Hour of day (during work hours)	...	54
Room temperature	...	54

*BP indicates blood pressure, and SBP and DBP indicate systolic and diastolic BP, respectively.

palpable as a firm “tube,” the sign is positive) is present,²⁴ the test’s usefulness remains debatable.^{25,91,92} For example, in 65 geriatric patients unanimously classified “Osler-positive” or “Osler-negative” by three observers, six other physicians demonstrated moderate intraobserver consistency ($\kappa=0.49$) and only modest interobserver agreement ($\kappa=0.37$).⁹³ Retaining “Osler-equivocal” patients in the study would almost certainly have further reduced agreement. Confirming pseudohypertension requires an intra-arterial BP measurement; fortunately, the condition is uncommon, affecting less than 2% of one otherwise healthy elderly group.²⁵

Technical Inaccuracies of Indirect BP.—Examiner biases include end-digit preference (ie, the tendency to over-record certain numbers, particularly 0 and 5^{64,75,94,95}), recording lower values at critical diagnostic cut points⁶⁴ presum-

ably to avoid institution of long-term drug treatment, and probably other analogous unconscious processes (eg, “observing” a hoped-for BP reduction consequent to instituting therapy). Physicians may also differ when labeling patients as hypertensive. A group of British general practitioners diagnosed hypertension after only one BP measurement in 58% of patients despite previously agreeing to use three readings as part of the group’s uniform diagnostic criteria.⁹⁶ Contrary to their local expert guidelines, about 37% of German out-of-hospital⁹⁷ physicians and British hospital clinicians⁹⁸ record phase IV (muffling) rather than the more accurate Korotkoff phase V. Perhaps the most common technical error is failure to use a sufficiently large cuff; indeed, in one survey, only 25% of primary care physicians even owned a large cuff.⁶³

Interestingly, even when an automatic

BP recorder is used, systematic differences between operators in the BP values obtained may remain,⁹⁹ suggesting differing examinee reactions to different examiners, as was seen in one careful study in children.¹⁰⁰

Directional equipment errors can occur. Aneroid instruments often go out of adjustment, usually downward.⁵² One survey found that 34% of practitioners used only aneroid units, of which 30% were off by 10 mm Hg or more.⁶³ A mercury unit can yield biased readings if the meniscus does not rest at 0 or if the mercury’s descent is impeded by clogged internal air vents.⁴¹ The stethoscope type seems relatively unimportant.⁴⁹ Although the recommended bell amplifies the Korotkoff sounds’ low frequencies in comparison with the diaphragm,¹⁰¹ the risk of exerting excessive pressure and obtaining a falsely low diastolic BP when using the bell²¹ may outweigh the benefit of amplification, particularly in thin patients (try a small bell with a rubber rim).

Examination errors are legion (Table 3); most overestimate the true BP. Note also that confirming an apparent difference between arms is not simple because it requires taking the averages of several alternating measurements from both sides or simultaneous measurements by two observers who then switch sides and remeasure.¹⁰²

Office BP vs Usual BP.—Shortly after entering the office, patients’ systolic BP declines by several millimeters of mercury, whereas diastolic BP remains relatively constant.^{53,59,60,66,100,103} Blood pressure remains fairly steady throughout the customary working daytime hours,⁵⁴ declines in the evening (ie, at home),^{104,105} and finally drops another 10% to 20% during sleep.^{106,107} In some patients, BP in a physician’s office is notably and consistently higher than daytime ambulatory BP. This phenomenon, termed “office” or “white coat” hypertension,¹⁰⁸ can even occur during self-measurement of cuff BP in the presence of a physician.²⁶ Approximately 10% to 40% of untreated and nominally borderline hypertensive patients show an appreciable “white coat” effect,^{27,109} and many treated patients will also show differences of greater than 20/10 mm Hg.^{109,110} The phenomenon may depend in part on patient factors such as sex, age, and BP level.¹¹¹ For example, one group of elderly patients showed an increase in BP of 17/7 mm Hg on entering the physician’s office; women showed a greater systolic BP rise than men.^{28,112} Who wears the white coat seems to matter, since nurses (who, along with technicians, have generally performed the BP measure-

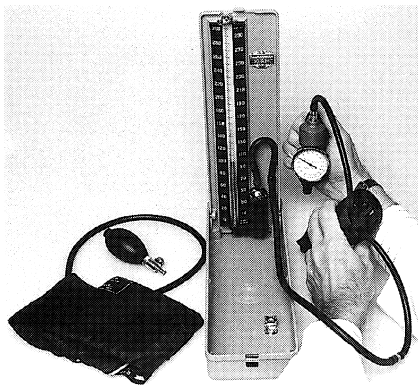


Figure 2.—How to calibrate a portable aneroid manometer against a mercury unit: (1) Disconnect both bulbs from the cuffs. Reconnect the aneroid bulb/dial assembly to the mercury unit's cuff (already connected to the column). Applying a few drops of water to the metal connector will facilitate its insertion into the rubber tubing. Close the aneroid assembly valve. Roll the cuff up loosely so that it is held by its Velcro strips. If necessary to prevent unrolling, first wrap the cuff around a sturdy large bottle or other surrogate "arm," or simply use a steady handgrip. (2) Gradually and steadily inflate the cuff until the mercury column rises to about 90 mm Hg. Holding the dial near the column, compare the two readings. (3) Obtain repeat readings at several other pressures, eg, 160 mm Hg, 220 mm Hg, 40 mm Hg. Modification for wall-mounted aneroid devices: Remove and set aside the aneroid cuff by disconnecting its tubing from its metal connector. Leave the dial tubing connector still coupled. Slightly inflate the mercury unit's cuff, and pinch the bulb tubing closed (fingers or clamp). Remove the bulb and replace it with the two coupled connectors running to the aneroid dial. Roll up the cuff. Squeeze the rolled-up cuff by hand to proceed as outlined in steps (2) and (3).

ments used for entry to the large clinical trials) seem to evoke a smaller BP increase than physicians.^{29,113}

THE ISSUE OF PREDICTION

BP Now vs BP Later

Systematic (and therefore at least partially predictable) changes in BP between visits occur for several reasons. As examinees (volunteers or patients) become more familiar with the examiner, environment, and procedure (including BP self-measurement²⁶), BP decreases by 0 to 7 mm Hg systolic and 2 to 12 mm Hg diastolic.⁵⁹⁻⁶¹ This habituation may be more marked in patients with anxiety trait.¹¹⁴ An additional and probably more important factor,¹¹⁵ regression to the mean, represents the tendency for any unusually high (or low) reading to fall closer to the population mean when repeated. These phenomena are distinguishable from a true "placebo" effect since they can occur in the absence of placebo treatment.^{13,59,116} Some BP changes likely represent currently unappreciated systematic influences; for example, a systematic reduction in BP of about 6 mm Hg occurs during warm vs cold seasons.⁵⁵⁻⁵⁷

Major outcome trials of antihypertensive pharmacotherapy have used two to three BP readings taken at each of two or more visits not only to increase precision (by "averaging out" minute-to-minute and between-day random fluctuations), but also to partially control for regression to the mean and habituation. In practice, following the same multivisit protocol helps ensure that published trial results will be applicable to individual patients. (A further refinement, used naturally by many experienced clinicians, is to conduct further follow-up visits when the BP is hovering near a diagnostic cut point. Patients whose true values are far from this threshold [above or below] logically need fewer visits for confident classification.¹¹⁷) In practice, the interval between visits should take into account both the BP level and the patient's clinical status. The Joint National Committee³ recommends remeasurement within 1 month for BP initially in the range of 160 to 179 mm Hg systolic or 100 to 109 mm Hg diastolic (ie, stage 2), within 2 months for stage 1, within 1 week for stage 3, and immediate evaluation for stage 4.

Relative Risk of Casual BP Elevation for Persistent Hypertension

Given high random variation, how well does the finding of a single elevated BP predict later definite hypertension? Casual BP, particularly systolic BP at one visit, is predictive of later BP elevation in young men,¹¹⁸ medical students,¹¹⁹ adults,¹³ and children.¹²⁰ (Tracking correlations vary widely; for example, $r=0.2$ to 0.7 , depending on the population, technique, and follow-up interval.) In a large prospective study,¹²¹ one diastolic BP reading greater than or equal to 90 mm Hg predicted a later definite diagnosis of hypertension in 69% of men and 49% of women; any BP elevation warrants careful follow-up. Looked at the other way, however, about one third to one half of subjects with initially elevated BP will ultimately prove not to have hypertension. In practice, regression to the mean guarantees that many individuals with initially elevated BP are really normotensive.⁷⁵ For example, among subjects with four diastolic BP measurements at two entry visits averaging between 95 to 104 mm Hg in a mild hypertension trial in Australia,¹²² 28% proved to have an average diastolic BP less than 90 mm Hg during the next 4 years on placebo. In a careful screening program, similar diastolic BP reductions were seen in the 105 to 114 mm Hg stratum from the first to second screen, and approximately 10% of subjects with diastolic BP greater than or equal to 115 mm Hg were normotensive (<90 mm Hg) at the next visit.¹¹⁶

Therefore, using the mean of several visits' BP readings improves the ability to predict not only future hypertension¹²⁰ but also CVD sequelae.¹³ Because he may be normotensive, the patient in our case scenario should not be told that he is hypertensive at this initial visit,⁴ but he should be carefully followed up.

Is a High BP Value Ever Normal?

In normotensive subjects, aerobic exercise, which is generally accepted to be good for health, causes systolic BP to increase moderately while diastolic BP changes little.^{123,124} Since increased BP forms part of the "fight or flight" response, pain (eg, a lacerated finger) and other stresses (eg, pulmonary edema) predictably raise BP, sometimes to extreme values. These reactive elevations of BP do not indicate the presence of "hypertension" if the BP returns to normal levels at rest.

How Do I Improve My Technique?

Checking one's equipment periodically is mandatory to preserve accuracy.¹⁹ Aneroid devices should be recalibrated at least every 6 months. The recommended approach involves inserting a Y-connector somewhere in the pressurized path between bulb and mercury column and attaching the aneroid device. An alternative method not requiring a Y-connector is given in Figure 2. Although one can measure arm circumference in each patient to select an appropriately sized cuff, one can more efficiently mark the limit of arm circumference directly on each cuff by drawing a line in indelible ink at a distance from the free bladder end equal to twice the measured bladder width.

Tape recordings can help standardize observers' identification of Korotkoff sounds.^{84,125} Alternatively, locate a two-headed stethoscope (and a second set of ears attached to a willing expert brain) for hands-on training. Initial formal training in the technique of BP measurement is necessary, but in addition, periodic review of technique and retraining as needed are recommended.⁴ Retraining can increase accuracy,⁸³ but may be needed every 1 to 2 months for optimal effect,¹²⁶ a frequency probably practical only in research settings. Atrial fibrillation requires a modified technique (discussed earlier). When faced with soft Korotkoff sounds, have the subject elevate the arm and then open and close the fist several times; inflate the cuff, lower the arm (with further inflation as needed), and listen again. In this situation, as permitted by some guidelines,⁴ more rapid deflation after determination of the systolic BP until the vicinity of the diastolic BP will minimize attenuation of the Korotkoff sounds arising from ve-

nous congestion without altering the measured BP.⁵³ Applying the cuff with its tubing emerging at the top¹⁹ will eliminate extraneous noises generated if tubing contacts the stethoscope.

For research purposes, random-zero sphygmomanometers will reduce but still not eliminate observer bias. Fully automatic devices, if otherwise technically accurate, should eliminate certain human foibles (eg, end-digit preference⁹⁴ and selective recording of "desirable" readings). Statistical monitoring⁵⁴ to detect end-digit preference or excessive variability followed by mandatory retraining should be helpful.

In practice, the grossest error, not checking BP at all, remains a common failing even among cardiovascular subspecialists.¹²⁷ Most measurement errors could be obviated if practitioners would only follow the published recommendations^{19,128-132}; alas, many do not.^{63,96-98}

Other Ways to Measure BP

Lacking the ability to hear properly, both systolic and diastolic BP (as noted by Janeway, among others) can be determined by palpation to within about 10 mm Hg.¹³³ Palpated systolic BP is about 7 mm Hg lower than the auscultatory value.¹³⁴

Potential Improvements in the Diagnosis of Hypertension

Elevated BP during aerobic exercise testing in subjects normotensive at rest has some predictive value for subsequent definite hypertension (relative risk from 2.3 to approximately 7).^{123,124,135} Because BP is so variable during daily activity, ambulatory BP monitoring¹³⁶⁻¹³⁸ ought to provide a better estimate of whole-day target organ exposure. Ambulatory BP monitoring correlates better with coexisting target organ damage¹³⁸ and a retrospective follow-up study suggested improved prediction for subsequent CVD.¹³⁹ However, some patients cannot tolerate ambulatory BP monitoring and accurate measurements are not always possible (eg, with marked arrhythmia or obesity).¹⁴⁰ Although appropriate studies have begun,¹⁴¹ no data yet exist to show that adding ambulatory BP monitoring results in clinical benefit, and issues such as cost-effectiveness remain.¹³⁶

Self-measurement of BP¹⁴² is also under active study. Concurrent accuracy,⁸² the meaning of differences in measurements at home and at work,¹⁴³ and concerns about selective reporting remain. When patients bring in their own, usually lower home BP readings, be certain to explain that only antihypertensive treatment of resting BP readings is of proven value, that daytime BP is rou-

tinely higher than evening BP, and that cardiac involvement may relate more closely to worktime BP.¹⁴⁴ Although the appeal of self-monitoring includes potentially desirable psychological and compliance effects, any benefit remains questionable¹⁴²; a 1-year trial of home BP monitoring found no difference in treatment, attained BP, or risk factor reduction.¹⁴⁵

THE BOTTOM LINE

Hypertension remains one of the most prevalent and most important public health problems. Measurement of BP has won its place in the recommended periodic health examination because hypertension is common, clinically silent, dangerous, and treatable. Accurately measuring BP by the indirect method requires minimal equipment combined with a willingness to make the effort; all health care practitioners should read and follow published guidelines.¹⁸ Attention to proper technique plus an appreciation of the inherent variability of BP should yield an accurate diagnosis in most patients. Occasional patients with suspected pseudohypertension or white coat syndrome may benefit from ancillary technology such as echocardiography or ambulatory BP monitoring to optimize diagnostic decision making. Conversely, in the far more common, otherwise low-risk patient, yearly BP readings will suffice to exclude the presence of severe or long-standing untreated hypertension. The patient in our clinical scenario would be served well by a return visit in a few weeks for repeat BP measurement,³ whereas immediately labeling him as hypertensive would be incorrect and, by causing him unnecessary concern, could be an immediate disservice.

Following expert treatment guidelines constitutes the physician's final responsibility, tying a proper diagnosis and proven therapy together to benefit the patient.

Dr Reeves was supported by a Career Award from the Pharmaceutical Manufacturers Association of Canada/Medical Research Council of Canada Combined Program.

References

1. Dept of National Health and Welfare. *Main Findings Report of the Canadian Blood Pressure Survey*. Ottawa, Ontario: Minister of National Health and Welfare; 1989:1.
2. Hypertension Detection and Follow-up Program. The Hypertension Detection and Follow-up Program: a progress report. *Circ Res*. 1977;40(suppl 1):I-106-I-109.
3. Joint National Committee. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V). *Arch Intern Med*. 1993;153:154-183.
4. Haynes RB, Lacourciere Y, Rabkin SW, et al. Report of the Canadian Hypertension Society Consensus Conference, 2: diagnosis of hypertension in

- adults. *Can Med Assoc J*. 1993;149:409-418.
5. Zanchetti A, Chalmers JP, Arakawa K, et al. The 1993 guidelines for the management of mild hypertension: memorandum from a WHO/ISH meeting. *Hypertension*. 1993;22:392-403.
6. Swales JD, Ramsay LE, Coope JR, et al. Treating mild hypertension, agreement from the large trials: report of the British Hypertension Society Working Party. *BMJ*. 1989;298:694-698.
7. Jackson R, Barham P, Bills J, et al. Management of raised blood pressure in New Zealand: a discussion document. *BMJ*. 1993;307:107-110.
8. Haynes RB, Sackett DL, Taylor DW, Gibson ES, Johnson AL. Increased absenteeism from work after detection and labeling of hypertensive patients. *N Engl J Med*. 1978;299:741-744.
9. Lefebvre RC, Hursey KG, Carleton RA. Labeling of participants in high blood pressure screening programs: implications for blood cholesterol screenings. *Arch Intern Med*. 1988;148:1993-1997.
10. MacMahon SW, Cutler JA, Furberg CD, Payne GH. The effects of drug treatment for hypertension on morbidity and mortality from cardiovascular disease: a review of randomized controlled trials. *Prog Cardiovasc Dis*. 1986;29:99-118.
11. Collins R, Peto R, MacMahon S, et al. Blood pressure, stroke, and coronary heart disease, part 2, short-term reductions in blood pressure: overview of randomized drug trials in their epidemiological context. *Lancet*. 1990;335:827-838.
12. Kannel WB. Some lessons in cardiovascular epidemiology from Framingham. *Am J Cardiol*. 1976;37:269-282.
13. Gordon T, Sorlie P, Kannel WB. Problems in the assessment of blood pressure: the Framingham Study. *Int J Epidemiol*. 1976;5:327-334.
14. Waaler HT. Specificity and sensitivity of blood pressure measurements. *J Epidemiol Community Health*. 1980;34:52-58.
15. Fujii I, Ueda K, Omae T, et al. Natural history of borderline hypertension in the Hisayama community, Japan—I: the relative prognostic importance of transient variability in blood pressure. *J Chronic Dis*. 1984;37:895-902.
16. Beard K, Bulpitt C, Mascie-Taylor H, O'Malley K, Sever P, Webb S. Management of elderly patients with sustained hypertension. *BMJ*. 1992;304:412-416.
17. Jennings D, Netsky MG. Essential hypertension: a sign in search of a disease. *Can Med Assoc J*. 1991;144:973-979.
18. Perloff D, Grim C, Flack J, et al. Human blood pressure determination by sphygmomanometry. *Circulation*. 1993;88:2460-2470.
19. Petrie JC, O'Brien ET, Littler WA, de Swiet M. Recommendations on blood pressure measurement. *BMJ*. 1986;293:611-615.
20. American Society of Hypertension. Recommendations for routine blood pressure measurement by indirect cuff sphygmomanometry. *Am J Hypertens*. 1992;5:207-209.
21. Londe S, Klitzner TS. Auscultatory blood pressure measurement: effect of pressure on the head of the stethoscope. *West J Med*. 1984;141:193-195.
22. Frohlich ED, Grim C, Labarthe DR, Maxwell MH, Perloff D, Weidman WH. Recommendations for human blood pressure determination by sphygmomanometers: report of a special task force appointed by the steering committee, AHA. *Hypertension*. 1988;11:210a-221a.
23. Spence JD, Sibbald WJ, Cape RD. Pseudohypertension in the elderly. *Clin Sci Mol Med*. 1978;55(suppl 4):399s-402s.
24. Messerli FH, Ventura HO, Amodeo C. Osler's maneuver and pseudohypertension. *N Engl J Med*. 1985;312:1548-1551.
25. Kuwajima I, Hoh E, Suzuki Y, Matsushita S, Kuramoto K. Pseudohypertension in the elderly. *J Hypertens*. 1990;8:429-432.
26. Mengden T, Böttig B, Edmonds D, et al. Self-measured blood pressures at home and during consulting hours: are there any differences? *J Hypertens Suppl*. 1990;8:S15-S19.
27. Pickering TG, James GD, Boddie C, Harshfield GA, Blank S, Laragh JH. How common is white

- coat hypertension? *JAMA*. 1988;259:225-228.
28. Shimada K, Ogura H, Kawamoto A, Matsubayashi K, Ishida H, Ozawa T. Noninvasive ambulatory blood pressure monitoring during clinic visit in elderly hypertensive patients. *Clin Exp Hypertens*. 1990;12:151-170.
29. Mancia G, Parati G, Pomidossi G, Grassi G, Casadei R, Zanchetti A. Alerting reaction and rise in blood pressure during measurement by physician and nurse. *Hypertension*. 1987;9:209-215.
30. Kenny RA, Brennan M, O'Malley K, O'Brien E. Blood pressure measurements in borderline hypertension. *J Hypertens*. 1987;5(suppl):483-485.
31. Laughlin KD, Sherrard DJ, Fisher L. Comparison of clinic and home blood pressure levels in essential hypertension and variables associated with clinic-home differences. *J Chronic Dis*. 1980;33:197-206.
32. Porchet M, Bussien JP, Waerber B, Nussberger J, Brunner HR. Unpredictability of blood pressures recorded outside the clinic in the treated hypertensive patient. *J Cardiovasc Pharmacol*. 1986;8:332-335.
33. Yagi S, Ichikawa S, Sakamaki T, Takayama Y, Murata K. Blood pressure in the paretic arms of patients with stroke. *N Engl J Med*. 1986;315:836.
34. Benowitz NL, Kuyt F, Jacob PI. Influence of nicotine on cardiovascular and hormonal effects of cigarette smoking. *Clin Pharmacol Ther*. 1984;36:74-81.
35. Robertson D, Wade D, Workman R, Woosley RL. Tolerance to the humoral and hemodynamic effects of caffeine in man. *J Clin Invest*. 1981;67:1111-1117.
36. Potter JF, Watson RDS, Skan W, Beevers G. The pressor and metabolic effects of alcohol in normotensive subjects. *Hypertension*. 1986;8:625-631.
37. Fagius J, Karhuvaara S. Sympathetic activity and blood pressure increases with bladder distension in humans. *Hypertension*. 1989;14:511-517.
38. Lynch JJ, Thomas SA, Long JM, Malinow KL, Friedmann E, Katcher AH. Blood pressure changes while talking. *Isr J Med Sci*. 1982;18:575-579.
39. Malinow KL, Lynch JJ, Foreman PJ, Friedmann E, Thomas SA. Blood pressure increases while signing in a deaf population. *Psychosom Med*. 1986;48:95-101.
40. Conceicao S, Ward MK, Kerr DNS. Defects in sphygmomanometers: an important source of error in blood pressure recording. *BMJ*. 1976;1:886-888.
41. Shaw A, Deehan C, Lenihan JMA. Sphygmomanometers: errors due to blocked vents. *BMJ*. 1979;1:789-790.
42. Steinfeld L, Alexander H, Cohen ML. Updating sphygmomanometry. *Am J Cardiol*. 1974;33:107-110.
43. Russell AE, Wing LMH, Smith SA, et al. Optimal size of cuff bladder for indirect measurement of arterial pressure in adults. *J Hypertens*. 1989;7:607-613.
44. Maxwell MH, Waks AU, Schroth PC, Karam M, Dornfeld LP. Error in blood-pressure measurement due to incorrect cuff size in obese patients. *Lancet*. 1982;2:33-36.
45. Webb CH. The measurement of blood pressure and its interpretation. *Primary Care*. 1980;7:637-651.
46. Trout KW, Bertrand CA, Williams MH. Measurement of blood pressure in obese persons. *JAMA*. 1956;162:970-971.
47. Mitchell PL, Parlin RW, Blackburn H. Effect of vertical displacement of the arm on indirect blood-pressure measurement. *N Engl J Med*. 1964;271:72-74.
48. Neussle WF. The importance of a tight blood pressure cuff. *Am Heart J*. 1956;52:905-907.
49. Cushman WC, Cooper KM, Horne RA, Meydrech EF. Effect of back support and stethoscope head on seated blood pressure determinations. *Am J Hypertens*. 1990;3:240-241.
50. Viol GW, Goebel M, Lorenz GJ, Ing TS. Seating as a variable in clinical blood pressure measurement. *Am Heart J*. 1979;98:813-814.
51. Silverberg DS, Shemesh E, Iaina A. The unsupported arm: a cause of falsely raised blood pressure readings. *BMJ*. 1977;2:1331.
52. Thulin T, Andersson G, Schersten B. Measurement of blood pressure: a routine test in need of standardization. *Postgrad Med J*. 1975;51:390-395.
53. King GE. Influence of rate of cuff inflation and deflation on observed blood pressure by sphygmomanometry. *Am Heart J*. 1963;65:303-306.
54. Canner PL, Borhani NO, Oberman A, et al. The Hypertension Prevention Trial: assessment of the quality of blood pressure measurements. *Am J Epidemiol*. 1991;134:379-392.
55. Izzo JL, Larrabee PS, Sander E, Lillis LM. Hemodynamics of seasonal adaptation. *Am J Hypertens*. 1990;3:405-407.
56. Kunes J, Tremblay J, Bellavance F, Hamet P. Influence of environmental temperature on the blood pressure of hypertensive patients in Montreal. *Am J Hypertens*. 1991;4:422-426.
57. Giacconi S, Palombo C, Genovesi-Ebert A, Marabotti C, Volterrani D, Ghione S. Long-term reproducibility and evaluation of seasonal influences on blood pressure monitoring. *J Hypertens*. 1988;6(suppl 4):S64-S66.
58. Mader SL. Effects of meals and time of day on postural blood pressure responses in young and elderly subjects. *Arch Intern Med*. 1989;149:2757-2760.
59. Armitage P, Rose GA. The variability of measurements of casual blood pressure. I: a laboratory study. *Clin Sci*. 1966;30:325-335.
60. Watson RDS, Lumb R, Young MA, Stallard TJ, Davies P, Littler WA. Variation in cuff blood pressure in untreated outpatients with mild hypertension: implications for initiating antihypertensive treatment. *J Hypertens*. 1987;5:207-211.
61. Shepard DS. Reliability of blood pressure measurements: implications for designing and evaluating programs to control hypertension. *J Chronic Dis*. 1981;34:191-209.
62. Cohn JN. Blood pressure measurement in shock: mechanism of inaccuracy in auscultatory and palpatory methods. *JAMA*. 1967;199:972-976.
63. McKay DW, Campbell NR, Parab LS, Chockalingam A, Fodor JG. Clinical assessment of blood pressure. *J Hum Hypertens*. 1990;4:639-645.
64. Patterson HR. Sources of error in recording the blood pressure of patients with hypertension in general practice. *BMJ*. 1984;289:1661-1664.
65. Hashimoto F, Hunt WC, Hardy L. Differences between right and left arm blood pressures in the elderly. *West J Med*. 1984;141:189-192.
66. van Loo JM, Peer PG, Thien TA. Twenty-five minutes between blood pressure readings: the influence on prevalence rates of isolated systolic hypertension. *J Hypertens*. 1986;4:631-635.
67. von Eiff AW, Plotz EJ, Beck KJ, Czernik A. The effect of estrogens and progestins on blood pressure regulation of normotensive women. *Am J Obstet Gynecol*. 1971;109:887-892.
68. Polefrone JM, Manuck SB. Effects of menstrual phase and parental history of hypertension on cardiovascular response to cognitive challenge. *Psychosom Med*. 1988;50:23-36.
69. Myers MG, Reeves RA. The effect of caffeine on daytime ambulatory blood pressure. *Am J Hypertens*. 1991;4:427-431.
70. Myers MG, Iazzetta JJ. Intranasally administered phenylephrine and blood pressure. *Can Med Assoc J*. 1982;127:365-368.
71. Veerman DP, van Montfrans GA, Karemaker JM, Wieling W. Inflating one's own cuff does not increase self-recorded blood pressure. *J Hypertens*. 1992;6(suppl 4):S77-S78.
72. Kraus JF, Conley A, Hardy R, Sexton M, Swezey Z. Relationship of demographic characteristics of interviewers to blood pressure measurements. *J Community Health*. 1982;3:3-12.
73. Reeves RA, Lewis JE, Hebert PC, Kernerman Y, Revah A. Gender, status and 'white coat' phenomenon in family practice. *Clin Invest Med*. 1991;14(suppl A):A69.
74. Holleman DR, Westman EC, McCrory DC, Simel DL. The effect of sleeved arms on oscillometric blood pressure measurement. *J Gen Intern Med*. 1993;8:325-326.
75. Hypertension Detection and Follow-up Program. Variability of blood pressure and the results of screening in the Hypertension Detection and Follow-up Program. *J Chronic Dis*. 1978;31:651-667.
76. Di Rienzo M, Parati G, Pomidossi G, Veniani M, Pedotti A, Mancia G. Blood pressure monitoring over short day and night times cannot predict 24-hour average blood pressure. *J Hypertens*. 1985;3:343-349.
77. Mancia G, Parati G, Pomidossi G, Casadei R, Di Rienzo M, Zanchetti A. Arterial baroreflexes and blood pressure and heart rate variabilities in humans. *Hypertension*. 1986;8:147-153.
78. Watson RDS, Stallard TJ, Flinn RM, Littler WA. Factors determining direct arterial pressure and its variability in hypertensive man. *Hypertension*. 1980;2:333-341.
79. Reeves RA. A review of the stability of ambulatory blood pressure: implications for diagnosis of hypertension. *Clin Invest Med*. 1991;14:251-255.
80. Reeves RA, Leenen FHH, Joyner CD. Reproducibility of nurse-measured, exercise and ambulatory blood pressure and echocardiographic left ventricular mass in borderline hypertension. *J Hypertens*. 1992;10:1249-1256.
81. Sykes D, Dewar R, Mohanaruban K, et al. Measuring blood pressure in the elderly: does atrial fibrillation increase observer variability? *BMJ*. 1990;300:162-163.
82. O'Brien E, Mee F, Atkins N, O'Malley K. Inaccuracy of seven popular sphygmomanometers for home measurement of blood pressure. *J Hypertens*. 1990;8:621-634.
83. Scherwitz LW, Evans LA, Hennrikus DJ, Vallbona C. Procedures and discrepancies of blood pressure measurements in two community health centers. *Med Care*. 1982;20:727-738.
84. Neufeld PD, Johnson DL. Observer error in blood pressure measurement. *Can Med Assoc J*. 1986;135:633-637.
85. White WB, Lund-Johansen P, Omvik P. Assessment of four ambulatory blood pressure monitors and measurements by clinicians versus intra-arterial blood pressure at rest and during exercise. *Am J Cardiol*. 1990;65:60-66.
86. Blank SG, West JE, Müller FB, et al. Wideband external pulse recording during cuff deflation: a new technique for evaluation of the arterial pressure pulse and measurement of blood pressure. *Circulation*. 1988;77:1297-1305.
87. Taguchi JT, Suwagool P. Pipe-stem brachial arteries: a cause of pseudohypertension. *JAMA*. 1974;228:733.
88. Wallace CT, Carpenter FA, Evins SC, Mahaffey JE. Acute pseudohypertensive crisis. *Anesthesiology*. 1975;43:588-589.
89. Keenan WF. Pseudohypertension mimicking a hypertensive emergency. *JAMA*. 1981;246:1088.
90. Saklayen MG. Pseudohypertension in a young woman. *Am J Med*. 1988;84:794-795.
91. O'Rourke M, Kelly R. Osler's maneuver and pseudohypertension. *N Engl J Med*. 1985;313:1300.
92. Tift CP. Are the days of the sphygmomanometer past? *Arch Intern Med*. 1988;148:518-519.
93. Hla KM, Samsa GP, Stoneking HT, Feussner JR. Observer variability of Osler's maneuver in detection of pseudohypertension. *J Clin Epidemiol*. 1991;44:513-518.
94. Hla KM, Vokaty KA, Feussner JR. Observer error in systolic blood pressure measurement in the elderly: a case for automatic recorders? *Arch Intern Med*. 1986;146:2373-2376.
95. Padfield PL, Jyothinagaram SG, Watson DM, Donald P, McGinley IM. Problems in the measurement of blood pressure. *J Hum Hypertens*. 1990;4(suppl 2):3-7.
96. Smith TD, Clayton D. Individual variation between general practitioners in labelling of hypertension. *BMJ*. 1990;300:74-75.
97. Weiland SK, Keil U, Spelsberg A, et al. Diagnosis and management of hypertension by physicians in the Federal Republic of Germany. *J Hypertens*. 1991;9:131-134.

98. Wilkinson LS, Perry IJ, Shinton RA, Beevers DG. An emerging consensus among clinicians on treating mild hypertension but persistent uncertainty as to how blood pressure should be measured. *J R Coll Physicians Lond*. 1991;25:116-119.
99. Bruce NG, Cook DG, Shaper AG. Differences between observers in blood pressure measurement with an automatic oscillometric recorder. *J Hypertens Suppl*. 1990;8:S11-S13.
100. Burke GL, Webber LS, Shear CL, Zinkgraf SA, Smoak CG, Berenson GS. Sources of error in measurement of children's blood pressure in a large epidemiologic study: Bogalusa Heart Study. *J Chronic Dis*. 1987;40:83-89.
101. Abella M, Formolo J, Penney DG. Comparison of the acoustic properties of six popular stethoscopes. *J Acoust Soc Am*. 1992;91:2224-2228.
102. Sapira JD. The vital signs. In: Sapira JD, ed. *The Art and Science of Bedside Diagnosis*. Baltimore, Md: Urban & Schwarzenberg; 1990:85-104.
103. Wietlisbach V, Rickenbach M, Burnand B, Hauser D, Gutzwiller F. Combining repeated blood pressure measurements to obtain prevalences of high blood pressure. *Acta Med Scand Suppl*. 1988;728:165-168.
104. Millar-Craig MW, Bishop CN, Raftery EB. Circadian variation of blood pressure. *Lancet*. 1978;1:795-797.
105. Gould BA, Hornung RS, Cashman PMM, Raftery EB. Ambulatory blood pressure—direct and indirect. In: Weber MA, Drayer JIM, eds. *Ambulatory Blood Pressure Monitoring*. New York, NY: Springer-Verlag NY Inc; 1984:9-19.
106. Littler WA, Honour AJ, Carter RD, Sleight P. Sleep and blood pressure. *BMJ*. 1975;1:346-347.
107. Millar-Craig MW, Mann S, Balasubramanian V, Raftery EB. Blood pressure circadian rhythm in essential hypertension. *Clin Sci Mol Med*. 1978;55:391s-393s.
108. Kleinert HD, Harshfield GA, Pickering TG, et al. What is the value of home blood pressure measurement in patients with mild hypertension? *Hypertension*. 1984;6:574-578.
109. Ornstein S, Markert G, Litchfield L, Zemp L. Evaluation of the DINAMAP blood pressure monitor in an ambulatory primary care setting. *J Fam Pract*. 1988;26:517-521.
110. Myers MG, Reeves RA. White coat phenomenon in patients receiving antihypertensive therapy. *Am J Hypertens*. 1991;4:844-849.
111. Silagy CA, McNeil JJ, McGrath BP. Isolated systolic hypertension: does it really exist on ambulatory blood pressure monitoring? *Clin Exp Pharmacol Physiol*. 1990;17:203-206.
112. Imai Y, Nakatsuka H, Ikeda M, et al. A cross-sectional survey of home blood pressure in a rural community in northern Japan. *Clin Exp Hypertens*. 1990;12:1095-1106.
113. Ellis PJ, Marshall E, Ellis SJ. Which blood pressure? *Lancet*. 1988;2:902-903.
114. McGrady A, Higgins JT Jr. Effect of repeated measurements of blood pressure on blood pressure in essential hypertension: role of anxiety. *J Behav Med*. 1990;13:93-101.
115. Haynes RB, Logan AG, Flanagan PT, Milne BJ. The effect of patient 'familiarity' with blood pressure assessment on the accuracy of follow-up readings. *J Hypertens*. 1983;1:91-94.
116. Hypertension Detection and Follow-up Program Cooperative Group. Blood pressure studies in 14 communities: a two-stage screen for hypertension. *JAMA*. 1977;237:2385-2391.
117. Perry HM Jr, Miller JP. Difficulties in diagnosing hypertension: implications and alternatives. *J Hypertens*. 1992;10:887-896.
118. Rabkin SW, Mathewson FA, Tate RB. Relationship of blood pressure in 20- to 39-year-old men to subsequent blood pressure and incidence of hypertension over a 30-year observation period. *Circulation*. 1982;65:291-300.
119. Thomas CB, Duszynski KR. Blood pressure levels in young adulthood as predictors of hypertension and the fate of the cold pressor test. *Johns Hopkins Med J*. 1982;151:93-100.
120. Shear CL, Burke GL, Freedman DS, Berenson GS. Value of childhood blood pressure measurements and family history in predicting future blood pressure status: results from 8 years of follow-up in the Bogalusa Heart Study. *Pediatrics*. 1986;77:862-869.
121. Trembath CR, Hickner JM, Bishop SW. Incidental blood pressure elevations: a MIRNET project. *J Fam Pract*. 1991;32:378-381.
122. Management Committee. The Australian therapeutic trial in mild hypertension. *Lancet*. 1980;1:1261-1267.
123. Dlin RA, Hanne N, Silverberg DS, Bar-Or O. Follow-up of normotensive men with exaggerated blood pressure response to exercise. *Am Heart J*. 1983;106:316-320.
124. Guerrero G, Melina D, Colivicchi F, Santoliquido A, Folli G. Abnormal blood pressure response to exercise in borderline hypertension: a two-year follow-up study. *Am J Hypertens*. 1991;4:271-273.
125. Rose GA. Standardisation of observers in blood-pressure measurement. *Lancet*. 1965;1:673-674.
126. Bruce NG, Shaper AG, Walker M, Wannamethee G. Observer bias in blood pressure studies. *J Hypertens*. 1988;6:375-380.
127. Warshaw LJ. Physicians: check that blood pressure. *JAMA*. 1989;262:1775-1776.
128. Campbell NR, Chockalingam A, Fodor JG, McKay DW. Accurate, reproducible measurement of blood pressure. *Can Med Assoc J*. 1990;143:19-24.
129. O'Brien ET, O'Malley K. ABC of blood pressure measurement: the observer. *BMJ*. 1979;2:775-776.
130. O'Brien ET, O'Malley K. ABC of blood pressure measurement: the sphygmomanometer. *BMJ*. 1979;2:851-853.
131. O'Brien ET, O'Malley K. ABC of blood pressure measurement: the patient. *BMJ*. 1979;2:920-921.
132. O'Brien ET, O'Malley K. ABC of blood pressure measurement: technique. *BMJ*. 1979;2:982-984.
133. Segall HN. A note on the measurement of diastolic and systolic blood pressure by the palpation of arterial vibrations (sounds) over the brachial artery. *Can Med Assoc J*. 1940;42:311-313.
134. Scordo K. Using radial artery palpation to monitor blood pressure. *Dimens Crit Care Nurs*. 1983;2:75-79.
135. Wilson NV, Meyer BM. Early prediction of hypertension using exercise blood pressure. *Prev Med*. 1981;10:62-68.
136. Reeves RA, Myers MG. Ambulatory blood pressure monitoring: an emerging technology. *Clin Invest Med*. 1991;14:198-201.
137. Health and Public Policy Committee, American College of Physicians. Automated ambulatory blood pressure monitoring. *Ann Intern Med*. 1986;104:275-278.
138. National High Blood Pressure Education Program Coordinating Committee. National High Blood Pressure Education Program Working Group report on ambulatory blood pressure monitoring. *Arch Intern Med*. 1990;150:2270-2280.
139. Perloff D, Sokolow M, Cowan R. The prognostic value of ambulatory blood pressures. *JAMA*. 1983;249:2792-2798.
140. Reeves RA. Patient and environmental factors affecting ambulatory blood pressure monitoring. *Clin Invest Med*. 1991;14:218-223.
141. Clement DL, the Steering Committee. Office versus ambulatory recordings of blood pressure (OvA): a European multicenter study. *J Hypertens Suppl*. 1990;8:S39-S41.
142. Canadian Coalition for High Blood Pressure Prevention and Control. Recommendations on self-measurement of blood pressure. *Can Med Assoc J*. 1988;138:1093-1096.
143. Pickering TG, James GD. Some implications of the differences between home, clinic and ambulatory blood pressure in normotensive and hypertensive patients. *J Hypertens Suppl*. 1989;7:S65-S72.
144. Devereux RB, Pickering TG, Harshfield GA, et al. Left ventricular hypertrophy in patients with hypertension: importance of blood pressure response to regularly recurring stress. *Circulation*. 1983;68:470-476.
145. Midanik LT, Resnick B, Hurley LB, Smith EJ, McCarthy M. Home blood pressure monitoring for mild hypertensives. *Public Health Rep*. 1991;106:85-89.