## HYPERTENSION \& PREHYPERTENSION

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Note: This care pathway is based on key source material rather than a complete literature search.
UWS care pathways and protocols provide evidence-informed, consensus-based guidelines to support clinical decision making. To best meet a patient's healthcare needs, variation from these guidelines may be appropriate based on more current information, clinical judgment of the practitioner, and/or patient preferences.

These pathways and protocols are informed by currently available evidence and developed by UWS personnel to guide clinical education and practice. Although individual procedures and decision points within the pathway may have established validity and/or reliability, the pathway as a whole has not been rigorously tested and therefore should not be adopted wholesale for broader use.

## ICD-9 Codes

796.2 elevated blood pressure without a diagnosis of hypertension
796.2 same code is used for pre-hypertension
401.0-9 hypertension
405.0 secondary hypertension

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## BACKGROUND

Updates of this care pathway have been based primarily on the following documents:

- Joint National Committee (JNC8) ${ }^{1}$ which focused only on pharmaceutical interventions without updating the rest of JNC 7 and supporting the lifestyle recommendations contained in AHA/ACC
- American Society of Hypertension-International Society of Hypertension (ASH-ISH) ${ }^{2}$
- The European HTN Guidelines (ESH-ESC) ${ }^{3}$
- American College of Cardiology/American Heart Association Task Force (AHA/ACC) ${ }^{4}$ which focused on lifestyle management to reduce cardiovascular risk and included recommendations specific for HTN.

Hypertension (HTN) is defined as systolic blood pressure (SBP) of 140 mmHg or greater, a diastolic blood pressure (DBP) of 90 mmHg or greater, or taking antihypertensive medication. In approximately $95 \%$ of hypertension cases the specific cause is unknown (essential hypertension or primary hypertension). High blood pressure appears to be the result of the interaction of several genes and is influenced by multiple environmental factors.

Hypertension that is secondary to other disease processes is estimated to be around $5 \%$ but there are no population data available reflecting the true prevalence. Identifiable causes of hypertension include sleep apnea, drug-induced or drug-related, chronic kidney disease, primary aldosteronism, renovascular

[^0]disease, chronic steroid therapy, Cushing syndrome, pheochromocytoma, coarctation of the aorta, and thyroid or parathyroid disease.

## Epidemiology

Recent statistics suggest that as many as 1 out of 3 adult Americans have hypertension. (Fields 2004) Nearly three-fourths of adult Americans with diagnosed hypertension are not controlling their blood pressure to be below 140/90 mmHg. Approximately $60 \%$ meet the criteria for stage 1 HTN.
According to the ASH-ISH guidelines (2013) there is a close relationship between blood pressure levels and the risk of strokes, cardiovascular and renal disease. "The risk of these outcomes is lowest at a blood pressure of around $115 / 75 \mathrm{~mm} \mathrm{Hg}$. Above $115 / 75 \mathrm{~mm} \mathrm{Hg}$, for each increase of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure, the risk of major cardiovascular and stroke events doubles."

The risk of cardiovascular related events is especially linked to systolic HTN. After the age of 50 or 60, diastolic pressure may actually start to drop while systolic pressure continues to rise. Individuals who are normotensive at 55 years of age have a $90 \%$ lifetime risk for developing hypertension.

The majority of persons with isolated systolic hypertension are not adequately controlling their blood pressure despite persuasive data from clinical trials documenting the benefit of treatment.

Approximately one of every six healthy adults without pre-existing heart disease, diabetes, peripheral artery disease or stroke, are still at intermediate to high risk of developing heart disease in the next 10 years. (Ford 2004) In the African-American population, hypertension is more common, occurs at a younger age, is more severe, and more often leads to strokes and severe kidney disease. (ASH-ISH 2013)

# EVALUATION 

## Evaluation Strategy

## SUMMARY of Evaluation Steps

Step 1. Establish if a diagnosis of hypertension or pre-hypertension is warranted.
Step 2. Determine the stage of hypertension.
Step 3. Evaluate patient for causes, CVD risk factors and end organ damage.
Step 4. Assess the patient's overall risk.

## STEP 1. Establish if a diagnosis of hypertension or pre-hypertension is warranted.

All new patients, all patients with a history of elevated blood pressure, and all returning patients who have not had their blood pressure taken within a year should be screened.

When taking blood pressure on a new patient for the first time, it is recommended to take a palpatory blood pressure first in one arm and then actual measures should be done bilaterally.

If blood pressure is above 120/80, repeat on the same visit. The classification of hypertension is based on the average of two (or more) properly measured seated blood pressure readings taken at least 1-2 minutes apart on each of two (or more) separate occasions.

Note that acute pain can temporarily raise blood pressure. In some cases, the practitioner will need blood pressure readings spread over more than 2 visits to differentiate true hypertension from a limited pain response.

Systolic readings greater than 180 and diastolic above 110 do not require serial confirmation before making decisions regarding intervention.

## Diagnosis of HTN = 2 readings $X 2$ visits

 (unless > 180 SBP or 110 DBP)*Proper Technique**

Since management recommendations will be made even for patients with pre-hypertension, it is important that proper technique is used to measure pressure as accurately as possible. (See Appendix 1.)

- Patients should be seated in a chair, feet flat (legs uncrossed) with their backs supported and their arms bared and supported at heart level.
- Patients should have empty bladders and refrain from smoking or ingesting caffeine during the 30 minutes preceding the measurement.
- Measurement should begin after at least 5 minutes of rest.
- The appropriate cuff size must be used to ensure accurate measurement. The bladder within the cuff should encircle at least $80 \%$ of the arm. Many adults will require a large adult cuff.
- Measurements can be with a variety of instruments: a mercury sphygmomanometer, a recently calibrated aneroid manometer, or a validated electronic device. Departing from other guidelines, ASH-ISH (2013) recommends using an electronic device which, if available, "is preferred because it provides more reproducible results than the older method and is not influenced by variations in technique or by the bias of

[^1]the observers." Automated blood pressure readings have been reported to be approximately 5-6/2-3 points lower than measurements using the traditional method. (Myers, 2011, Nelson 2009)

- It is recommended that a palpatory blood pressure be first taken to help detect the presence of any auscultatory gap. There is evidence to suggest that patients with auscultatory gaps have an increased risk for cardiovascular disease. Palpate the brachial or radial artery and pump up the cuff until the pulse disappears, then continue pumping another 30 mmHg before starting to release the air and proceed to take an auscultatory reading.
- The descent rate of the needle or mercury column should be no more than about 2-3 mmHg per second.
- Although the diaphragm of the stethoscope should make tight contact with the skin, care should be taken so that excess pressure is not applied.
- Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) should be recorded. The first appearance of sound (phase 1) is used to define SBP. The disappearance of sound (phase 5 ) is used to define DBP.


## Bilateral measurements

Blood pressure should be taken in both arms on the first visit; the arm with the higher reading is then used for follow up measurements if necessary. (ESH-ESC 2013)

While discrepancies > 10 mm Hg represent a higher cardiovascular risk factor, differences $>20 \mathrm{~mm}$, if confirmed, suggest possible vascular abnormalities (e.g., subclavian steal syndrome, peripheral artery disease). However, unless the readings are taken simultaneously, the difference may simply be normal variation in blood pressure. (ESH-ESC 2013). These readings should be repeated to confirm the discrepancy.

## StANDING MEASUREMENTS

Patients who have a history of dizziness, light headedness, syncope or pre-syncopal symptoms with transitional movements should be screened for orthostatic hypotension. Optionally, the practitioner can also choose to screen elderly and diabetics patients in general to see if this is a problem. The blood pressure is taken seated and then measured again at 1 then 3 minutes after standing (orthostatic hypotension is detected by a drop in SBP $\geq$ $20, \mathrm{DBP} \geq 10$ ). The presence of orthostatic hypotension may be a result from simple dehydration but may require a more extensive work up. (ESH-ESC 2013)

## INTERPRETATION AND FOLLOW UP

Systolic blood pressure $\geq 140 \mathrm{~mm} \mathrm{Hg}$ or the diastolic pressure $\geq 90 \mathrm{~mm} \mathrm{Hg}$, or both, measured on at least two separate visits are necessary to confirm a diagnosis of hypertension. The second visit should usually be no longer than 1 to 4 weeks after the first measurement. (AS-ISH 2013)

When pressure decreases from the first to the second evaluation, it may be worthwhile obtaining several additional measurements to prevent misdiagnosis.

## Recommendations for Follow-up Based on Initial Blood Pressure Measurements for Adults

| Systolic | Diastolic | Follow-up Recommended |
| :---: | :---: | :--- |
| $<120$ | $<80$ | Recheck in 1-2 years. |
| $120-139$ | $80-89$ | Confirm on subsequent visits <br> (within 1 year) |
| $140-159$ | $90-99$ | Confirm on subsequent visits <br> (within 2 months) <br> Consider home monitoring |
| $160-179$ | $100-109$ | Confirm on subsequent visits. <br> (within 1 month) <br> Consider home monitoring |
| $>180$ | $>110$ | Refer to source of care <br> immediately or within 1 week <br> depending on clinical <br> circumstances. |

Timelines for follow-up should be further modified according to reliable information about past blood pressure measurements,
other cardiovascular risk factors, or the presence of target organ disease.

## Home Measurements

Self-recorded blood pressure measurements may be recommended. ASH-ISH (21013) states that "it can be helpful to measure blood pressures at home. If available, the electronic device is simpler to use and is probably more reliable than the sphygmomanometer."

Patients who are able to do home measures should be given written instructions to improve the quality of the measures. (See Appendix 2.)

The patient should take a morning and an evening reading, preferably at the same time every day for 5-7 days. Patients should be given the following instructions:
(ESH-ESC 2013):

- Take your blood pressure in a quiet room, seated with your back and arm supported after resting for 5 minutes.
- Take your pressure a second time (1-2 minutes apart).
- Write down the numbers and the time of day in a log book.

The values are averaged and it is recommended that averages above 135 mmHg systolic or $\underline{85} \mathrm{mmHg}$ diastolic may be considered hypertensive. (These are the same values recommended for ambulatory readings, see below.)

Home blood pressure monitoring, using devices that incorporate inflatable cuffs, is associated with improved control of hypertension than blood pressure monitoring in the health care system.

NOTE: "Home BP is more closely related to [i.e. correlated with] hypertension-induced OD [organ damage] than office BP, particularly LVH [left ventricular hypertrophy], and recent metaanalyses of the few prospective studies in the general population, in primary care and in hypertensive patients, indicate that the prediction of CV morbidity and mortality is significantly better with home BP than with office BP." (ESH-ESC 2013)

Night-time readings may be particularly important. Roush (2014) reported that for every 10 mm Hg increase in SBP, the risk of MI and stroke rose $25 \%$.

Home measurement devices should be checked regularly for accuracy.

Cuffs that fit on the finger or wrist are often inaccurate and generally not recommended. (ASH-ISH)

## Ambulatory Blood Pressure Measurements

It is well established that ambulatory blood pressure correlates more closely than clinical blood pressure with a variety of measures of target organ damage, such as left ventricular hypertrophy (LVH). Prospective data suggest that in patients in whom an elevated clinic pressure is the only abnormality ambulatory monitoring may identify a group at relatively low risk of morbidity.

Ambulatory blood pressure monitoring requires substantial investment in equipment and personnel as well as special training. Monitors are programmed to take readings every 15 to 30 minutes as the patient goes about his or her daily activities and are then downloaded onto a computer. Ambulatory blood pressure is not commonly done.

## STEP 2. Determine the stage of hypertension.

NOTE: When SBP and DBP fall into different categories, the higher category should be selected to classify the individual's blood pressure.

The categories in the table below are based on the patient not taking anti-hypertensive drugs and not being acutely ill.

## Blood Pressure, mmHg

| Category | Systolic | Diastolic |  |
| :--- | :--- | :--- | :--- |
| Normal | $<120$ | and | $<80$ |
| Pre-Hypertension | $120-139$ or | $80-89$ |  |
| Hypertension | Stage 1 <br> Stage 2 |  |  | | $140-159$ | or | $90-99$ |  |
| :--- | :--- | :--- | :--- |
|  | $\geq 160$ | or | $\geq 100$ |

Optimal blood pressure with respect to cardiovascular risk is less than 120/80 mmHg . However, unusually low readings should be evaluated for clinical significance.

Isolated systolic hypertension is defined as systolic blood pressure 140 mmHg or greater and diastolic blood pressure less than 90 mmHg and staged appropriately (e.g., 170/82 mmHg is defined as stage 2 isolated systolic hypertension). In patients older than 50 years, systolic blood pressure greater than 140 mmHg is a much more important cardiovascular disease risk factor than diastolic blood pressure.

## STEP 3. Evaluate patient for causes, CVD risk factors and end organ damage.

Once a diagnosis of hypertension has been confirmed and staged, the patient should be assessed with three objectives in mind:

1) identifying possible causes of secondary hypertension (uncommon, about 5\% of cases);
2) identifying possible target organ damage (TOD) as a result of the hypertension; and
3) identifying the presence of cardiovascular disease (CVD), other risk factors for CVD and concomitant disorders (such as diabetes).

## If the practitioner and patient elect for a referral for medical assessment/drug therapy, many of these procedures can be done by the co-manager.

## 1. Medical History

It is recommended that specific history information be elicited from patients with hypertension. The history should include the following:

- known duration and prior levels of elevated blood pressure
- symptoms including angina, shortness of breath (CHD or CHF); swelling in the lower extremity, orthopnea (CHF); dizziness, fainting, balance problems, confusion (CHD or stoke); polyuria, polydipsia, polyphagia, polyneuritis (diabetes); heat intolerance, weight loss, tremor (hyperthyroidism); history of recent, unexplained changes in weight
- symptoms suggesting a cause of hypertension (see Secondary Hypertension below)
- personal history of sleep apnea, coronary heart disease (CHD) (including MI, angina, coronary revascularizations), congestive heart failure (CHF), cerebrovascular disease (including stroke, TIAs, dementia), peripheral vascular disease, chronic renal disease, diabetes mellitus, dyslipidemia, gout, sexual dysfunction, hyperthyroidism, other concomitant conditions
- family history (especially first degree relatives) of high blood pressure, premature CHD, stroke, diabetes, dyslipidemia, or renal disease
- lifestyle including leisure-time physical activity levels; smoking or other tobacco use; dietary assessment including intake of sodium, alcohol, saturated fat, and caffeine
- medications including history of all prescribed and over-the-counter medications (especially hypertensive medications and those drugs that can elevate blood pressure or interfere with hypertensive medication). See Secondary Hypertension.
- psychosocial and environmental factors that may influence hypertension control (e.g., family situation, employment status, working conditions, and educational level).


## Secondary Hypertension

Historical findings suggesting identifiable causes of hypertension (secondary) include both nonspecific and specific clues.

## Nonspecific clues

- A sudden increase in blood pressure which had been previously normal or previously controlled by antihypertensive therapy.
- A gradual increase in pressure over a year in previously normal middle-aged person without changes in medication, weight, alcohol intake, or salt intake.
- Hypertension in young people (aged <40 years) and those with a family history of hypertension or stroke at age $<50$ years. (Hammer 2009)


## Specific clues

- A history of persistent urinary tract infections may suggest pyelonephritis.
- Labile hypertension or paroxysms of hypertension with headache, palpitations, pallor, and perspiration suggest pheochromocytoma.
- Medications that may cause hypertension (Viera 2010):
o older high dose oral contraceptives
o herbals: ephedra (ma huang), ginseng
o amphetamines, cocaine
o NSAIDS: Cyclooxygenase-2 inhibitors, ibuprofen, naproxen (Naprosyn)
o psychiatric: Buspirone (Buspar), carbamazepine (Tegretol), clozapine (Clozaril), fluoxetine (Prozac), lithium, tricyclic antidepressants
o steroids: methylprednisolone (DepoMedrol), prednisone
o decongestants
o diet pills
o thyroid medications,
o migraine medications,
- Alcohol and other recreational drugs (e.g., cocaine) and "performance enhancing" supplements.


## Causes of secondary HTN (Viera 2010)

| Age | Most common diseases |
| :--- | :--- |
| $\mathbf{1 9}$ to 39 | Thyroid dysfunction <br> Fibromuscular dysplasia <br> Renal disease |
| $\mathbf{4 0}$ to 64 | Aldosteronism <br> Thyroid dysfunction <br> Obstructive sleep <br> apnea <br> Cushing syndrome |
| $\geq 65$ | Pheochromocytoma <br> Atherosclerotic renal <br> artery stenosis <br> Renal failure <br> Hypothyroidism |

Special tests necessary to work up possible secondary hypertension are listed in Appendix 3.

## Pregnancy

Elevated blood pressure occurring during pregnancy can signal pre-eclampsia when combined with other symptoms such as edema (especially of hands, feet, and face leading to > 4lb weight gain in one week), headache, visual disturbances, or abdominal pain (URQ/epigastric).

Blood pressure > 140/90 or an increase >30 systolic or >15 diastolic in women with preexisting HTN on repeated measures is significant. Sometimes a random rise may occur due to physical exertion, caffeine consumption or stress that then returns to normal on the repeat measures.

This condition is most common in the $3^{\text {rd }}$ trimester, but the onset can range from 6 to 20 weeks post-partum. It may be mild or progress to life-threatening. The patient's
obstetrician or primary provider for the pregnancy should be notified immediately.

## 2. Physical Examination

It is recommended that specific physical findings be assessed for patients with hypertension. The assessment should focus on causes of secondary HTN and possible end organ damage.

## SUMMARY of Procedures

- Take blood pressure (see page 4 for details).
- Measure height, weight, waist circumference; calculate BMI (weight in kilograms divided by the square of the height in meters).
- Examine fundus (optional)
- Auscultate heart and major vessels.
- Palpate chest wall for thrills, heaves, heart size.
- Check peripheral pulses (and for edema).
- Palpate thyroid gland.
- Auscultate lungs.
- Exam abdomen for bruit, masses and enlarged abdominal aorta.
- Perform neurological screen.


## Measurement of height, weight and WAIST/HIP CIRCUMFERENCE

Based on height and weight, the body mass index BMI can be calculated. (See http://www.nhlbi.nih.gov/guidelines/obesity/ BMI/bmicalc.htm) (ASH-ISH 2013)
The deposition of excess fat in the upper part of the body (visceral or abdominal), as evidenced by a waist circumference of $>102$ cm (40 inches) in men or $>88 \mathrm{~cm}$ ( 35 inches) in women also has been associated with the risk for metabolic syndrome, hypertension, dyslipidemia, diabetes and CHD mortality. (ASH-ISH 2013). Circumferential measurements are done at the level of the umbilicus and greater trochanters with the tape measure parallel to the floor, making sure that the patient is not holding his/her breath. Waist to hip ratio should be less than 1:1 for men; less than 0.8:1 for women.

## Fundoscopic examination for hYPERTENSIVE RETINOPATHY

ASH-ISH (2013) recommends "If possible, the optic fundi should be checked for hypertensive or diabetic changes and the areas around the eyes for findings such as xanthomas." On the other hand, ESH-ESC (2013) does not recommend fundoscopic examinations in mild-moderate hypertensive patients unless they have diabetes, are resistant to therapy, or are young, based on consensus of expert opinion. Patients with diabetes require a complete fundoscopic assessment with dilated pupils. (Saudek, 2012)

Findings classically associated with stage I changes (arteriolar narrowing, focal arteriolar constrictions) and stage II changes (arteriolar sclerosis, arteriovenous crossing changes) have been considered unreliable indicators of hypertensive retinopathy. However, recent findings suggest that retinal arteriolar narrowing predicts a 5-year incidence of severe hypertension.

Stage III changes (hemorrhages, exudates, areas of retinal edema) and stage IV changes (disc edema, central retinal vein or artery occlusion) are considered very reliable indicators of hypertensive retinopathy.

## CARDIOVASCULAR EXAM

A number of exam procedures should be performed to assess the health of the cardiovascular system:

- Check for distended jugular veins (suggesting CHF).
- Auscultate the neck for carotid bruits and examine for distended veins.
- Check heart rate (resting heart rate values correspond with risk for CV events). (ESHESC 2013).
- Check peripheral pulses in lower extremity (absence of one or more pulses in the extremities, except the dorsal pedis pulse which is often absent in the general population, can suggest peripheral vascular disease with or without the presence of intermittent claudication).
- Check the heart for abnormalities: increased size (shifted PMI) or precordial heave.
- Auscultate heart (clicks, murmurs, and third and fourth heart sounds).
- Examine the extremities for edema (suggesting CHF).


## Thyroid exam

Check for an enlarged thyroid gland or other signs of thyroid disease.

## LUNG EXAM

Examine lungs for rales (CHF) and evidence of bronchospasm.

## Abdominal exam

Examine the abdomen for bruits (especially renal, suggesting a cause of secondary HTN), enlarged liver (CHF), enlarged kidneys, masses, and abnormal aortic pulsation.

## NeUROLOGIC EXAMINATION

A brief neurological screen should be performed to detect any sequelae resulting from prior undiagnosed strokes. (ASH-ISH). Check some or all of the following: visual fields, cardinal fields of gaze (especially horizontal), cranial nerve VII (facial expression), pronator drift (10 seconds), finger to nose, heel-shin tests, gait, stretch reflexes (i.e., DTRs) and pathological reflexes in the upper and lower extremities (recommendations based partly on the NIH stroke scale).

## Secondary Hypertension

Physical exam findings suggesting causes of secondary hypertension include the following:

- abdominal bruits (especially if they lateralize to the renal area) suggest renovascular disease
- an abdominal or flank mass may be polycystic disease
- delayed (compared to right radial) or absent femoral artery pulses and decreased blood
pressure in the lower extremities
(accompanied by rib notching on x-ray) may indicate aortic coarctation,
- purple striae and truncal obesity suggest

Cushings syndrome.

## 3. Ancillary Studies

## Routine tests

Patients with newly diagnosed HTN should undergo routine laboratory testing (including an ECG), primarily to identify any indicators of asymptomatic end organ damage. ESH-ESC (2013) reports that the presence of microalbuminuria, increased pulse wave velocity, left ventricular hypertrophy (LVH), or carotid plaquing carries increased risk for CV mortality independent of commonly used risk factor scoring cards.

Because this damage may currently be asymptomatic, the practitioner cannot base the decision to order these tests on the presence or absence of findings from the history or physical. Identifying asymptomatic end organ damage is especially important if a therapeutic trial of non-pharmacological interventions is going to be initiated. The presence of end organ damage can have a significant effect on whether and when referral for drug therapy is indicated. In the case of patients already on drug therapy, these tests are usually done at the discretion of the primary medical provider, and the results can be requisitioned.

Most of the recommended tests can be captured by a standard metabolic panel, CBC and dipstick UA. Note that medications the patient is on may interfere with test values. (See Appendix 4.)

## TEST SUMMARY

## Routine tests

- Hemoglobin and/or Hematocrit.
- Fasting plasma glucose.
- Serum total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol.
- Fasting serum triglycerides.
- Serum potassium and sodium.
- Serum uric acid.
- Serum creatinine (with estimation of GFR).
- Urine analysis: microscopic examination; urinary protein by dipstick test; test for microalbuminuria.

| - $\quad$ 12-lead ECG. |
| :--- |
| Additional tests, based on history, <br> physical examination, and findings <br> from routine laboratory tests |
| - Hemoglobin $\mathrm{A}_{1 \text { c }}$ if fasting plasma glucose <br> is >5.6 mmol/L (102 mg/DL) or previous <br> diagnosis of diabetes |
| - Quantitative proteinuria if dipstick test <br> is positive; urinary potassium and <br> sodium concentration and their ratio |
| - Home and 24-h ambulatory BP |
| monitoring |
| - Echocardiogram |
| - Holter monitoring in case of arrhythmias |
| - Carotid ultrasound |
| - Peripheral artery/abdominal ultrasound |
| - Pulse wave velocity |
| - Ankle-brachial index |
| - Fundoscopy |
| Extended evaluation (mostly domain <br> of the specialist) |
| - Further search for cerebral, cardiac, |
| renal, and vascular damage, mandatory |
| in resistant and complicated |
| hypertension |
| - Search for secondary hypertension when |
| suggested by history, physical exam- |
| ination, or routine and additional tests |

ASH-ISH (2013) recommends the following ancillary studies:

## Metabolic Panel

o Electrolytes. Special emphasis on potassium: high levels, especially if combined with elevated creatinine, suggest renal disease. Low values suggest aldosterone excess.
o Fasting glucose concentration (>126 indicates diabetes, > 100 pre-diabetes). If elevated, consider ordering Hgb A1C.
o Serum creatinine and blood urea nitrogen. Increased creatinine levels can indicate kidney disease; creatinine is also used to calculate eGFR. (Strong recommendation, level B evidence, ESHESC)
o Lipids (including total cholesterol, HDL, LDL, and triglycerides). For interpretation of lipid profiles, see Appendix 5.
o Liver function tests. These can be used to establish useful baselines if drug therapy is initiated.

## CBC

Hemoglobin/hematocrit. Anemia may be associated with chronic kidney disease.

## URINALYSIS

Proteins. The presence of albuminuria can indicate renal disease and is also associated with an increased risk of cardiovascular events. "Ideally, an albumin/creatinine ratio should be obtained, but even dipstick evidence of albuminuria ( +1 or greater) is helpful." (ASHISH) (Strong recommendation, level $B$ evidence ESH-ESC)

## Electrochardiography

ECG can help identify evidence of previously undetected target organ damage such as silent myocardial infarctions or left atrial and ventricular hypertrophy, increasing the importance for good BP control in a timely manner. ECG might also identify cardiac arrhythmias such as atrial fibrillation (which would indicate the use of certain drugs) or conditions such as heart block (which would contraindicate certain drugs, e.g., B-blockers, rate-slowing calcium channel blockers). ESHESC makes a strong recommendation that a 12-lead ECG "should be part of the routine assessment" of HTN patients" based on level B evidence.

[^2]
## Renal imaging

In the case of suspected secondary hypertension in a young adult, renal artery stenosis should be assessed by magnetic resonance angiography (sensitivity and specificity $>90 \%$ ). Indications include presence of a renal artery bruit or if laboratory screening tests for primary hyperaldosteronism and pheochromocytoma are negative. (Hammer 2009).

## Optional tests

- Creatinine clearance
- 24-hour urinary protein
- Serum calcium
- Uric acid
- Glycosylated hemoglobin ( $\left.\mathrm{HgbA}_{1} \mathrm{C}\right)$
- TSH

See Appendix 3 for further description of routine and optional tests.

## In Selected patients

Echocardiography, if available, is more accurate than an ECG for detecting and diagnosing left ventricular hypertrophy (LVH) a key indicator of target organ damage. It can also be used to quantify the ejection fraction in patients with suspected heart failure. This test, however, is not ordered routinely. (ASH ISH 2013)

Other optional tests include examination of structural alterations in arteries by ultrasonography, measurement of ankle/arm index, and assessment of plasma renin activity/urinary sodium. Patients with cardiac or other serious health problems need a more thorough evaluation, often including a cardiac stress test, referral to a specialist, or medically supervised exercise program.

## LABORATORY FINDINGS SUGGESTING IDENTIFIABLE CAUSES OF SECONDARY HYPERTENSION INCLUDE:

- Unprovoked hypokalemia (initial serum K < $3.5 \mathrm{mEq} / \mathrm{L}$ ) on no medication suggests primary aldosteronism.
- Hypercalcemia suggests possible hyperparathyroidism.
- Elevated creatinine levels or abnormal urinalysis suggests renal disease.

NOTE: If the patient is on drug therapy for HTN, be aware of its effect on any subsequent lab work ordered (see Appendix 6).

## STEP 4. Assess the patient's overall risk.

Group A - No major risks.
Group B - No end organ damage. This includes no heart disease (LVH, angina or prior MI, prior coronary revascularization surgery or heart failure), stroke, TIAs, renal disease, peripheral artery disease or retinopathy. The patient has one or more major risk factors: smoking, poor lipid profile (see Appendix 5), age over 60, male or postmenopausal women, family history of cardiovascular disease (women under 65years old or men less than 55).

Group C - Evidence of target organ damage or cardiovascular disease or diabetes. NOTE: The presence of LVH on ECG or echocardiogram implies a major independent risk for cardiac death, MI and morbid CV events.

Clinical Tip: Use one of the available risk calculators to estimate patient's overlap CVD risk (e.g., NIH http://cvdrisk.nhlbi.nih.gov/)

## Charting Guidelines

When blood pressure is elevated on any single visit for a patient who is not acutely ill, this finding (but not the diagnosis of hypertension) should be recorded in the SOAP. The blood pressure can be recorded in the $O$ and a note to check BP on the next visit should be recorded in the $P$.

Additionally, it should be entered immediately on the problem list as an isolated finding (e.g., "ICD-9 796.2 elevated blood pressure without diagnosis of hypertension").

If the subsequent readings reveal this to be hypertension, it must be added to the problem list.
o The stage can be noted as well as a notation as to whether there is any evidence of end-organ damage. For example, "stage I with retinal changes."
o If medically managed, a notation should be made.
o The presence of any significant end organ damage (e.g., renal disease, LVH) must be added to the problem list.

Concomitant diseases such as diabetes or established coronary artery disease should be entered as separate problems. A timeline for treatment and dates of follow-up tests must be clearly charted in the management plan.

## MANAGEMENT

## Management Strategy

The management strategy for patients with hypertension consists of a number of specific steps and decisions. Except in rare instances, the overall approach should target decreasing all modifiable risk factors for cardiovascular disease rather than simply improving blood pressure numbers.

The patient must first be educated regarding the potential risks of hypertension (or pre-hypertension) and the importance of addressing this health problem.

The practitioner can then help each patient decide whether a non-pharmaceutical intervention or combined lifestyle modifications with an immediate referral for drug therapy is appropriate.

Significant barriers to compliance with lifestyle modifications include education level, income level, health literacy, patient motivation, and where they are positioned on the "willingness to change" continuum. Therapeutic success in chronic disease management is dependent on each patient's interest and effort. Therefore a patient-centered approach that includes shared decision making and recommendations tailored to each patient's resources and stage of change is essential.

Offers of therapy may be more readily accepted when they are based on patient preferences. Assaying each patient's level of literacy and understanding can allow the provider to offer appropriate, tailored information and educational messages regarding the risks and treatment options of hypertension.

## SUMMARY of Management Steps

1. Establish appropriateness of nonpharmaceutical intervention.
2. Educate the patient on risks and options.
3. Establish a treatment protocol.
4. Set a timeline with specific therapeutic goals.

## STEP 1: Establish appropriateness of non-pharmaceutical intervention.

JNC 8 (2013), although focused on pharmacological therapy for HTN, states that "for all persons with hypertension, the potential benefits of a healthy diet, weight control, and regular exercise cannot be overemphasized."

The ESH-ESC Guidelines (2013) report that life-style modifications can be as effective at lowering blood pressure as drug monotherapy.* Appropriate lifestyle changes may have the following therapeutic effects:

- delay or prevent hypertension
- delay or prevent medical therapy in grade 1 hypertensive patients
- contribute to improved BP allowing for a reduction in the number and doses of antihypertensive agents
- contribute to the control of other CV factors and clinical conditions

In addition, the results of the Treatment of Mild Hypertension Study (TOMHS) indicate that aggressive lifestyle therapy alone may also modestly reduce left ventricular hypertrophy.

[^3]The decision to recommend nonpharmaceutical interventions alone or in combination with drug therapy will hinge on the following considerations.

- the stage/grade of hypertension
- the presence of end-organ damage (symptomatic or not). Examples of end organ damage include left ventricular hypertrophy (LVH), renal damage, and retinal changes.
- The presence of additional risk factors. (See Step 4 in Evaluation section, page 12)



## Pre-hypertension

Patients with pre-hypertension should minimally receive counseling on risks and lifestyle changes. The treatment goal is to improve blood pressure as much as possible (without a specific BP target) and to prevent or slow the progression to stage 1 hypertension. Referral for antihypertensive drug therapy is not appropriate.

## Stage 1 hypertension

NOTE. Patients with pre-hypertension or uncomplicated stage 1 hyper-tension should be encouraged to start with lifestyle changes.

ESH-ESC (2013) recommends for grade 1 HTN to start with lifestyle changes for several months. If the target of < 140 systolic and < 90 diastolic is not met, anti-hypertensive medications should be added until that target is achieved. ASH-ISH (2013), differs somewhat, suggesting that for stage 1 hypertension a therapeutic trial could extend for as much as 6-12 months; drug therapy,
however, should be considered sooner if the patient is not adequately responding or there are significant risk factors or evidence of end organ damage.

If the patient is on hypertension medication, later management strategies can aide at weaning the patient on to lower doses or off medication altogether. This attempt must be done in cooperation with the prescribing physician.

The threshold for initiating drug therapy and the strength of the evidence supporting these thresholds varies depending on the subpopulation being treated.

## Age Dependent

The drug treatment threshold as well as the quality and quantity of evidence supporting these recommendations varies, in part, based on age.

1. Patients under 60. The JNC 8
guidelines (2013) recommend that for those $<60$ years an SBP of 140 or a DBP of 90 mm Hg should continue to be used to initiate pharmacologic treatment (presumably if life style changes are insufficient).

## Supporting Evidence

For the systolic treatment threshold of 140 mm Hg , there is a surprising lack of studies focusing on patients younger than 60. As a result, the 140 threshold recommendation is based on expert opinion. (JNC 8)

In contrast, the diastolic threshold of 90 mm Hg for ages 30 through 59 carries a strong recommendation (grade A)* based on high-quality evidence ${ }^{* *}$ from 5 trials demonstrating a reduction of

[^4]cerebrovascular events, heart failure, and overall mortality. (JNC 8) For patients < 50, diastolic blood pressure may be especially important to manage, making lowering the pressure below 90 a priority (ASH-ISH).

On the other hand, the diastolic threshold for ages 18 through 29 is based on expert opinion only because of a lack of good or even fair-quality RCTs.
2. Young, healthy males. ESH-ESC states that some young healthy males have an SBP > 140 and yet the central measurement of their blood pressure may be normal. "No evidence is available that they benefit from antihypertensive treatment; on the contrary there are prospective data that the condition does not necessarily proceed to hypertension." Unfortunately, central measurements are not commonly done in clinical practice. Practitioners should consider making lifestyle recommendations, monitoring, but not treating with medications.
3. Older patients. A treatment threshold of 150 SBP for drug therapy (rather than 140) may be more appropriate in older patients. But there is significant controversy regarding at what age the threshold should be raised to 150 .

JNC 8 recommends a 150 mm Hg SBP threshold starting at age 60. This is a strong recommendation with grade A evidence that it will reduce the risks of stroke, CHF and CHD. But JNC 8 reported that there are a lack of studies targeting patients with blood pressures between 140 and 150 systolic in this age group, and so the panel chose the higher threshold because of the lack of data. Not all panel members agreed on this approach and in a minority report recommended keeping the treatment threshold at 140 until or unless new research suggested otherwise.*
(Jackson 2014) ESH-ESC, ASH-ISH and the Canadian HTN guidelines (2013) are essentially in concert with the minority report, recommending the 150 SBP threshold only for patients $>80$ years old.

## Treatment Targets: How low to go?

For most patients the treatment goal is simply lowering BP below 140/90. This represents a change from past practice (JNC 8).

There are important issues to consider when deciding how low to set blood pressure targets for drug therapy. JNC 8 states "The relationship between naturally occurring BP and risks is linear down to very low $B P$, but the benefit of treating to these lower levels with antihypertensive drugs is not established." The lower risk of cardiovascular morbidity and mortality must be balanced against any potential adverse effects from the drug therapy itself.

There is no solid research evidence that using drug therapy to reach specific BP targets results in better patient-oriented outcomes. The ASH-ISH guidelines concur, stating "even though a blood pressure of $115 / 75 \mathrm{~mm} \mathrm{Hg}$ is ideal... there is no evidence to justify treating hypertension down to such a low level."

For patients > 60 years old, JNC 8 recommends lowering BP to under 150/90, but not aggressively increasing dosage or adding drugs to achieve pressures below 140 systolic or below $80-85$ diastolic. The panel reports that there is low quality evidence suggesting that treatment goals below 140 in this age group do not provide additional benefit. However, if medical
the panel minority, believed that evidence was insufficient to increase the SBP goal from its current level of less than 140 mm Hg because of concern that increasing the goal may cause harm by increasing the risk for CVD and partially undoing the remarkable progress in reducing cardiovascular mortality in Americans older than 60 years."

[^5]therapy does achieve lower numbers (e.g., < 130 SBP) "without adverse effects on quality of life," the medications need not be adjusted (JNC 8 expert opinion, ASH-ISH).

## SPECIAL POPULATIONS

Patients with stage 1 HTN with concomitant renal disease, diabetes, coronary disease, history of stroke or CHF should be considered for immediate drug therapy along with nonpharmacological interventions.

## Diagnostic Thresholds

Although some guidelines have recommended diagnostic values of 130/80 mm Hg for patients with diabetes or chronic kidney disease, neither ASH-ISH nor the JNC 8 guidelines support this lower threshold.

## Treatment Targets

Although lower BP treatment targets have been proposed for some sub-populations, the JNC 8 panel did not find sufficient evidence to support this more aggressive approach. For special higher risk populations (i.e., African Americans, individuals with chronic kidney disease or CVD including stroke, and those with multiple risk factors), the JNC 8 panel members concluded that drug therapy should simply lower BPs below 140 (SBP) as opposed to a specified lower target (e.g., 120, 130 or 135) due to a lack of evidence to the contrary. The ASH-ISH guidelines concur. However, the Canadian HTN guidelines (2013) still recommend a target of 130 SBP for diabetics, and ESHESC recommends targeting DBP $<85$.

## Stage 2 hypertension

For stage 2 HTN, ESH-ESC suggests starting with lifestyle modification for "several weeks." Drug monotherapy and additional anti-hypertensives can then be added until the BP is sufficiently lowered.

Signs of end organ damage, however, should trigger a more immediate consideration for drug therapy.

Note also that in cases of very high stage 2 HTN (systolic BP $\geq 180$ or diastolic $\geq 110$ ), drug therapy should be initiated immediately.

| $\begin{aligned} & \text { BP } \\ & \text { Class } \end{aligned}$ | $\begin{aligned} & \text { Systolic } \\ & \text { BP, mmHg } \end{aligned}$ | Diastolic BP, mmHg | Lifestyle Modification | Without Compelling Indications | With <br> Compelling Indications* |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Normal | < 120 | $<80$ | Encourage |  |  |
| Pre-hypertension | 120-139 | 80-89 | Yes | No anti-HTN drug(s) indicated | -------- |
| Stage 1 hypertension | 140-159 | 90-99 | Yes | Therapeutic trial of lifestyle changes or referral for antiHTN drugs. Some guidelines suggest drug therapy starting SBP 150 I patients > 60 years old (or >80) | Referral for drugs for compelling indication(s), other antiHTN drugs |
| Stage 2 hypertension | $\geq 160$ | $\geq 100$ | Option of aggressive lifestyle changes | Referral for 2-drug combination in most cases | Referral for drugs for compelling indication(s), other antiHTN drugs |

* Compelling indications include heart failure, post-myocardial infarction, high coronary disease risk, diabetes, chronic kidney disease, and recurrent stroke prevention

STEP 2: Educate the patient on risks and options.

## SUMMARY of Key Points

- Educate patients about overall effect on health (morbidity and mortality).
- Discuss the pros and cons of lifestyle change versus anti-hypertensive medication.
- Gauge their willingness to make lifestyle changes.
- Discuss the issues of compliance and longterm dedication to lifestyle interventions.

The first step is effective patient education (compliance is traditionally poor).

Although most individuals have a general understanding that high blood pressure is unhealthy, pre-hypertension and stage 1 hypertension are most often underappreciated. Patients must understand the significant risks associated with their stage of hypertension as well as any other cardiovascular risks they might have. On the other hand, the lifestyle changes recommended may not only decrease elevated blood pressure, but may also decrease the mortality and morbidly associated with cardiovascular disease and improve overall quality of life.

## Prehypertension

Studies and commentary published since JNC 7's introduction of the term hypertension have pointed out inconsistencies and controversies in calculating risks for morbidity and mortality based on the diagnosis of prehypertension.

Three meta analyses published in 2014 by Huang and colleagues investigated the association of prehypertension with coronary heart disease (Huang 2014a), CVD mortality, stroke mortality (Huang 2014b) and endstage renal disease (ESRD). (Huang 2014c)

The investigators found that prehypertension is associated with an increased risk for all of these conditions and especially with stroke mortality (but not with all-cause mortality).

For example, there was nearly a $60 \%$ relative increase in the incidence of ESRD (RR 1.59; $95 \%$ CI 1.39-1.91). Higher range BP readings (130-139/85-89) accounted for the majority of increased risk.

Therefore, even individuals with a systolic blood pressure of $120-139 \mathrm{mmHg}$ or a diastolic blood pressure of $80-89 \mathrm{mmHg}$ require health-promoting lifestyle modifications to prevent cardiovascular disease. The impact of prehypertension on the development of hypertension and target organ damage is usually a surprise to most patients.

## Stage 1 Hypertension

Stage 1 HTN is a major health risk. Patients may be tempted to wait until their blood pressure increases to a "more serious" stage before taking action. It is important for them to understand that $60 \%$ of the deaths attributable to hypertension involve patients with DBP readings between 90 and 104 mmHg , and most of those were within stage 1.

The PARQ should cover the patient's options which usually include non-pharmaceutical therapy only, drug therapy, or a combination of both. The risks of no treatment should also be part of this conversation.

The provider can use one of the various risk calculators available to predict the patient's overall cardiovascular risk and counsel them accordingly. (NIH http://cvdrisk.nhlbi.nih.gov/) The calculator is a good educational tool, demonstrating how the patient's individual risks are summative. It can also serve as a natural segue way into demonstrating how lifestyle interventions can simultaneously address multiple risk factors.

A patient-centered care approach is recommended. If a drugless therapeutic trial is considered, it is important to gauge the patient's readiness to make significant lifestyle changes. Employing a motivational interview approach may be the most effective.

The most effective therapy prescribed by the most competent clinician will control hypertension only if patients are motivated. Motivation improves when patients have positive experiences and trust in the clinician. This trust can be built over the course of multiple visits while treating them for their primary complaint as well as the provider demonstrating a willingness to listen to their issues and obstacles relative to making large life style changes.

Specific information on nutrition and lifestyle changes should be given and reinforced by developing a plan with the patient on how to introduce these changes. Goal setting and goal monitoring are essential. It is important to emphasize the positive benefits these can
make. The patient should be given advice on food preparation, meal selection, shopping strategies, and exercise choices. Patients should be encouraged to share barriers and problems that they may be encountering so that the clinician can aid in problem solving.

Although discussing these issues is pivotal to the initiation of the treatment plan, they must be periodically re-visited to increase the chances for success. Relapse management will likely be part of the treatment plan. It is important that patients understand that they can usually expect treatment to be a life-long commitment. (ASH-ISH) Setting a positive, energetic tone is very useful.

## STEP 3: Establish a treatment protocol.

SUMMARY: Lifestyle Modifications for Hypertension Prevention and Management

| ADVICE | Drop in Systolic <br> Blood Pressure |
| :--- | :--- |
| Lose weight if overweight (at least 22 lbs or 10 kg ). | 5 to 20 points for <br> every $10 \%$ loss |
| DASH diet: Reduce intake of dietary saturated and total fat; eat low-fat dairy foods; <br> increase fruits and vegetables. | 8 to 14 points |
| Increase aerobic physical activity ( $30-45 \mathrm{~min}$ most days of the week). | 4 to 9 points |
| Reduce sodium intake to no more than 2400 mg (1500 mg may be better) of sodium <br> or 6000 mg of sodium chloride. | 2 to 8 points |
| Limit alcohol intake - have no more than: 2 drinks/day for men; 1 drink/day for women <br> ( 1 drink $=12$ oz/360 ml beer, 5 oz/150 ml wine, or 1.5 oz/30 ml 80 -proof whiskey) | 2 to 4 points |
| Stop smoking. | unknown |
| Supplementation* | 11 points |
| CoQ 10 (100-200 $\mathrm{mg} /$ day) | 8 to16 points |
| Fresh garlic powder, yielding at least 4000 mcg allicin per daily dose | 3 to 6 points |
| Fish and fish oils, delivering at least 3000 mg daily of long-chain omega-3 (EPA + <br> DHA). | 3 points |
| Dark chocolate with high polyphenol content, at least one ounce (28 grams) <br> containing at least $50 \%$ cocoa content. |  |
| Maintain adequate intake of dietary calcium and magnesium for general health. |  |
| Maintain adequate intake of dietary potassium (approximately 90 mmol/day). |  |

* Note that in some cases the purported effect of supplements may be greater than that of dietary or lifestyle changes.

The supporting evidence, however, tends to be less robust for supplementation. Taking individual supplements is also more likely to narrowly target blood pressure as opposed to having a wider effect on multiple cardiac risk factors.

## Dietary and Lifestyle Modifications

Most patients with HTN have additional risk factors such as lipid abnormalities, glucose intolerance/diabetes, obesity, and cigarette smoking (ASH-ISH). The chief component of any nonpharmaceutical intervention for hypertension should be lifestyle modifications. A management plan to address HTN can roughly be divided into 6 arms: 1) weight loss, 2 ) dietary patterns (e.g., DASHlike diets), 3) salt reduction, 4) exercises, 5) alcohol reduction and 6) smoking cessation. Lifestyle modification has greater general acceptance and a deeper evidence base than simply treating piecemeal with supplements. A healthy lifestyle is more likely to impact other key cardiovascular risk factors as well.

## 1. Weight loss

## Strength of recommendation: Strong (ESHESC) <br> Level of evidence: A (ESH-ESC) <br> Treatment effect: 4.4/ 3.6 (with average loss of 5 kg ) <br> Response time: Starting with 5 kg of weight loss

This is one of the more effective changes a patient can make, especially if obese. Blood pressure can drop an estimated 5-20 points for every $10 \%$ weight loss. The target for obese patients is to lose at least 10 kg (22 pounds). In many cases this amount of loss can preclude the need for anti-hypertensive medications. At 5 kg some blood pressure changes will be noted. ESH-ESC recommends targeting a BMI of 25 and a
waist circumference of $<102 \mathrm{~cm}$ in men and $<88 \mathrm{~cm}$ in women unless contraindicated. There is a floor effect after which further weight loss will not result in still lower blood pressure. In addition, there is controversy whether or not weight loss is appropriate in all cases. Observational data reflect that the elderly and patients with established CVD findings may have a worse prognosis following weight loss regimens. (ESH-ESC)

## Options

The following are four different approaches to a weight loss program. Regardless of what method is used, target weights must be maintained.
A) Calorie reduction: Inspect the patient's diet. Screen for high amounts of empty-calorie foods (excessive fat, sugar and/or alcohol). Identify these to the patient and suggest lowercalorie alternatives, high in fiber whenever possible (see Appendix 6).
B) Fat reduction: Determine the optimum calorie intake for a patient based on basal requirements and activity level. Determine a permissible limit for daily fat intake ( $20-30 \%$ of total calories converted to grams of fat per day); saturated fats and trans-fatty acids should selectively be cut. Teach the patient to read food labels and evaluate non-labeled food items for fat content.
C) Healthful choices: Teach the patient to distinguish between 1) whole-grain and refined-grain products (white rice, white flour products, potatoes, pasta, and sweets) and 2) healthful oils (non-hydrogenated plant oils, oily fish, nuts and seeds) and unhealthful fats (red meat and dairy fat, commercial fried and baked foods, regular margarine and shortening). Emphasize daily consumption of high quality, low-glycemic index fruits, vegetables and legumes. Have the patient begin to monitor their diet with a diary. (Support documents for this weight loss option can be found in Appendices 9 and 10.)

[^6]D) Carbohydrate reduction Teach the patient how to track carbohydrate intake or refer them to resources on healthy low carbohydrate diets (e.g., OmniHeart, South Beach or Paleo diet) which offer specific detail on dietary patterns that emphasize replacing high carbohydrate foods with non-starchy vegetables, foods high in protein (lean meats, fish, poultry, vegetarian meat substitutes) or healthy fats (nuts, seeds, avocado, healthy oils). A low-carbohydrate diet is defined as consumption of $20-130 \mathrm{~g}$ of net carbohydrate (total carbohydrate minus fiber) per day or $30-45 \%$ of total daily calories. Note that the very low end of the range recommended in some of these diets is limited to the initial stage of the diet. The patient must be carefully monitored for ketoacidosis during this period.

## 2. DASH and DASH-Like Diets

Modifying eating habits can be very effective in reducing blood pressure and cardiovascular risk and should be considered as the first line of non-pharmaceutical intervention.

A number of dietary approaches can be recommended. They include the DASH (Dietary Approach to Stop Hypertension) and DASH-like diets (e.g., DASH with salt reduction, the Mediterranean diet, the OmniHeart diet).

A common denominator of these diets is:

- Increasing fiber, fruit and vegetable intake
- Increasing potassium, calcium and magnesium
- Decreasing sugar, partially hydrogenated and saturated fats.

Increase fiber to about 7-21 grams of mixed (soluble and insoluble) per day.
Recommendations are for at least 4-5 servings of fruit and 4-5 servings vegetables per day. Note: if fiber is taken as a supplement, be sure there is adequate water intake (drink 2 cups of water for every cup of fiber).

## DASH Diet

Strength of recommendation: $\mathrm{I}^{* *}$ (AHA/ ACC) Level of evidence: A (AHA/ ACC)
Treatment effect: decrease $5-6 / 3 \mathrm{mmHg}$ Response time: 2 weeks

This AHA/ ACC recommendation is based on level A evidence, derived largely from studies of the DASH dietary pattern (DASH and DASH-Sodium), which provided the highest quality evidence for this food-based dietary pattern causing improvements in lipid profiles and BP."

The evidence suggests the following:

- The BP lowering effect persists as long as the dietary pattern is continued.
- The beneficial effect has been demonstrated in adults with HTN and preHTN, in men and women, African Americans and non-African Americans, and in older and younger adults.
- The effect is independent of changes in weight and sodium intake.
- The magnitude of effect is sufficient to prevent progression from pre-HTN to HTN, promote nonpharmacological BP control in those with HTN, and supplement pharmacological BP lowering.

Reductions may be similar to those achieved by single drug therapy. The higher the blood pressure or more risk factors, the more useful this diet may be, even while taking medication.

ESH-ESC (2013) reports that the combination of the DASH diet with exercise and weight loss lead to greater reductions in BP and left ventricular mass than the DASH diet alone.

[^7]The DASH diet has demonstrated clinical success in normalizing stage 1 hypertension within two weeks. (See Appendix 7.)

Combine the DASH dietary pattern with lower sodium intake.

Strength of recommendation: I (AHA/ ACC) Level of evidence: A (AHA/ ACC)
Treatment effect: greater than the DASH diet
Response time: 2 weeks
"Both a healthy dietary pattern as exemplified by DASH and reduced sodium intake independently reduces BP. However, the BPlowering effect is even greater when these dietary changes are combined." (ACC/AHA)

## The Mediterranean Diet

Strength of recommendation: I (AHA/ ACC) Level of evidence: Iow (AHA/ ACC)
Treatment effect: decrease 2-7/ 1-3 mmHg (depending on patient population)
Response time: 6 weeks
Lower quality evidence suggests that various Mediterranean diet patterns also can reduce BP.

AHA/ACC reports that there is no uniform definition of the Mediterranean diet across the various RCTs and cohort studies. In general this dietary approach has the following characteristics (very similar to the DASH diet):

- Moderate total fat (32-35\% of total calories); higher in omega-3's while lower in saturated fats ( $9-10 \%$ ); an emphasis on fatty fish, olive/canola oil (less dependence on butter), nuts (walnuts, almonds or hazelnuts); and less quantity of red meat (with an emphasis on lean meats).
- High in fiber (27-37 g/day) from fruits and vegetables (especially roots and greens) and whole grains (e.g., cereals, breads, rice, pasta)

Counseling to eat a Mediterranean diet compared to minimal advice to consume a lower fat diet reduced BP 6-7/2-3 mm Hg in middle aged and older subjects with diabetes of 3 or more CVD risk factors. In one observational study of healthy younger adults, it lowered BP 2-3/1-2. (AHA/ACC)

## The OmniHeart dietary pattern

Strength of recommendation: ? (AHA/ACC)
Level of evidence: moderate (AHA/ ACC)
Treatment effect: decrease 1-3 SBP > than the DASH diet
Response time: 6 weeks

The OmniHeart dietary pattern is similar to the DASH but decreases the carbohydrate component. The trial demonstrated that substituting either more healthy protein (lean meat, legumes, nuts, seeds) or more healthy fat (healthy oils, nuts, seeds) for $10 \%$ of carbohydrate in the DASH diet led to modestly better improvements in lipid and blood pressure risk factors (the equivalent of about $48 \%$ carbohydrates vs the $58 \%$ in the DASH diet).

## Other low/lower carb dietary patterns

Although not addressed in any of the HTN guidelines cited in this care pathway, there is evidence that some popular, lowcarbohydrate diets may be suitable for patients who are motivated to adopt them.

Santos (2012) in a moderate quality systematic review and meta-analysis (based on 23 reports, $\mathrm{N}=1,141$ obese patients) reports short-term improvements in blood pressure: SBP -4.81 (95\% CI -5.33,-4.29); DBP -3.10 (95\% CI -3.45,-2.74).
Improvements in weight, fasting glucose, and some measures of blood lipids have also been demonstrated, but LDL cholesterol has either been unaffected, or has risen. (Santos 2012, Hession 2009, Nordmann 2006)

Low-carbohydrate diets have not been tested for long-term safety in clinical trials, but observational studies suggest they are safest when their protein content emphasizes plant-
based proteins, fish, or poultry. (Hu 2006) In fact, large cohort studies to date have found that low-carbohydrate diets are only associated with reduced risks of diabetes, coronary heart disease, and all-cause mortality when they are based primarily on vegetable sources of protein and fat. (Halton 2008, deKoning 2011, Halton 2006, Fung 2010). In contrast, diets high in red meat and processed meats are consistently associated with increased risk of diabetes, coronary heart disease, and all-cause mortality. (Pan 2011, Bernstein 2010, Sinha 2009).

## 3. Decrease salt (sodium chloride)

Strength of recommendation: I (AHA/ ACC, ESH-ESC)
Level of evidence: A (AHA/ACC, ESH-ESC) Treatment effect: depends on amount of decrease
"There is strong and consistent clinical trial evidence that reducing sodium intake lowers BP." (AHA/ACC)

This BP-lowering effect has been demonstrated in adults with HTN and preHTN, in men and women, in African Americans and non-African Americans, and in older and younger adults.

Trials include well-controlled feeding studies as well as studies in which participants were counseled to lower sodium. Reducing sodium intake can result in a number of benefits (AHA/ACC):

- prevent progression from pre-HTN to HTN,
- promote nonpharmacological BP control in those with HTN,
- lower the risk of CV events in people with and without HTN (based on observational studies).

A variety of controlled and observational studies also suggest further potential benefits such as

- reduced diuretic-induced potassium wastage
- LVH regression
- protection from osteoporosis and renal stones through reduction in urinary calcium excretion.

The effect of reducing sodium intake on BP is independent of changes in weight.

Individual response of blood pressure to reduced sodium intake differs widely, depending on an individual's "salt sensitivity." However, in a 1997 overview of 32 RCTs, Cutler suggests that most patients are probably salt sensitive. Cutting intake in half will reduce pressure in some patients; others will have no response unless salt is more significantly reduced.

ASH-ISH reports that more African Americans appear to be salt sensitive than Caucasians. This sensitivity may partly account for why young African Americans tend to have earlier and more severe hypertension than other groups.

Whether a patient is truly "salt sensitive" and will respond to sodium reduction can be determined by either a period of trial therapy or (less commonly) by testing for low plasma renin levels after a few days of strict salt restriction.

The average American consumption is variously reported as ranging from 7-11 g/day of salt (Misserli 2014) or an average of 3.5 $\mathrm{g} /$ day of sodium (Brown 2009). ${ }^{\dagger \dagger}$

[^8]$1 / 4$ teaspoon salt $=600 \mathrm{mg}$ sodium
$1 / 2$ teaspoon salt $=1,200 \mathrm{mg}$ sodium
$3 / 4$ teaspoon salt $=1,800 \mathrm{mg}$ sodium
1 teaspoon salt $=2,400 \mathrm{mg}$ sodium

There is good consensus that reducing salt intake from its current averages will lower blood pressure and will decrease strokes and coronary heart disease mortality. ESHESC expert panel declares that there is no evidence that lowering from a high intake to a moderate intake is harmful.

But a controversial issue is whether there is a $J$ curve distribution of the risks and benefits of reducing sodium intake. (Messerli 2014) Is there a threshold under which the benefits of lower blood pressure are offset by other potentially deleterious physiological effects such as increased renin activity and aldosterone levels? Salt restriction to under $3 \mathrm{~g} /$ day ( $<1,200 \mathrm{mg}$ of sodium) may augment these risks. In the ONTARGET study there was a J-shaped association with an increase in congestive heart failure and cardiovascular death (but not with MIs or stroke). (Mancia 2011) Studies have demonstrated that in heart failure patients those in a very low sodium cohort ( $<1,800 \mathrm{mg} / \mathrm{dy}$ ) had a higher mortality and morbidity rate. (Misserli 2014)

Cook (2014), however, reports that in the TOHP Follow-up Study, which used perhaps the most accurate assessment of sodium intake (based on measuring sodium excretion from routine 24 -hour urine collections), there was a continued decrease in CVD events with sodium levels as low as 1500 $\mathrm{mg} /$ day and no evidence of a J curve. So the issue remains unsettled.

## Decrease patient's baseline intake by $1,000 \mathrm{mg}$

Strength of recommendation: IIa (AHA/ ACC) Level of evidence: B (AHA/ ACC)
Treatment effect: decrease $3-4 / 1-2 \mathrm{mmHg}$ Response time: 1-3 months

An initial target may be to reduce sodium intake by at least $1,000 \mathrm{mg} /$ day. Even this amount of sodium reduction can reduce CVD events by about 30\% (AHA/ACC).

## Reduction Targets: $\mathbf{2 , 4 0 0} \mathbf{~ m g}$

Strength of recommendation: $11 a^{\ddagger \ddagger}$ (AHA/ACC)<br>Level of evidence: B (AHA/ ACC)<br>Treatment effect: decrease $4-5 / 1 \mathrm{mmHg}$<br>Response time: 1-3 months

The usual salt intake in many countries is between 9 and $12 \mathrm{~g} /$ day. A decrease to about $5 \mathrm{~g} / \mathrm{day}$ ( $<2500 \mathrm{mg}$ of sodium) has a small SBP lowering effect ( $1-2 \mathrm{~mm} / \mathrm{SBP}$ ) in normotensive subjects and as much as 4-5 mm in hypertensives. (ESH-ESC). The evidence varies for stricter control. There have been fewer studies trying to elucidate the most effective sodium target and so the evidence for any specific number is less robust. Limiting sodium intake to no more than $2,400 \mathrm{mg} /$ day is considered a mild restriction and is more useful in conjunction with other therapies. This level of restriction allows light use of salt in cooking, no salt added at the table, high salt foods must be avoided. (See Appendix 8)

## Reduction Target: 1,500 mg

Strength of recommendation: IIa (AHA/ ACC) Level of evidence: B (AHA/ACC) Treatment effect: decrease $7 / 3 \mathrm{mmHg}$ (greater with the 2,400 restriction)
Response time: 1-3 months
Further reduction of sodium intake to 1,500 mg /day may result in even greater reduction in BP. There remains controversy as to whether levels this low actually do confer this added benefit-- the Institute of Medicine 2013 report, for example, questions this target (Strom 2013.) There is also a question as to whether this lower threshold may even carry increased risk for

[^9]cardiovascular events, especially in patients already suffering from heart failure.

To achieve this reduction, no salt is used in cooking or at the table. No salty foods are permitted. Only low-salt canned foods are permitted.

Changes are typically seen in one month, maximum changes in about three.

## The challenge

Many patients find that reducing the amount of sodium in the diet can be a challenge.

Educational materials with strategies to help patients lower sodium intake are provided by several Federal and private sources

It is estimated that $80 \%$ of sodium intake is in the form of "hidden salt." (ESC-ESH). Eating out (especially fast food) and eating processed foods are the two largest contributors to the salt in the American diet. Seventy-five percent of sodium intake is derived from processed food alone. Most patients do not realize the large amount of salt that are in common foods such as breads, canned foods, soups and processed meats.

There is some evidence to indicate that patients having trouble adhering to a low sodium diet may benefit from increasing dietary intake of calcium or taking supplements. (See calcium, p. 29)

## 4. ExERCISE

Strength of recommendation: IIa (AHA/ACC) Level of evidence: A (AHA/ ACC)
Treatment effect: decrease 2-7/ 1-4 mmHg (AHA/ ACC, ESH-ESC)

Exercise can play an important role in the treatment of stage 1 and 2 hypertension. Additionally, exercise may help prevent the development of hypertension (and presumably prehypertension) in healthy, normotensive adults. A prospective 1984
study of 6,039 normotensive subjects followed for a median of 4 years found that increased physical fitness was significantly associated with decreased risk of developing elevated BP. (Blair 1984)

Besides lowering blood pressure, exercise can improve key patient-centered end points such as the rate of cardiovascular disease. AHA/ACC reports that one exercise study estimated that improved blood pressure accounted for $27 \%$ of the activity-related reduction in CVD rates and another 35\% of the reduction was due to a beneficial effect on serum lipids.

Advice can start with encouraging patients to walk more, use bicycles, climb stairs when possible and overall integrating physical activity into everyday life.

But to attain more meaningful BP reduction in patients, more moderately intense physical activity should be advised (40\%-60\% of maximum oxygen consumption), such as 30 to 45 minutes of brisk walking most days of the week. Patients should exercise at an intensity in which they readily can perceive the exertion but are under no distress (no pain or trouble breathing).

## Frequency: 3 to $4 x /$ wk (or more)

Level of evidence: Strong evidence (ESHESC)
Duration: average 40 minutes per session, Intensity: moderate-to-vigorous intensity

This AHA/ACC recommendation is based on a meta-analysis and reviews of studies rated as having fair to good quality (published from 2001 and later) combined with recommendations from the 2008 Physical Activity Guidelines Advisory Committee Report. The federal Physical Activity Guidelines state that "most health benefits occur with at least 150 minutes (2 hours and 30 minutes) a week of moderate intensity physical activity, such as brisk walking.
Additional benefits occur with more physical activity." (AHA/ACC)

Patients should be told that exercise levels must be maintained; otherwise, beneficial changes will disappear within about 3 weeks.

Resistance Exercise Training and BP. A review of ten studies assessing resistance training demonstrated beneficial changes in systolic BP, with less consistent improvement in diastolic BP. The magnitude of reduction was not specified. (AHA/ACC)

Patients with uncontrolled HTN (e.g., SBP $\geq$ 180/110) should not lift weights. Moderate weightlifting, however, may be beneficial for other hypertensives.

The Mayo Institute's web site offers some useful information regarding weight training:

- Warn patients not to hold their breaths while exerting themselves because e this could cause dangerous spikes in blood pressure.
- They should be encouraged to use lighter weights and increase the repetitions.
- Alternating between upper and lower body exercises allows muscles to rest.


## Starting an exercise program

NOTE: Patients with multiple risk factors or established coronary disease should have a stress EKG performed prior to prescribing an exercise program.

Although a little complicated, a good method for projecting a safe target heart rate is to use Karvonen's formula. First subtract the patient's age from 220 and then subtract the patient's resting heart rate (HR) from that result. Multiply the answer by $85 \%$ and add back the resting heart rate. ${ }^{\S \S}$

Karvonen's formula = [220 - (pt. age + resting HR) x 85\%] + resting HR

[^10]Example: 220-60 (the patient's age) $=160$. His resting heart rate is 90 , so $160-90=70$. The patient has no major risk factors for cardiovascular disease, so it is safe to work out at $85 \%, 70 \times .85=59.5$ (round to 60 ). To complete the calculation add back the resting heart rate of $90.60+90=150$. The patient should use 150 as his target training rate. If he were a smoker with hyperlipidemia, a safer rate would be based on $65 \%$ and would calculate out to 140.

## 5. Decrease alcohol intake

Moderate alcohol consumption can be helpful in protecting against cardiovascular events, but greater amounts of alcohol can raise blood pressure. Men should not exceed 2 drinks a day, women 1 per day.

Two drinks is the rough equivalent of $1 \mathrm{oz}(30$ ml ) of ethanol [e.g., $24 \mathrm{oz}(720 \mathrm{ml})$ of beer, 10 $\mathrm{oz}(300 \mathrm{ml})$ of wine, or $2 \mathrm{oz}(60 \mathrm{ml})$ of $100-$ proof whiskey]. Patients who cut down to these limits may see changes in their blood pressure within 3 weeks.

## 6. Smoking cessation

Although smoking causes a transitory increase in blood pressure, evidence is poor that stopping smoking will lower blood pressure. Nonetheless, it is important to eliminate this risk for heart disease and stroke. (ASH-ISH, ESH-ESC)

## Other useful dietary options

Both vegan and less strict vegetarian diets (permitting milk and/or egg products) are associated with lower prevalence of hypertension in observational studies. Randomized controlled trials have demonstrated blood pressure lowering effects for switching from omnivorous to lacto-ovovegetarian diets in either hypertensive or normotensive subjects. Weight reduction and increased intake of potassium, magnesium, calcium, and other nutrients are all likely contributors to this effect. (Appleby 2002, Margetts 1986, Rouse 1983)
A 2014 meta-analysis of RCTs and observational studies further supports the
effectiveness of a vegetarian diet. A combined pool of 7 small RCTs ( $\mathrm{N}=311$ ) demonstrated a mean reduction of 4.8 SBP ( $95 \% \mathrm{CI}-6.6$ to -3.1 ) and 2.2 DBP ( $95 \% \mathrm{CI}-$ 3.5 to -1.0). A pool of 32 observational studies ( $N=21,604$ ) yielded even greater reductions. (Yokoyama 2014)

## Consume about 4-6 ounces of fish per

day. Daily intake of 3.65 grams of omega-3
fatty acids had an independent blood pressure-lowering effect in a randomized controlled trial. Less frequent fish consumption (at least 1-2 times a week) has been associated with reduced risk of heart disease and thrombotic stroke in observational studies, but has not been investigated for blood pressure lowering. (Bao 1998, Kris-Etheron 2003, Skerrett 2003)

Due to environmental contaminants in fish women who may become pregnant or are pregnant/ breastfeeding, as well as young children, should follow FDA and EPA recommendations for limiting consumption of certain fish. Other individuals can probably consume larger amounts of fish, but should avoid larger, predatory fish such as shark, swordfish and king mackerel, and should remove visible fat and skin before cooking or eating.

Increase dietary intake of potassium to 5,000 or $7,000 \mathrm{mg} / \mathrm{d}$ (by increasing fruits, vegetables, legumes, nuts and seeds, and using salt substitutes). (See supplements, $p$ 29 and Appendix 11)

Consider a strict supervised fast. Goldhamer and others have reported an antihypertensive effect of fasting for an average of 1-2 weeks. (Goldhamer 2001, Goldhamer 2002, Lakovlev 1997, Vertes 1979) Average reductions reported in two trials were 37 and 20 mmHg systolic, and 13 and 7 mmHg diastolic. Proposed mechanisms for this effect include weight loss, sodium depletion, reduced circulatory fluid volume, and reduced insulin secretion. Most studies were conducted in in-patient programs which may limit their applicability to an outpatient-based practice.

Goldhamer's approach begins with a prefasting diet for at least two days consisting of fruits and vegetables only, followed by wateronly fasting for up to 28 days until blood pressure stabilizes. After fasting, a transitional juice diet is used for one or more days, followed by the introduction of a diet of whole natural foods. The lifestyle changes inherent in an inpatient treatment program make it difficult to assess the contribution of fasting alone. Also, Goldhamer has not investigated the durability of blood pressure changes after patients return to their normal life. However, a Russian group reported a persistent clinical effect at three months' follow-up apparently without further treatment. (Murav'ev 2003)

## Avoid/reduce consumption of sugar sweetened and artificially sweetened

 beverages. Based on large retrospective observational studies, there appears to be an increased risk for hypertension (and diabetes) among those who drink 1 or more artificially or sugar sweetened beverages per day (HR $1.13 ; 95 \% \mathrm{Cl} 1.09$ to 1.17). The increased risk appeared to be stronger with colacontaining beverages and carbonated beverages, but was not directly explained by fructose concentrations. Possible mechanisms are unclear. (Cohen 2012)Consume small amounts of chocolate. The chocolate should be of high polyphenol content, at least one ounce ( 28 grams) with $50 \%$ or more cocoa. A Cochrane metaanalyses of 20 studies involving 856 mainly healthy participants revealed a statistically significant blood pressure reducing effect of flavanol-rich cocoa products compared with control in short-term trials of 2-18 weeks duration. SBP dropped on average - 2.77 ( $95 \% \mathrm{Cl}-4.72,-0.82$ ) mm Hg and DBP -2.20 ( $95 \% \mathrm{Cl}-3.46,-0.93$ ) mm Hg. Long term outcomes or effect on CVD has not been reported. (Reid 2012)

Manage caffeine intake. Caffeine consumption can acutely raise blood pressure, particularly in patients who are already hypertensive. To what degree this translates into chronic hypertension and what form the caffeine must be in is less clear. Caffeinated soft drinks and energy drinks may contribute to HTN, but coffee is not likely one of the offenders, perhaps because it is a more complex substance.

While older meta-analyses (Kahn 2007, Clement 2009) found a small increase in SBP in coffee drinkers ( $1-2 \mathrm{mmHg}$ ), a more recent review found the opposite. Moderate consumption of coffee (3-4 cups/day delivering 300-400 mg of caffeine) appeared to be a low risk behavior and may even provide health benefits. (Mesas 2011)

Most observational research finds coffee not to be associated with increased CVD risk and in a large 2010 cohort study coffee actually was associated with reduced risk (LopezGarcia 2009, de Koning 2010).

In addition, the risk for developing type 2 diabetes was found to be lower in association with consumption of four or more cups of coffee per day compared to less than two cups per day. (Muley 2012)

The Nurses' Health Studies I and II ( $\mathrm{N}=140,544$ ) found that caffeinated colas, but not habitual coffee intake, carried an increased risk for HTN.

Some groups, including people with HTN, children, adolescents, and the elderly, may be more vulnerable to the adverse effects of caffeine. In addition, currently available evidence suggests that it may be prudent for pregnant women to limit coffee consumption to 3 cups/day providing no more than 300 $\mathrm{mg} /$ day of caffeine. (Higdon 2006)

## Dietary Supplements/Botanicals

If changes in diet and lifestyle are insufficient or compliance is poor, consider one or more of the following approaches. Care should be taken that the diet includes sufficient potassium (see below), magnesium and calcium.

Роtassium ( $35 \mathrm{~g} / \mathrm{d}$ )
Many people eat diets that are low in potassium, and they should be taught about available sources of dietary potassium.
(ACC/AHA) (See Appendix 11.)
A 2013 systematic review and meta-analysis (Aburto 2013) reported that high quality evidence demonstrated that increased potassium intake reduces SBP by 3.49 ( $95 \%$ CI 1.82 to 5.15 ) mm Hg and DBP by 1.96 ( 0.86 to 3.06 ) mm Hg in hypertensive adults. This was based on 22 RCTs ( $\mathrm{N}=$ 1606 ) and 11 cohort studies ( $\mathrm{N}=127,038$ ). Systolic blood pressure was reduced by 7.16 (1.91 to 12.41 ) mm Hg at higher potassium intake levels (90-120 mmol/day). No significant adverse effects on renal function, blood lipids, or catecholamine concentrations were reported. Improved outcomes included a decrease in stroke risk ( $\mathrm{RR} 0.76,0.66$ to $0.89)$. Reductions, however, did not achieve statistical significance in the case of cardiovascular disease (RR 0.88, 0.70 to 1.11) or coronary heart disease (RR 0.96 , 0.78 to 1.19).

The individual practitioner will need to decide whether to recommend supplementation. Potassium intake target should be 90 $\mathrm{mmol} / \mathrm{day}$, or about $3500 \mathrm{mg} /$ day.

## Note: To achieve $3500 \mathrm{mg} / \mathrm{dy}$ prescription strength supplementation is necessary and so a referral is required.

Special considerations for patients taking diuretics:

- if K wasting: ensure adequate $\mathrm{K}, \mathrm{Mg}, \mathrm{Ca}$ replacement
- if K sparing: do not supplement
- K dosage: $10-20 \mathrm{mEq}$ ( $60 \mathrm{mEq} / \mathrm{d}=2-3 \mathrm{~g}$ of K ); 1 tsp KCL has 60-70 mEq.

NOTE: K citrate may cause less GI irritation ("K-Lyte").

Increasing dietary K allows 80\% of patients to cut anti-hypertensive medications in half.

Contraindications: renal disease, K+ sparing drugs, angiotensin converting enzyme inhibitors or NSAIDs.

OMEGA-FATtY ACIDS (FROM FISH OIL) $3 \mathrm{~g} / \mathrm{d}$ Large amounts of omega-3 fatty acids may lower blood pressure; however, some patients experience abdominal discomfort. One study found no significant effect in preventing hypertension.

CALCIUM SUPPLEMENTATION ( $1-2 \mathrm{~g} / \mathrm{d}$ ) Calcium supplementation may cause a drop of $6 / 3$ over a 2-3 month period (especially if the patient has decreased serum renin, decreased serum calcium or increased calcium secretion).

Calcium targets range from a threshold effect of 800 to about $1,000 \mathrm{mg} / \mathrm{d}$. Although in a 1996 meta-analysis of 22 RCTs, Allender concluded that the resulting decrease in diastolic blood pressure was too small to support a recommendation of calcium supplementation; other authors suggest that adequate dietary calcium may offset the blood pressure raising effects of sodium chloride. This protective effect may be of particular use in salt-sensitive patients.

MAGNESIUM SUPPLEMENTATION (300-400 $\mathrm{mg} / \mathrm{d}$ )
The highest food content: green leafy vegetables, unrefined grains, and nuts. Contraindications: renal failure or heart block without artificial pacemaker.

Coenzyme $\mathbf{Q}_{10}(100-200 \mathrm{mg} / \mathrm{d})$
Three small randomized controlled trials (one on hypertensive diabetics) and several uncontrolled trials suggest $\mathrm{CoQ}_{10}$ can significantly reduce blood pressure and facilitate weaning the patient from medication. (Singh 1999, Burke 2001, Hodgson 2002) A

2009 Cochrane review reported a systolic drop of about -11 mmHg . (Ho 2009)

The dose may be split in half and taken twice a day for better absorption. It takes 3-4 weeks for the anti-hypertensive effect to take place and only $7-10$ days for its effects to recede if supplementation is discontinued.

GARLIC POWDER (STANDARDIZED FOR ALLICIN YIELD, $4000-5000 \mathrm{mcg}$ )
Garlic may affect vascular tone via the nitric oxide (Al-Qattan 2006, Pedraza-Chaverri 1998) or other (Benavides 2007, Kaye 2000) pathways. Two recent systematic reviews found BP reductions averaging 8/7 (based on 11 RCTs) (Ried 2008) and 16/9 (based on 10 RCTs (Reinhart 2008) among hypertensive subjects.

## Additional Interventions

## Special Cases: Stress reduction

If the patient's history suggests stress as a major contributor or if treatment response is poor, consider referring for stress reduction or counseling. Choose one based on availability, cost effectiveness, and most importantly, the patient's own interest: yoga, meditation, biofeedback or behavioral modification counseling.

## CHIROPRACTIC MANIPULATIVE THERAPY

A 2012 (Mangum et al) systematic review reported that there was a lack of high quality evidence to support the use of manipulation as an effective treatment for hypertension. The two best studies with the lowest threat of bias utilized diversified (Goertz 2002,) or Gonstead adjusting (Plauger 2002). While these two different techniques did lower blood pressure, they demonstrated no clinical benefit over their controls. In fact, the addition of diversified manipulation added nothing to dietary control and Gonstead was outperformed by both massage and no treatment (although this difference did not reach statistical significance).

Studies with poorer methodology and an unclear risk of bias reported mixed results. Two demonstrated a statistically and clinically significant drop in blood pressure either with a single atlas adjustment (Bakris 2007) or using an activator (Abram 1988). On the other hand, Morgan 1985, utilizing osteopathic manual therapy, showed no benefit in a cross over design.

## An overview of the reports and research

Both anecdotal reports and controlled trials have been published concerning manual therapy and high blood pressure. One study reported a possible relationship between the spine and blood pressure. Three osteopaths examined the spines of 102 patients and detected a particular pattern of sensitive and restricted joints in patients with high blood pressure (17/22 hypertensives had this pattern as compared 20/80 normotensives). (Johnston 1980)

In individual case reports, chiropractic treatments to the cranium (Connelly 1998), the neck (McGee 1992) and other specific areas of the spine (Plaugher 1993) have resulted in a reduction of blood pressure over time and reduction in use of medication. However, published clinical trials of various types of manipulation have yielded mixed results.

Fichera (1969) compared 35 patients with normal blood pressure to 22 patients with pressure in the mild to moderate range (i.e.< 180/110). All subjects received a soft tissue manipulation (not described). $73 \%$ of the hypertensives and $43 \%$ of the normotensives experienced a decrease in blood pressure of more than 3 mmHg . Another small group study with no controls reported a significant reduction in both systolic and diastolic pressure (an average of 27 mmHg and 13 mmHg , respectively) when treated over a two month period. (Goodman 1992). Another small observational study looked at subjects who had normal blood pressure. Manual therapy applied to the upper cervical spine
resulted in no significant drop in blood pressure. In fact, about half of the patients had an increase in blood pressure right after the treatment. (Tran 1977)

Four controlled trials looked at relatively short-term effects of a single manipulation. Three found a blood pressure lowering effect and one found no such effect. Dulgar (1980) found that 5 subjects with normal blood pressure experienced a drop in pressure (11\% decrease) after manipulation of the neck compared to both no significant change in 5 control subjects receiving a sham manipulation and 5 more controls who received no intervention at all (but were allowed to rest for the same length of time allotted to treatment). A larger trial compared the results of chiropractic adjustments of the neck in a mixed group (subjects with both normal blood pressure and those with elevated blood pressure) with a group that received no intervention. The treatment group as a whole experienced a small average decrease of 3 mmHg which probably is not very useful therapeutically. Within the group, those subjects who actually had hypertension experienced a more dramatic average decrease, some ranging between 8 and 20 mmHg . (McKnight 1988) Yates (1988) reported on a group of 21 hypertensives that were divided into no treatment, an adjustment with an activator instrument, and a sham treatment with an activator on the off position. The treated patients experienced an average 14.7 mmHg drop in systolic and 13 mmHg drop in diastolic pressure with little or no change in the other subgroups. Abram (1988) reported a statistically and clinically significant drop in blood pressure compared to placebo (-17.7/-13.0 vs -1.43/-1.43) 1 hour after assessing patient's spines by palpation and treating with an activator instrument. There were only 21 patients in the study, of which 7 received the active intervention. It is unclear whether allocation concealment and blinding was appropriate. There was no follow up after the intervention phase.

A more recent study (Bakris 2007) reported the effects of NUCCA adjustments to the Atlas of 25 patients with hypertension compared to 25 controls. The intervention
phase lasted 8 weeks, but most of the patients required only one treatment. The manipulation group had a statistically significant drop in blood pressure compared to controls (-17.2/-10.3 vs -3.2/-1.8). These were findings were clearly clinically significant, but it is unclear whether allocation concealment and blinding was appropriate. There was no follow up after the intervention phase.

On the other hand, Nansel (1991) compared a single cervical manipulation to simply setting up as if to apply the manipulation on normotensive subjects. While the range of neck movement significantly improved in the treatment group, there was no difference between the two groups in heart rate and blood pressure which were checked at 5,30 , 60, 120 and 240 minutes post intervention.

Each of these trials looked only at the immediate effect of one treatment. They did not evaluate the effect of multiple visits, whether the effect was only temporary, and whether it improved the course of hypertension over time and would reduce dependence on medication.

Two small controlled studies looked at manipulation of the cranium by applying controlled pressure on specific suture lines of the skull. One study showed only a slight, insignificant change in the treatment group, no difference in a sham treatment or a "resting" control group. (Maloney 1990) In contrast, another smaller study, reported a 10 mmHg drop in systolic pressure in the treatment group and no change in the resting control group. There was no sham treatment in this study. (Unger 1993)

Four controlled studies looking at longer-term effects on blood pressure have shown no benefit from manipulation. One trial of osteopathic manipulation (Morgan 1985) demonstrated no difference between those who were treated over a six-week treatment period compared to those who received a sham treatment. This was a crossover design, where the same subjects underwent both real and sham treatment regimen. Two controlled studies looked at a series of treatments (range 7-10) and the effect they
had on blood pressure and aldosterone. One of these studies found no change in blood pressure (Mannino 1979), the other reported only a temporary reduction. (Wagnon 1988) Interestingly, both reported significant aldosterone-lowering effects. Whether continued reduction of aldosterone over a longer treatment time would have eventually lowered blood pressure is not known. The largest RCT to date (Goertz 2002) included 140 subjects with high normal and stage 1 hypertension. The authors found no added benefit to manipulation plus dietary recommendations compared to dietary recommendations alone. Patients were manipulated $3 x /$ week for four weeks.

Although there is some evidence both supporting and refuting the notion that a single treatment with manual therapy may temporarily lower blood pressure in some patients, the higher quality studies have had negative results. Currently, no research has succeeded in demonstrating that serial treatments are effective in long-term reduction of high blood pressure.

In some cases, manipulation may assist in resolving secondary problems that are preventing a patient from complying with recommended lifestyle changes. For example, a patient with musculoskeletal discomfort due to joint dysfunction may be unwilling or unable to effectively exercise. In such cases, manipulation can be an important component of the overall management program.

STEP 4: Set a timeline with specific therapeutic goals.

The ESH-ESC Guidelines (2013) report that life-style modifications can be as effective in lowering blood pressure as drug monotherapy although the major drawback is the low level of adherence over time-which requires special attention to overcome.

## Management Timeline (16-17)

Stage 1 HTN (systolic 140-149 or diastolic 90-99): 6 months to 1 year.

Patients with stage 1 hypertension who do not have evidence of end organ damage nor significant risk factors are good candidates for nonpharmacological management.

- If patients are coming in for regular care, monitor every visit or weekly.
- If patients are not coming in routinely, it is recommended to contact them weekly to encourage adherence to the program. Check $B P$ at the end of one month. Address any compliance problems.
- Patients should be encouraged to track their own BP with a home unit.
- A combination of phone support and close monitoring should continue until target $B P$ is met.
- Re-evaluate every 3 months until BP target is met. If BP increases to stage 2 or there are new significant risk factors or new evidence of target organ damage, then consider referral for the addition of pharmaceutical therapy.
- When BP target has been consistently met, encourage the patient to continue selfmonitoring and to seek care if they again become consistently hypertensive. If they do not have a home unit, encourage them to have their BP checked at least once a year.


## Stage 2 or Stage 1 HTN with target organ damage / significant risk factors: Referral.

Patients in the above categories should be referred for medical consult. At the same time, the patient should be educated about appropriate lifestyle and dietary changes. When the hypertension is pharmacologically controlled, management can then be aimed at reducing or eliminating medication (in cooperation with the prescribing physician).

In several clinical trials, hypertension did not occur in 30-70\% of patients who discontinued anti-hypertensive therapy. In patients who have been taking monotherapy for at least one year and whose respective diastolic and systolic blood pressure is persistently below 85 and 130 mmHg , it may be worthwhile to
stop drug treatment and to monitor at frequent intervals, perhaps every 6 weeks to 2 months. If a patient has significant endorgan damage, the discontinuation of medication should probably not be considered, even if pressure persists in an acceptable range.

## Medications

For the patients referred for pharmaceutical intervention, refer to the JNC-8, ASH-ISH or ECC guidelines.

Thiazide-type diuretics should be used in drug treatment for most patients with uncomplicated hypertension, either alone or combined with drugs from other classes. Certain high-risk conditions are compelling
indications for the initial use of other antihypertensive drug classes (angiotensinconverting enzyme inhibitors, angiotensinreceptor blockers, $\beta$-blockers, calciumchannel blockers).

Most patients with hypertension will require 2 or more different anti-hypertensive medications to achieve blood pressure goals.

If blood pressure is more than $20 / 10 \mathrm{mmHg}$ above goal blood pressure, considerations should include initiating therapy with two agents, one of which usually should be a thiazide-type diuretic.

APPENDICES follow on next page.

## Appendix 1: Factors Affecting the Immediate Accuracy of Office Blood Pressure

## Factor

- Magnitude
o Missed ausculatory gap
- Examiner
o Impaired hearing
o Reading to next lowest 5 or 10 mmHg or expectation bias
- Examination
o Cuff too narrow
o Cuff not centered
o Cuff over clothing
o Elbow too low
o Cuff too loose
o Too short rest period
o Back unsupported
o Arm unsupported
o Too slow deflation
o Too fast deflation
o Parallax error
o Using phase IV (adult)
o Too rapid remeasure
o Noisy environs
o Bell vs. diaphragm
- Examinee
o "White coat" reaction
o Pain, anxiety
o Acute smoking
o Acute caffeine
o Acute ethanol ingestion
o Distended bladder
o Talking, signing
o Recent meal
o Menstrual phase
o Chronic caffeine ingestion
o Phenylephrine nasal spray
o Hour of day (during work hours)
o Room temperature


## SBP/DBP, mmHg

DBP (rare, huge)
DBP
Probably < 10
-8 to $+10 / 2$ to 8
4/3
5 to 50
6
Not stated
Varied estimates
6 to 10
1 to $7 / 5$ to 11
-1 to $+2 / 5$ to 6
DBP only
2 to 4
6 DBP
1/1
SBP
...
11 to $28 / 3$ to 15
May be large
6/5
11/5
8/8
15/10
7/8
-1 to $1 / 1$ to 4
...
...
...
...
...

SBP = systolic blood pressure
DBP = diastolic blood pressure
... = magnitude of effect not reported

## Appendix 2: Home Blood Pressure Units

| Brand | Model | Price | Accuracy <br> and <br> Convenience | Comfort | Score |
| :--- | :--- | :--- | :--- | :--- | :--- |
| CVS | BP3MV1- <br> 1ECVS | $\$ 66.00$ | Excellent for <br> both | Very good | 92 |
| Omron | 10 series <br> BP785 | $\$ 80.00$ | Excellent for <br> both | Very good | 89 |
| Rite Aid | Deluxe <br> Automatic <br> BP3ARI- <br> 4DRITE | $\$ 60.00$ | Excellent for <br> both | Good | 85 |
| iHealth | Doc BP3 | $\$ 100.00$ | Excellent for <br> accuracy <br> Good for <br> convenience | Very good | 84 |
| LifeSource <br> Advanced <br> One Step | UA-767PVL | $\$ 60.00$ | Excellent for <br> accuracy <br> Very good for <br> convenience | Very good | 81 |

Based on Consumer Reports, June 2013 (p.44).

Turn page for patient instructions.

## Home Blood Pressure Measurement

## The ABCDs



To begin, measure twice a day (at the same time), every day for 5-7 days.

## Achieve a calm state

- Make sure you are quiet and relaxed
- Sit calmly without talking for about 5 minutes
- Make sure your reading isn't affected by: caffeine, alcohol, exercise or smoking


## Body posture is important

- Sit in a chair with feet on the floor
- Legs should not be crossed
- Arm should be bare and should be supported at heart level


## Calibrate \& check equipment

- Use a properly calibrated and validated instrument
- Check the cuff size and fit


## Double check and document

- If blood pressure registers high, take two readings 5 minutes apart
- Confirm any elevated readings in the opposite arm
- Document your reading; write down number in log book


## Appendix 3: Working Up Specific Causes of Secondary Hypertension

(adapted from tables in Viera 2010)

| Signs/symptoms | Possible secondary hypertension cause | Diagnostic test options | Test Likelihood Ratios |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
| - Arm to leg systolic BP difference >20 <br> - Delayed/absent femoral pulses | Coarctation of the aorta | ```Magnetic resonance imaging (adults)``` | Echocardiography | 47.0 | 0.06 |
| - $\uparrow$ in serum creatinine $(\geq 0.5$ to 1 mg per dL [ 44.20 to $88.40 \mu \mathrm{~mol}$ per L]) after starting ACE inhibitor or angiotensin receptor blocker <br> - Renal bruit | Renal artery stenosis | Computed tomography angiography Magnetic resonance imaging with gadolinium contrast media Doppler ultrasonography of renal arteries | CT angiography MRI with gadolinium contrast media | $\begin{aligned} & 13.4 \\ & 13.9 \end{aligned}$ | $\begin{aligned} & 0.06 \\ & 0.03 \end{aligned}$ |
| - Bradycardia/tachycar dia <br> - Cold/heat intolerance <br> - Constipation/diarrhea <br> - Irregular, heavy, or absent menstrual cycle | Thyroid disorders | Thyroid-stimulating hormone |  |  |  |
| - Hypokalemia | Aldosteronism | Aldosterone/renin ratio | Aldosterone/renin ratio > 20* <br> Aldosterone/renin ratio > 30* | $\begin{gathered} 4.6 \\ 28.0 \end{gathered}$ | $\begin{aligned} & 0.27 \\ & 0.16 \end{aligned}$ |
| - Apneic events during sleep <br> - Daytime sleepiness <br> - Snoring | Obstructive sleep apnea | Polysomnography (sleep study) <br> Sleep Apnea Clinical Score with nighttime pulse oximetry | Overnight <br> polysomnography <br> Sleep Apnea Clinical Score with nighttime pulse oximetry | 5.2 | 0.25 |
| - Flushing or sweating <br> - Headaches <br> - Labile blood pressures <br> - Orthostatic hypotension <br> - Palpitations <br> - Syncope | Pheochromocytoma | 24-hour urinary fractionated metanephrines | 24-hour urinary total metanephrines Plasma free metanephrines | $\begin{aligned} & \hline 8.0 \\ & 5.5 \end{aligned}$ | $\begin{aligned} & 0.13 \\ & 0.01 \end{aligned}$ |
| - Buffalo hump <br> - Central obesity <br> - Moon facies <br> - Striae | Cushing syndrome | 24-hour urinary cortisol <br> Late-night <br> salivary cortisol Low-dose dexamethasone suppression | 24-hour urinary free cortisol Late-night salivary cortisol Low-dose dexamethasone suppression | $\begin{gathered} \hline 10.6 \\ 8.8 \\ 11.6 \end{gathered}$ | $\begin{aligned} & 0.16 \\ & 0.07 \\ & 0.09 \end{aligned}$ |

[^11]
## Appendix 4: Hypertension Drugs and the Laboratory

Most common diuretics used are the following.

## 1. Thiazide diuretics:

## Side effects in blood, serum or plasma

Amylase - ?
Bilirubin - normal
Cholesterol - ?
Coombs (Direct) - normal
CPK - normal
Glucose - ?
Phosphatase AK - normal
Potassium - ?
Prothrombin - normal
SGOT/SGPT - normal
Urea Nitrogen - ?
Uric acid

## Urine

Color - normal
Catecholamin - normal
Glucose - hyperglycemia
Cortisol - slight ?
All else - normal

## 2. Beta blockers - Inderal (Proprandol)

## Side-effects in blood, serum, plasma

Amylase - normal
Bilirubin - false ? (by SMA 12/60)
Cholesterol - normal
Coombs - normal
CPK - normal
Glucose - ?
Thyroxine - Total and free thyroxine ? by doses of 160 mg or greater serum Thriiodothyronine?
Phos Alk - normal
Potassium - normal
SGOT/SGPT - ?
Urea Nitrogen - ?
Uric Acid - normal

Urine
Color - normal
Catecholamines - false ?
All else - normal

## Appendix 5: Risk Factors and Serum Lipids

| Total Blood Cholesterol |  |
| :---: | :---: |
| < $180 \mathrm{mg} / \mathrm{dL}$ | Optimal (especially if you have multiple risks) |
| $<200 \mathrm{mg} / \mathrm{dL}$ | Desirable |
| 200 to $239 \mathrm{mg} / \mathrm{dL}$ | Borderline to high risk |
| > $240 \mathrm{mg} / \mathrm{dL}$ | High risk |
| Note: For every 1\% rise in total cholesterol above $180 \mathrm{mg} / \mathrm{dL}$, risk for MI goes up $2 \%$. |  |
| LDL-Cholesterol |  |
| < $70 \mathrm{mg} / \mathrm{dL}$ | Recommended for cardiac patients |
| $<130 \mathrm{mg} / \mathrm{dL}$ | Desirable |
| 130 to $159 \mathrm{mg} / \mathrm{dL}$ | Borderline to high risk |
| > $160 \mathrm{mg} / \mathrm{dL}$ | High risk |
| Note: This risk factor is independent of the total cholesterol level. For every $1 \mathrm{mg} / \mathrm{dL}$ rise in LDL, risk of cardiovascular events increases by $2 \%$. |  |
| HDL-Cholesterol |  |
| > $59 \mathrm{mg} / \mathrm{dL}$ | Protective |
| 45 to $59 \mathrm{mg} / \mathrm{dL}$ | Desirable |
| > $40 \mathrm{mg} / \mathrm{dL}$ | Recommended for cardiac patients |
| 35 to $44 \mathrm{mg} / \mathrm{dL}$ | Borderline to high risk |
| < $35 \mathrm{mg} / \mathrm{dL}$ | High risk |
| Note: This risk factor is independent of the total cholesterol level and the LDL-cholesterol level. For every $1 \mathrm{mg} / \mathrm{dL}$ rise in HDL, there is a decrease risk of cardiovascular events by $4 \%$. |  |
| Total Cholesterol / HDL ratio |  |
| < 5.0 | Desirable |
| 5.1 to 6.0 | Borderline to high risk |
| > 6.0 | High risk |
| Triglycerides |  |
| $<100 \mathrm{mg} / \mathrm{dL}$ | Cardiac patients |
| < $200 \mathrm{mg} / \mathrm{dL}$ | Desirable |
| 200 to $400 \mathrm{mg} / \mathrm{dL}$ | Borderline to high risk |
| > $400 \mathrm{mg} / \mathrm{dL}$ | High risk |
| Note: Levels above $250 \mathrm{mg} / \mathrm{dL}$ coupled with HDL below $40 \mathrm{mg} / \mathrm{dL}$ may double your risk of MI even if total cholesterol and LDL-cholesterol levels are acceptable (part of the so-called Syndrome X, often associated with abdominal obesity, elevated blood pressure, blood sugar and uric acid). |  |

## Appendix 6: CALORIE-DENSE FOODS AND ALTERNATIVES

Compiled by Jim Gerber, DC.

| Calorie-dense foods | Alternatives (emphasize natural foods) |
| :--- | :--- |
| Red meats - corned beef, prime rib, sausage, <br> ribs, lunchmeats, frankfurters | Lean beef (round, sirloin flank, tenderloin), wild <br> game, ham, Canadian bacon, pork tenderloin, <br> veal chops and roasts, 95+\% lean lunchmeat, <br> low-fat vegetarian meat substitutes |
| Poultry - fried chicken, frankfurters, duck, <br> goose | Skinless chicken, turkey, Cornish hen |
| Seafood - fried seafood, oil-pack tuna | Non-fried seafood, water-pack tuna |
| Dairy products - most cheeses, whole <br> milk/yogurt, regular and premium dairy <br> desserts | Cottage cheese, parmesan cheese, low/non-fat <br> cheeses/milk/yogurt/desserts or dairy alternatives <br> (soy, rice, etc.) |
| Eggs and egg dishes | Dishes prepared with egg whites only or low- <br> calorie egg substitutes |
| Fats - butter, margarine, mayonnaise, oil, <br> cream products, non-dairy creamer, rich <br> sauces/gravies/salad dressings, nuts, seeds, <br> peanut butter, olives, avocado, coconut | Low-calorie mayonnaise, spray oil for cooking, <br> low-fat/condensed non-fat milk, low-fat <br> sauces/gravies/salad dressings |
| Breakfast breads and cereals - doughnuts, <br> pastries, croissants, gourmet muffins, high-fat <br> cereals and granola, pancakes, waffles, <br> French toast | Toast/bagel/English muffin, low-fat muffins and <br> pastries, cooked cereals, low sugar/low fat <br> breakfast cereals and granola, low-fat/sugar <br> recipe pancakes and waffles |
| Lunch/dinner entrees - casseroles/noodle <br> dishes/stews/soups with meat/cheese/ <br> cream/eggs, many fast food sandwiches, <br> many Mexican/Asian/Italian dishes, fried foods | Tomato-based or other dishes without <br> meat/cheese/fat, low-calorie salad entrees, broth <br> soups, lean meat sandwiches w/o cheese, low-fat <br> international dishes, broiled/baked/steamed foods |
| Starchy snacks - fried chips, rich crackers, <br> regular popcorn, French fries, onion rings | Pretzels, bread sticks, low/non-fat crackers, chips <br> and popcorn |
| Sweet snacks - regular cookies, cakes, pies, <br> frozen desserts, granola bars, candy, soda <br> pop | Fresh fruit, flavored nonfat yogurt, nonfat frozen <br> desserts, sherbet/fruit ices, gelatin desserts, angel <br> food cake, animal crackers, low-fat fig/fruit bars, <br> low/nonfat cookies/cakes, sugarless candy, <br> diluted unsweetened fruit juices |
| Alcoholic beverages - beer, wine, wine <br> coolers, mixed drinks, liqueurs | Light beer, low-calorie non-alcoholic beverages |

## Appendix 7: The DASH Diet

| The DASH Diet <br> Note: the DASH diet improves insulin sensitivity as well as hypertension. |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Food Group | Daily Servings | 1 Serving Equals | Examples and Notes | Significance of each food group to the DASH Diet pattern |
| Grains \& grain products | 7-8 | 1 slice of bread <br> $1 / 2 \mathrm{C}$ dry cereal <br> $1 / 2 C$ cooked rice, pasta, or cereal | whole wheat breads, English muffin, pita bread, bagel, cereals, grits, oatmeal | major sources of energy and fiber |
| Vegetables | 4-5 | 1 C raw leafy vegetable $1 / 2 C$ cooked vegetable 6 oz vegetable juice | tomatoes, potatoes, carrots, peas, squash, broccoli, turnip greens, collards, kale, spinach, artichokes, beans, sweet potatoes | rich sources of potassium, magnesium, and fiber |
| Fruits | 4-5 | 6 oz fruit juice 1 medium fruit <br> $1 / 4 \mathrm{C}$ dried fruit <br> $1 / 2 \mathrm{C}$ fresh, frozen, or canned fruit | apricots, bananas, dates, grapes, oranges, orange juice, grapefruit, grapefruit juice, mangoes, melons, peaches, pineapples, prunes, raisins, strawberries, tangerines | important sources of potassium, magnesium, and fiber |
| Low fat or nonfat dairy foods | 2-3 | 8 oz milk 1 cup yogurt 1.5 oz cheese | skim or 1\% milk, skim or low fat buttermilk, nonfat or low fat yogurt, part skim mozzarella cheese, nonfat cheese | major sources of calcium and protein |
| Meats, poultry, fish | 2 or less | 3 oz cooked meats, poultry, or fish | select only lean; trim away visible fats; broil, roast, or boil, instead of frying; remove skin from poultry | rich sources of calcium and magnesium |
| Nuts | 1-2 | $\begin{aligned} & 1.5 \text { oz, } 1 / 3 \mathrm{C} \text { or } 2 \mathrm{Tbsp} \\ & \text { seeds } \\ & 1 / 2 \mathrm{C} \text { cooked legumes } \end{aligned}$ | almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils | rich sources of energy, magnesium, potassium, protein, and fiber |

## APPENDIX 7 CONTINUED...

| THE DASH DIET • | Sample Me | - bas | d on 2,000 calo | s/day |
| :---: | :---: | :---: | :---: | :---: |
| Food | Amount | Servings Provided | Total number of servings in 2,000 calories/day |  |
| BREAKFAST >>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>> |  |  |  |  |
| orange juice | 6 oz | 1 fruit |  |  |
| 1\% low fat milk | $8 \mathrm{oz} \mathrm{(1} \mathrm{C)}$ | 1 dairy | Food Group | Servings |
| corn flakes (with 1 tsp sugar) | 1 C | 2 grains | Grains | = 8 |
| banana | 1 medium | 1 fruit | Vegetables | $=4$ |
| whole wheat bread | 1 slice | 1 grain | Fruits | = 5 |
| (with 1 Tbsp jelly) |  |  | Dairy Foods | = 3 |
| soft margarine | 1 tsp | 1 fat | Meats, Poultry \& Fish | $=2$ |
|  |  |  | Nuts | = 1 |
| chicken salad | $3 / 4 \mathrm{C}$ | 1 poultry | Fats \& Oils | $=2.5$ |
| pita bread | 1/2 slice, large | 1 grain |  |  |
| raw vegetable medley: <br> - carrot \& celery sticks <br> - radishes <br> - loose-leaf lettuce | 3-4 sticks each <br> 2 <br> 2 leaves | 1 vegetable | Tips on Eating the DASH Way: |  |
|  |  |  | Start small. Make gradual changes in your eating habits. |  |
| part skim mozzarella cheese | 1.5 slice (1.5 oz) | 1 dairy | - Center your meal around carbohydrates, such as pasta, rice, beans, or vegetables. |  |
| 1\% low fat milk | 8 oz | 1 dairy |  |  |
| fruit cocktail in light syrup | 1/2 C | 1 fruit |  |  |
|  |  |  |  |  |
| herbed baked cod | 3 oz | 1 fish |  |  |
| scallion rice | 1 C | 2 grains | Treat meat as one p whole meal, instead | of the |
| steamed broccoli | $1 / 2 \mathrm{C}$ | 1 vegetable |  |  |
| stewed tomatoes | $1 / 2 \mathrm{C}$ | 1 vegetable |  |  |
| spinach salad: <br> - raw spinach <br> - cherry tomatoes <br> - cucumber | $\begin{array}{\|l} \hline 1 / 2 \mathrm{C} \\ 2 \\ 2 \text { slices } \\ \hline \end{array}$ | 1 vegetable | Use fruits or low fat, foods such as suga gelatin for desserts | ow-calorie <br> free <br> nd |
| light Italian salad dressing | 1 Tbsp | $1 / 2$ fat |  |  |
| whole wheat dinner roll | 1 small | 1 grain |  |  |
| soft margarine | 1 tsp | 1 fat |  |  |
| melon balls | $1 / 2 \mathrm{C}$ | 1 fruit |  |  |
| SNACKS ${ }^{\text {c }}$ (>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>>> |  |  |  |  |
| dried apricots | $1 \mathrm{oz}(1 / 4 \mathrm{C})$ | 1 fruit |  |  |
| mini-pretzels | $1 \mathrm{oz}(3 / 4 \mathrm{C})$ | 1 grain |  |  |
| mixed nuts | $1.5 \mathrm{oz}(1 / 3 \mathrm{C})$ | 1 nuts |  |  |
| diet ginger ale | 12 oz | 0 |  |  |

## Appendix 8: High-Sodium sources (UNLESS LABELED LOWSALT/SODIUM)

Fast foods: pizza, sandwiches, fried chicken, Mexican foods, Chinese foods
Prepared dishes: canned soups, casseroles, cheese dishes
Processed meats: ham, bacon, sausage, hot dogs, lunch meats/cold cuts, kosher meats
Grain products: chips, pretzels, crackers, popcorn, some breakfast cereals and side dishes
Vegetable products: pickled vegetables, olives, tomato sauces, tomato/vegetable juice drinks, some prepared vegetable dishes

Seasonings: soy sauce, monosodium glutamate, some gravies and sauces
Medications: antacids containing sodium

## Appendix 9: Diet Quality Questionnaire

List two typical examples of each of the following meals that the patient regularly eats. If the patient tends to skip any of these meals, leave them blank. Indicate whether a food was fried, whether it was made with whole or refined grain, or when a food is a low-fat, nonfat, or sugar-free version of that food. Remember to include sauces, dressings, spreads, gravies, and other extras.

| Meal | Example \#1 | Example \#2 |
| :--- | :---: | :---: |
| Breakfast |  |  |
| Mid-morning snack |  |  |
| Lunch |  |  |
| Mid-afternoon snack |  |  |
| Dinner |  |  |
| Late-evening snack |  |  |

In addition to listing the typical meals above, answer the following questions to help understand some of the details of the meals that were listed. After obtaining these answers, go back to the table above and fill in missing details about the listed foods.

Indicate how many times in a typical day the patient eats a serving of each of the following foods:
$\qquad$ Grain products, breads, cereals, etc. made primarily with a whole grain ingredient (i.e., whole wheat flour, bran, brown rice, oats)

Grain products, breads, cereals, etc. made primarily with a refined grain ingredient. For example, white flour, white bread, "wheat" bread not described as "whole wheat," white rice, regular pasta, regular pizza. Tell patient to check labels if unsure and assume unlabeled grain products are primarily refined.
$\qquad$ A food made with vegetable oils that were not partially-hydrogenated. These might include olive, canola, soy, corn, sunflower, peanut, other vegetable oils, and condiments such as salad dressings and sauces containing these oils; also include avocado.

A food made with butter, cream, or vegetable oils that were partially-hydrogenated. Tell patient to check labels if unsure and assume that unlabeled fried and commercially baked foods such as chips, baked snacks, breakfast pastries and other sweets used butter or partially-hydrogenated oil.
$\qquad$ Butter, cream, half and half, coffee creamer, or condiments (spreads, sauces, dressings, gravies) containing partially-hydrogenated vegetable oil or animal fats such as butter, cream, or cheese
$\qquad$ A sweet-tasting food or beverage with real sugar, honey, corn sweetener, fruit juice concentrate, or another calorie-containing sweetener.
$\qquad$ Does the patient take any supplements containing vitamins and/or minerals? If so, list them here:
$\qquad$ Does the patient drink any type of alcoholic beverage? If so, estimate how many drinks per week:

## Analysis of Diet Quality

For each column of example meals on the previous page, indicate the number of times (frequency) foods in each category are consumed in a day. Some foods may belong in more than one category, in which case all categories should be indicated. Examples: regular crust pizza with tomato sauce and cheese would be counted as Dairy, Unhealthy Fat, and Refined Carbohydrate; broiled salmon would be counted as Fish and Healthy Oils; granola-type health bar would be counted as Whole Grain, Fortified Food, and Oil or Fat according to content.

## Typical Daily FrequencyCategory (recommended frequency)

## Example 1 Example 2

Whole Grain Foods (at most meals)
Healthy Oils (non-hydrogenated plant oils, fish oil) (no restriction)

## Suggestions for Changes to Improve Diet Quality:

## Appendix 10: Low Glycemic Index Diet for Glucose INTOLERANCE DISORDERS

This chart should be followed when choosing carbohydrate-containing foods to support normal insulin secretion and sensitivity.*


* Individual responses to foods may vary.
** If these foods must be used, they should be combined with low glycemic index foods or protein foods.


## Appendix 11: Sources of Dietary Potassium

| Description | Measure | Potassium (mg) <br> Per Measure | Sodium, (mg) <br> Per Measure |
| :---: | :---: | :---: | :---: |
| Radishes, oriental, dried | 1.0 cup | 4053 | 322 |
| Potatoes, mashed, dehydrated, granules with milk, dry form | 1.0 cup | 3696 | 164 |
| Beans, white, mature seeds, raw | 1.0 cup | 3626 | 32 |
| Soybeans, mature seeds, raw | 1.0 cup | 3342 | 4 |
| Beans, small white, mature seeds, raw | 1.0 cup | 3315 | 26 |
| Seeds, breadnut tree seeds, dried | 1.0 cup | 3218 | 85 |
| Lima beans, large, mature seeds, raw | 1.0 cup | 3069 | 32 |
| Soy meal, defatted, raw | 1.0 cup | 3038 | 4 |
| Beans, black, mature seeds, raw | 1.0 cup | 2877 | 10 |
| Lima beans, thin seeded (baby), mature seeds, raw | 1.0 cup | 2834 | 26 |
| Beans, kidney, California red, mature seeds, raw | 1.0 cup | 2742 | 20 |
| Beans, pinto, mature seeds, raw | 1.0 cup | 2688 | 23 |
| Beans, kidney, all types, mature seeds, raw | 1.0 cup | 2587 | 44 |
| Beans, great northern, mature seeds, raw | 1.0 cup | 2538 | 26 |
| Soybeans, mature seeds, roasted, no salt added | 1.0 cup | 2528 | 7 |
| Beans, kidney, red, mature seeds, raw | 1.0 cup | 2501 | 22 |
| Beans, kidney, royal red, mature seeds, raw | 1.0 cup | 2477 | 24 |
| Beans, navy, mature seeds, raw | 1.0 cup | 2465 | 10 |
| Beans, French, mature seeds, raw | 1.0 cup | 2421 | 33 |
| Cowpeas, catjang, mature seeds, raw | 1.0 cup | 2296 | 97 |
| Soy flour, low-fat, crude protein basis ( $\mathrm{N} \times 6.25$ ) | 1.0 cup, stirred | 2262 | 16 |
| Apricots, dehydrated (low-moisture), sulfured, uncooked | 1.0 cup | 2202 | 15 |
| Peas, split, mature seeds, raw | 1.0 cup | 1933 | 30 |
| Orange juice, frozen concentrate, unsweetened, undiluted | 1.0 cup | 1914 | 9 |
| Carrot, dehydrated | 1.0 cup | 1880 | 204 |
| Tomatoes, sun-dried | 1.0 cup | 1851 | 133 |
| Lentils, raw | 1.0 cup | 1834 | 12 |
| Apricots, dehydrated (low-moisture), sulfured, stewed | 1.0 cup | 1813 | 12 |
| Chickpeas (garbanzo beans, bengal gram), mature seeds, raw | 1.0 cup | 1750 | 48 |
| Potatoes, Russet, flesh and skin, baked | 1.0 potato large (3" to 4-1/4" dia.) | 1644 | 42 |
| Potatoes, red, flesh and skin, baked | 1.0 potato large ( $3^{\prime \prime}$ to 4-1/4" dia. | 1630 | 36 |
| Potatoes, white, flesh and skin, baked | 1.0 potato large ( $3^{\prime \prime}$ to 4-1/4" dia.) | 1627 | 21 |
| Peaches, dried, sulfured, uncooked | 1.0 cup, halves | 1594 | 11 |
| Broadbeans (fava beans), mature seeds, raw | 1.0 cup | 1593 | 20 |

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[^0]:    ${ }^{1}$ J ames PA, MD, Oparil S, MD, Carter BL, PharmD, et al. 2014 Evidence-based guideline for the management of high blood pressure in adults - report from the panel members appointed to the eighth Joint National Committee (JNC 8). J AMA 2013 Dec 18. doi: 10.1001/jama.2013.284427
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[^1]:    * ASH-ISH \& JNC 7 suggest 4 readings so as not to delay a diagnosis especially in an environment where patients see their PCP infrequently. A 2011 study, however, suggested that it may take at least 10 measurements to classify that a patient is normotensive or hypertensive with 80\% or more certainty-compared to 5 home measurements. (Asayama 2013)
    Technique recommendations are based on the JCN 7, European and US Prevention Services reports.

[^2]:    * Data derived from a single RCT or large non-randomized studies.

[^3]:    * Note that often drug monotherapy is not sufficient to achieve treatment goals and it is common for patients to be prescribed more than one anti-hypertension medication.

[^4]:    A grade A JNC 8 recommendation suggests that "there is high certainty based on evidence that the net benefit is substantial."
    High quality JNC 8 evidence is defined as well designed, well -executed RCTs, well conducted meta-analyses of such RCTs with high confidence in the estimate of therapeutic effect.

[^5]:    * "The majority embraced the view that in the absence of definitive evidence, increasing the SBP goal was the optimum approach. We,

[^6]:    * To compute a patient's basal caloric requirements, a validated on-line calculator can be used (see http://fnic.nal.usda.gov/fnic/interactiveDRI/. However, more commonly, a simple alternative is to inspect the current diet for high-fat foods and recommend reducing them or using lower-fat alternatives.

[^7]:    ** A recommendation of I indicates that the benefits greatly outweigh the risks and the guidelines panel indicates that the treatment SHOULD be followed.

[^8]:    ${ }^{\dagger \dagger}$ Sodium chloride or table salt is approximately $40 \%$ sodium. To convert salt grams into sodium grams, divide by 2.5. In terms of teaspoons:

[^9]:    $\ddagger \ddagger$ A level lla recommendation indicates that the benefits outweigh the risks but that additional studies are needed with more focused objectives. The guidelines panel suggests that based on current evidence it is REASONABLE to administer the treatment. The Institute of Medicine, on the other hand, concluded that there was insufficient evidence to recommend for/ against reductions to $<2,300 \mathrm{~g} / \mathrm{dy}$.

[^10]:    §§ If the patient has a major cardiac risk factor, like smoking, use $75 \%$ instead of $85 \%$. (Subtract $10 \%$ for every major cardiac risk factor, all the way down to $55 \%$.)

[^11]:    *     - When plasma aldosterone is reported in ng per dL and plasma renin activity is reported in ng per mL per hour and accompanied by an aldosterone level greater than 15 ng per dL ( 416.10 pmol per L).

