CME CREDIT

RADHIKA KATAKAM, MD
Instructor in Medicine, Henry Ford Health System, Detroit, MI

KIRSTEN BRUKAMP, MD
University of Pennsylvania, Philadelphia

RAYMOND R. TOWNSEND, MD*
Renal Electrolyte and Hypertension Division, University of Pennsylvania, Philadelphia

What is the proper workup of a patient with hypertension?

ABSTRACT

Because hypertension is common and many tests are available, an uncritical approach to laboratory and radiologic evaluation leads to unnecessary expenses. However, in most patients, accurate blood pressure measurement, a focused history and physical examination, and a handful of basic tests are enough. In this review we address the key questions in the evaluation of the patient with an elevated pressure reading, ie, does the patient have sustained high blood pressure? And if so, is the hypertension primary or secondary, are other cardiovascular risk factors present, and is there evidence of target organ damage?

KEY POINTS

To confirm the diagnosis of hypertension, multiple readings should be taken at various times.

Proper technique is important in measuring blood pressure, including using the correct cuff size, having the patient sit quietly for 5 minutes before taking the pressure, and supporting the arm at the level of the heart.

If white-coat hypertension is suspected, one can consider ambulatory or home blood pressure measurements to confirm that the hypertension is sustained.

How extensive a workup does a patient with high blood pressure need?

On one hand, we would not want to start therapy on the basis of a single elevated reading, as blood pressure fluctuates considerably during the day, and even experienced physicians often make errors in taking blood pressure that tend to falsely elevate the patient's readings. Similarly, we would not want to miss the diagnosis of a potentially curable cause of hypertension or of a condition that increases a patient's risk of cardiovascular disease. But considering that nearly one-third of adults in the United States have hypertension and that another one-fourth have prehypertension (formerly called high-normal blood pressure),[1] if we were to launch an intensive workup for every patient with high blood pressure, the cost and effort would be enormous.

Fortunately, for most patients, it is enough to measure blood pressure accurately and repeatedly, perform a focused history and physical examination, and obtain the results of a few basic laboratory tests and an electrocardiogram, with additional tests in special cases.

In this review we address four fundamental questions in the evaluation of patients with a high blood pressure reading, and how to answer them.

ANSWERING FOUR QUESTIONS

The goal of the hypertension evaluation is to answer four questions:

- Does the patient have sustained hypertension? And if so—
- Is the hypertension primary or secondary?
- Does the patient have other cardiovascular risk factors?

*Dr. Townsend has disclosed that he has received grant support from Novartis and the National Institutes of Health; consultant fees from GlaxoSmithKline, NiCox, and Pfizer; and honoraria from BMS and Merck.

TABLE 1

Instructions for taking blood pressure

1. Have the patient relax for at least 5 minutes before taking the blood pressure. Feet should be on the floor, with the back supported.
2. The patient's arm should be supported (ie, resting on a desk) for the measurement.
3. The stethoscope bell, not the diaphragm, should be used for auscultation.
4. Blood pressure should be checked in both arms with the patient sitting. Note which arm gives the higher reading. This arm (the higher arm) should then be used for all other (standing, lying down) future readings.
5. All measurements should be separated by 2 minutes.
6. Measure the blood pressure in the sitting, standing, and lying positions.
7. Use the correct cuff size and note if a larger or smaller than normal cuff size is used.

Blood pressure cuff size criteria

ARM CIRCUMFERENCE	WEIGHT (LB) FEMALE	WEIGHT (LB) MALE	CUFF SIZE TO USE
24–32 cm	< 150	< 200	Regular
32–42 cm*	> 150	> 200	Large
38–50 cm*	–	–	Thigh

*Either cuff size is acceptable in the overlap circumference zone

8. Record systolic (onset of first sound) and diastolic (disappearance of sound) pressures.
9. DO NOT round off results to zeros or fives: record exact results to nearest even number.

A diagnosis of hypertension has an impact on the patient's quality of life

- Does he or she have evidence of target organ damage?

DOES THE PATIENT HAVE SUSTAINED HYPERTENSION?

It is important to measure blood pressure accurately, for several reasons. A diagnosis of hypertension has a measurable impact on the patient's quality of life.[2] Furthermore, we want to avoid undertaking a full evaluation of hypertension if the patient doesn't actually have high blood pressure, ie, systolic blood pressure greater than 140 mm Hg *or* diastolic pressure greater than 90 mm Hg. However, many people have blood pressures in the prehypertensive range (ie, 120–139 mm Hg systolic; 80–89 mm Hg diastolic). Many people in this latter group can expect to develop hypertension in time, as the prevalence of hypertension increases steadily with age unless effective preventive measures are implemented, such as losing weight, exercising regularly, and avoiding excessive consumption of sodium and alcohol.

The steps involved in taking blood pressure are simple (**TABLE 1**)[3,4] but often are not followed in busy clinical practices, and the job is frequently relegated to the least-well-trained staff in the office. The most common errors (failure to have the patient sit quietly for 5 minutes before a reading is taken, lack of arm or foot support, using too small a cuff relative to the size of the arm, deflating the cuff too rapidly) tend to falsely elevate the readings, leading to an overestimate of blood pressure. To reduce the variability commonly noted in staff-obtained manual blood pressure, some of-

fice practices use an automated system such as the BpTRU.[5]

The best position to use is sitting, as the Framingham Heart Study and most randomized clinical trials that established the value of treating hypertension used this position for diagnosis and follow-up.[6]

Proper patient positioning, the correct cuff size, calibrated equipment, and good inflation and deflation technique will yield the best assessment of blood pressure levels. But even if your technique is perfect, blood pressure is a dynamic vital sign, so it is necessary to repeat the measurement, average the values for any particular day, and keep in mind that the pressure is higher (or lower) on some days than on others, so that the running average is more important than individual readings. This leads to two final points about blood pressure measurement:

- Take it right, at least two times on any occasion
- Take it on at least two (preferably three) separate days.

Following up on blood pressure

After measuring the blood pressure, it is necessary to plan for follow-up readings, guided by both the blood pressure levels (TABLE 2) and your clinical judgment.

If the systolic and diastolic blood pressures fall into different categories, you should follow the recommendations for the shorter follow-up time.

TABLE 2

Follow-up recommendations for subsequent blood pressure readings

INITIAL BLOOD PRESSURE (MM HG) SYSTOLIC	DIASTOLIC	FOLLOW-UP RECOMMENDATION
< 120	< 80	Recheck in 2 years
120–139	80-89	Recheck in 1 year
140–159	90-99	Confirm within 2 months
≥ 160	≥ 100	Evaluate within 1 month
		For those with higher pressures (eg, ≥ 180/110 mm Hg), evaluate and treat immediately or within 1 week, depending on clinical situation and complications

ADAPTED FROM CHOBANIAN AV, BAKRIS GL, BLACK HR, ET AL. SEVENTH REPORT OF THE JOINT NATIONAL COMMITTEE ON PREVENTION, DETECTION, EVALUATION, AND TREATMENT OF HIGH BLOOD PRESSURE. HYPERTENSION 2003; 42:1206–1252.

IS THE HYPERTENSION PRIMARY OR SECONDARY?

Most patients with hypertension have primary ("essential") hypertension and are likely to remain hypertensive for life. However, some have secondary hypertension, ie, high blood pressure due to an identifiable cause. Some of these conditions (and the hypertension that they cause) can be cured. For example, pheochromocytoma can be cured if found and removed. Other causes of secondary hypertension, such as parenchymal renal disease, are infrequently cured, and the goal is usually to control the blood pressure with drugs.

The sudden onset of severe hypertension in a patient previously known to have had normal blood pressure raises the suspicion of a secondary form of hypertension, as does the onset of hypertension in a young person (< 25 years) or an older person (> 55 years). However, these ages are arbitrary; with the increasing body mass index in young people, essential hypertension is now more commonly diagnosed in the third decade. And since systolic pressure increases throughout life, we can expect many older patients to develop essential hypertension.[7] Indeed, current guidelines are urging us to pay more attention to systolic pressure than in the past.

Most errors in taking blood pressure falsely elevate the pressure

WHAT IS THE PATIENT'S CARDIOVASCULAR RISK?

The relationship between blood pressure and risk of cardiovascular disease is linear, continuous, and independent of (though additive to) other risk factors.[1] For people 40 to 70 years old, each increment of either 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure doubles the risk of cardiovascular disease across the entire range from 115/75 to 185/115 mm Hg.[1] If the patient smokes or has elevated cholesterol, other cardiovascular risk factors, or the metabolic syndrome, the risk is even higher.[8]

TABLE 3

Some things to ascertain in taking the history in hypertensive patients, and why

Age at onset, duration, and severity
Onset at younger (< 25 years) or older (> 55 years) age suggests secondary hypertension
New-onset, severe hypertension may be secondary

Contributing factors
Significant salt intake, inactivity, psychosocial stress, sleep apnea may contribute to higher blood pressure; some can be addressed separately

Concomitant medications
Common offenders include non-aspirin nonsteroidal anti-inflammatory drugs, oral contraceptives, corticosteroids, licorice, cough/cold/weight-loss sympathomimetics (pseudoephedrine, Ma Huang, ephedrine)

Risk factors for cardiovascular disease
Diabetes, smoking, family history of premature cardiovascular disease, particularly in a first-degree relative (parent or sibling)

Symptoms suggesting secondary causes
Palpitations or tachycardia, spontaneous sweating, migraine-like headaches in paroxysms (catecholamine excess)
Muscle weakness, polyuria (decreased potassium from aldosterone excess)
Personal or family history of renal disease or findings (proteinuria, hematuria) or symptoms such as ankle edema
Thinning of skin and stigmata of cortical excess
Snoring and daytime somnolence (sleep apnea)
Heat intolerance and weight loss (hyperthyroidism)

Target organ damage
Chest pain or chest discomfort (possible coronary artery disease)
Neurologic symptoms consistent with stroke or transient ischemic attack
Dyspnea and easy fatigue (possible heart failure)
Claudication (peripheral arterial disease)

The usual goal of antihypertensive treatment is systolic pressure less than 140 mm Hg *and* diastolic pressure less than 90 mm Hg. However, the target is lower—less than 130/80 mm Hg—for those with diabetes[9] or target organ damage such as heart failure or renal disease.[1,10] Thus, it is important to try to detect these conditions in the evaluation of the hypertensive patient.

Another reason it is important is that reducing such risk sometimes calls for using (or avoiding) antihypertensive drugs that are likely to alter these factors. For example, the use of beta-blockers in patients with a low level of high-density lipoprotein cholesterol (HDL-C) can lower HDL-C further.[11]

DOES THE PATIENT HAVE TARGET ORGAN DAMAGE?

Target organ damage is very important to detect because it changes the goal of treatment from primary prevention of adverse target organ outcomes into the more challenging realm of secondary prevention. For example, if a patient has had a stroke, his or her chance of having another stroke in the next 5 years is about 20%. This is much higher than the risk in an average hypertensive patient without such a history. For such patients, the current guidelines[1] recommend the combination of a diuretic and an angiotensin-converting enzyme inhibitor, a combination shown to reduce the risk of a second stroke.[12] Thus, we need to discover whether the patient had a stroke in the first place.

HISTORY

The history (TABLE 3) helps elucidate whether hypertension is primary or secondary, the degree of cardiovascular risk, and whether target organ damage is present. One should try to ascertain:

TABLE 4

Things to note in the physical examination of hypertensive patients

General appearance, skin lesions, distribution of body fat
Patient may fit criteria for metabolic syndrome (added cardiovascular risk)
Evidence of prior stroke from gait and posture
Rarely, secondary forms are evident as striae (Cushing syndrome) or mucosal fibromas (multiple endocrine neoplasia type II)

Funduscopy
See text for lesion grades
Retinal changes reflect severity of hypertension (target organ damage to the eye) as well as future cardiovascular risk

Examination of neck for thyroid enlargement, carotid bruits
Diffuse multinodular goiter indicating Graves disease
Presence of carotid bruits suggests potential stroke risk

Cardiopulmonary examination
Rales and cardiac gallops consistent with target organ damage (heart enlargement or heart failure)
Interscapular murmur during auscultation of the back (coarctation of the aorta)

Abdominal examination
Palpable kidneys suggest polycystic kidney disease
Mid-epigastric bruits may indicate renal arterial disease

Neurologic examination
Signs of previous stroke (reduced grip, hyperreflexia, spasticity, Babinski sign, muscle atrophy, gait disturbances)

Pulse examination
Delayed or absent femoral pulses may reflect coarctation of the aorta or atherosclerosis

- The duration (if known) and severity of the hypertension
- The degree of blood pressure fluctuation
- Concomitant medical conditions, especially cardiovascular or renal problems
- Dietary habits
- Alcohol consumption
- Tobacco use
- Level of physical activity
- A family history of hypertension, renal disease, cardiovascular problems, or diabetes mellitus
- Past medications, with particular attention to their side effects and their efficacy in controlling blood pressure
- Current medications, including over-the-counter preparations. One reason: nonsteroidal anti-inflammatory drugs other than aspirin can decrease the efficacy of antihypertensive drugs, presumably through mechanisms that inhibit the effects of vasodilatory and natriuretic prostaglandins and potentiate those of angiotensin II.[13]

PHYSICAL EXAMINATION

The physical examination, like the history, give clues about secondary hypertension, cardiovascular risk, and target organ damage (TABLE 4).

The physical examination starts with measurement of height, weight, waist circumference, and blood pressure—in both arms and the leg if coarctation of the aorta is suspected. Measurements with the patient supine, sitting, and standing are usually taken at the first visit, though such an approach is more suited to a hypertension specialty clinic than a primary care setting, in which time constraints usually limit the blood pressure readings to two or three seated values. Most prospective data on the benefits of hypertension treatment are based on a seated blood pressure, so we favor that measurement for follow-up.

Special attention in the physical examination is directed to:

The retina (to assess the vascular impact of the high blood pressure). Look for arteriolar narrowing (grade 1), arteriovenous com-

TABLE 5

Initial laboratory assessment of hypertension

TEST	FINDINGS AND IMPLICATIONS	QUESTIONS ANSWERED		
		PRIMARY VS SECONDARY HYPERTENSION?	CARDIOVASCULAR RISK?	TARGET ORGAN DAMAGE?
Hemoglobin/ hematocrit	Anemia (eg, in kidney disease)			✓
Urinalysis	Detects protein, blood, or glucose	✓	✓	✓
Serum potassium	Hypokalemia may signal aldosterone excess	✓		
Serum creatinine	Increased values signal kidney disease	✓	✓	✓
Blood glucose	Increased values signal diabetes		✓	
Lipid profile	High triglycerides or cholesterol, low high-density lipoprotein cholesterol		✓	
Electrocardiography	Left ventricular hypertrophy; Q waves			✓

pression (grade 2), hemorrhages or exudates (grade 3), and papilledema.[2] Such findings not only relate to severity (higher grade = more severe blood pressure) but also predict future cardiovascular disease.[14]

An S4 is one of the earliest physical findings of hypertension

The blood vessels. Bruits in the neck may indicate carotid stenosis, bruits in the abdomen may indicate renovascular disease, and femoral bruits are a sign of general atherosclerosis. Bruits also signal vascular stenosis and irregularity and may be a clue to vascular damage or future loss of target organ function. However, bruits may simply result from vascular tortuosity, particularly with significant flow in the vessel.

Also check the femoral pulses: poor or delayed femoral pulses are a sign of aortic coarctation. The radial artery is about as far away from the heart as the femoral artery; consequently, when palpating both sites simultaneously the pulse should arrive at about the same moment. In aortic coarctation, a palpable delay in the arrival of the femoral pulse may occur, and an interscapular murmur may be heard during auscultation of the back. In these instances, a low leg blood pressure (usually measured by placing a thigh-sized adult cuff on the patient's thigh and listening over the popliteal area with the patient prone) may confirm the presence of aortic obstruction. When taking a leg blood pressure, the large cuff and the amount of pressure necessary to occlude the artery may be uncomfortable, and one should warn the patient about the discomfort before taking the measurement.

Poor or absent pedal pulses are a sign of peripheral arterial disease.

The heart (to detect gallops, enlargement, or both). Palpation may reveal a displaced apical impulse, which can indicate left ventricular enlargement. A sustained apical impulse may indicate left ventricular hypertrophy. Listen for a fourth heart sound (S4), one of the earliest physical findings of hypertension when physical findings are present. An S4 indicates that the left atrium is working hard to overcome the stiffness of the left ventricle. An S3 indicates an impairment in left ventricular function and is usually a harbinger of underlying heart disease. In some cases, lung rales can also be heard, though the combination of an S3 gallop and rales is an unusual office presentation in the early management of the hypertensive patient.

The lungs. Listen for rales (see above).

The lower extremities should be examined for peripheral arterial pulsations and edema. The loss of pedal pulses is a common finding, particularly in smokers, and is a clue to increased cardiovascular risk.

Strength, gait, and cognition. Perform a brief neurologic examination for evidence of

remote stroke. We usually observe our patients' gait as they enter or leave the examination room, test their bilateral grip strength, and assess their judgment, speech, and memory during the history and physical examination.

A great deal of research has linked high blood pressure to future loss of cognitive function,[15] and it is useful to know that impairment is present before beginning treatment, since some patients will complain of memory loss after starting antihypertensive drug treatment.

LABORATORY EVALUATION

Routine tests

The routine evaluation of hypertensive patients should include, at a minimum:

- A hemoglobin or hematocrit measurement
- Urinalysis with microscopic examination
- Serum electrolyte concentrations
- Serum creatinine concentrations
- Serum glucose concentration
- A fasting lipid profile
- A 12-lead electrocardiogram (TABLE 5).

Nonroutine tests

In some cases, other studies may be appropriate, depending on the clinical situation, eg:

- **Serum uric acid** in those with a history of gout, since some antihypertensive drugs (eg, diuretics) may increase serum uric acid and predispose to further episodes of gout
- **Serum calcium** in those with a personal or family history of kidney stones, to detect subtle parathyroid excess
- **Thyroid-stimulating hormone** or other thyroid studies if the history suggests thyroid excess, or if a thyroid nodule is discovered
- **Limited echocardiography,** which is more sensitive than electrocardiography for detecting left ventricular hypertrophy.

We sometimes use echocardiography if the patient is overweight but seems motivated to lose weight. In these cases we might not start drug therapy right away, choosing rather to wait and see if the patient can lose some weight (which might lower the blood pressure and make drug therapy unnecessary)—but only if the echocardiogram shows that he or she does not have left ventricular hypertrophy.

We also use echocardiography in patients with white-coat hypertension (see below), in whom office pressures are consistently high but whom we have elected to either not treat or not alter treatment. In these cases the echocardiogram serves as a "second opinion" about the merits of not altering therapy and supports this decision when the left ventricular wall thicknesses are normal (and remain so during long-term follow-up). In cases of suspected white-coat hypertension, home or ambulatory blood pressure monitoring is valuable to establish or exclude this diagnosis.[1]

Urinary albumin excretion. Microalbuminuria is an early manifestation of diabetic nephropathy and hypertension. Although routine urine screening for microalbuminuria is typically done in the management of diabetes, it is still not considered a standard of care, though the growing literature on its role as a cardiovascular risk predictor[16–18] and its value as a therapeutic target in diabetes[19,20] make it an attractive aid in the overall assessment of patients with hypertension.

Plasma renin activity and serum aldosterone concentrations are useful in screening for aldosterone excess, but are usually reserved as follow-up tests in patients with either hypokalemia or failure to achieve blood pressure control on a three-drug regimen in which at least one drug is a diuretic.[1,21]

Of note, primary aldosteronism is not as rare as previously thought. In a study of patients referred to hypertension centers, 11% had primary aldosteronism according to prospective diagnostic criteria, almost 5% had curable aldosterone-producing adenomas, and 6% had idiopathic hyperaldosteronism.[22]

Delayed femoral pulses are a sign of coarctation of the aorta

If secondary hypertension is suspected

Sometimes the history, examination, or initial testing leads one to suspect that a secondary form of hypertension may be present. TABLE 6 lists some of the common ways to pursue such suspicions. Readers are referred to several excellent reviews of secondary hypertension for further details.[23–25]

A search for secondary forms of hypertension is usually considered in patients with moderate or severe hypertension that does

TABLE 6

Suggested approaches to pursuing possible secondary hypertension

Coarctation of the aorta
Chest film (rib notching; reverse "3" sign)
Two-dimensional echocardiography
Aortography (coarctation directly seen)
Magnetic resonance imaging (MRI)

Cushing syndrome
Dexamethasone suppression test (failure to suppress cortisol)
24-hour urinary free cortisol (elevated)
Computed tomography (CT) (adrenomegaly)

Primary aldosteronism
Plasma aldosterone-to-renin ratio (increased)
Aldosterone excretion rate during salt loading (increased)
Adrenal CT (adenoma with low Hounsfield units)

Pheochromocytoma
Plasma catecholamines or metanephrines (increased)
Urine catecholamines or metanephrines (increased)
Clonidine suppression test (failure to suppress plasma norepinephrine after clonidine administration)
Adrenal CT, MRI (adrenal tumor; T2-weighted MRI has characteristic appearance)
Iodine 131 metaiodobenzylguanidine scan (significant adrenal or extra-adrenal tumor uptake)

Renal vascular disease
Captopril renography (some limitations)
Renal duplex sonography (requires good operators; higher velocity in the renal artery than in the aorta velocities suggests stenosis)
Magnetic resonance angiography (renal vessel narrowing)
CT angiography (renal vessel narrowing)
Angiography (gold standard; renal vessel narrowing)
Renal vein renin ratio (not commonly done)

Renal parenchymal disease
24-hour urine protein and creatinine levels
Renal ultrasonography (small kidney size, unusual architecture)
Glomerular filtration rate (low)
Renal biopsy (usually done to determine type of glomerular disease)
Serum thyroid hormone level (increased in hyperthyroidism)
Serum calcitonin level (when multiple endocrine neoplasia is suspected)

Hyperparathyroidism
Calcium and phosphorus levels (increased and decreased, respectively)
Serum parathyroid hormone level (increased)

Thyroid disease
Thyrotropin level (suppressed in hyperthyroidism)

not respond to antihypertensive agents. Another situation is in hypertensive patients younger than 25 years, since curable forms of hypertension are more common in this age group. In older patients, the prevalence of secondary hypertension is lower and does not justify the costs and effort of routine elaborate workups unless there is evidence from the history, physical examination, or routine laboratory work for suspecting its presence. An exception to this rule is the need to exclude atherosclerotic renovascular hypertension in an elderly patient. This cause of secondary hypertension is common in the elderly and may be amenable to therapeutic intervention.[26]

WHEN TO CONSIDER HOME OR AMBULATORY MONITORING

Most patients with hypertension do not need ambulatory blood pressure monitoring, but in selected cases (TABLE 7), it may help in clinical management. However, Medicare and Medicaid pay for it only for the specific indication of white-coat hypertension. Readers are referred to a recent excellent review for further information.[27]

Suspected white-coat hypertension

Blood pressure can be influenced by an environment such as an office or hospital clinic. This has led to the development of ambulatory blood pressure monitors and more use of self-measurement of blood pressure in the home. Blood pressure readings with these techniques are generally lower than those measured in an office or hospital clinic. These methods make it possible to screen for white-coat hypertension. In 10% to 20% of people with hypertensive readings, the blood pressure may be elevated persistently only in the presence of a physician.[28] When measured elsewhere, including at work, the blood pressure is not elevated in those with the white-coat effect. Although this response may become less prominent with repeated measurements, it occasionally persists in the office setting, sometimes for years in our experience.

Suspected nocturnal hypertension ('nondipping' status)

Ambulatory blood pressure is also helpful to screen for nocturnal hypertension. Evidence

is accumulating to suggest that hypertensive patients whose pressure remains relatively high at night ("nondippers," ie, those with less than a 10% reduction at night compared with daytime blood pressure readings) are at greater risk of cardiovascular morbidity than "dippers" (those whose blood pressure is at least 10% lower at night than during the day).[29]

An early morning surge

Ambulatory monitoring can also detect morning surges in systolic blood pressure,[30] a marker of cerebrovascular risk. Generally, these patients have an increase of more than 55 mm Hg in systolic pressure between their sleeping and early-hour waking values, and we may wish to start or alter treatment specifically to address these high morning systolic values.[31]

TABLE 7

Potential indications for ambulatory blood pressure monitoring

Unusual variability of blood pressure
Possible white-coat hypertension
Nocturnal hypertension
Drug-resistant hypertension
Determining the efficacy of drug treatment over 24 hours
Hypertension in pregnancy
Symptomatic hypotension on various medications, suggesting that the patient may be normotensive
Episodic hypertension or autonomic dysfunction

'PIPESTEM' VESSELS AND PSEUDOHYPERTENSION

Occasionally, one encounters patients with vessels that are stiff and difficult to compress. If the pressure required to compress the brachial artery and stop audible blood flow with a standard blood pressure cuff is greater than the actual blood pressure within the artery as measured invasively, the condition is called pseudohypertension. The stiffness is thought to be due to calcification of the arterial wall.

A way to check for this condition is to inflate the cuff to at least 30 mm Hg above the palpable systolic pressure and then try to "roll" the brachial or radial artery underneath your fingertips, a procedure known as Osler's maneuver.[32] If you feel something that resembles a stiff tube reminiscent of the stem of a tobacco smoker's pipe (healthy arteries are not palpable when empty), the patient may have pseudohypertension. However, the specificity of Osler's maneuver has been questioned, particularly in hospitalized elderly patients.[33]

Pseudohypertension is important because the patients in whom it occurs, usually the elderly or the chronically ill (with diabetes or chronic kidney disease), are prone to orthostatic or postural hypotension, which may be aggravated by increasing their antihypertensive treatment on the basis of a cuff pressure that is actually much higher than the real blood pressure.[33]

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***ADDRESS**: Raymond R. Townsend, MD, Department of Medicine, 122 Founders Building, University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104; e-mail townsend@mail.med.upenn.edu.*