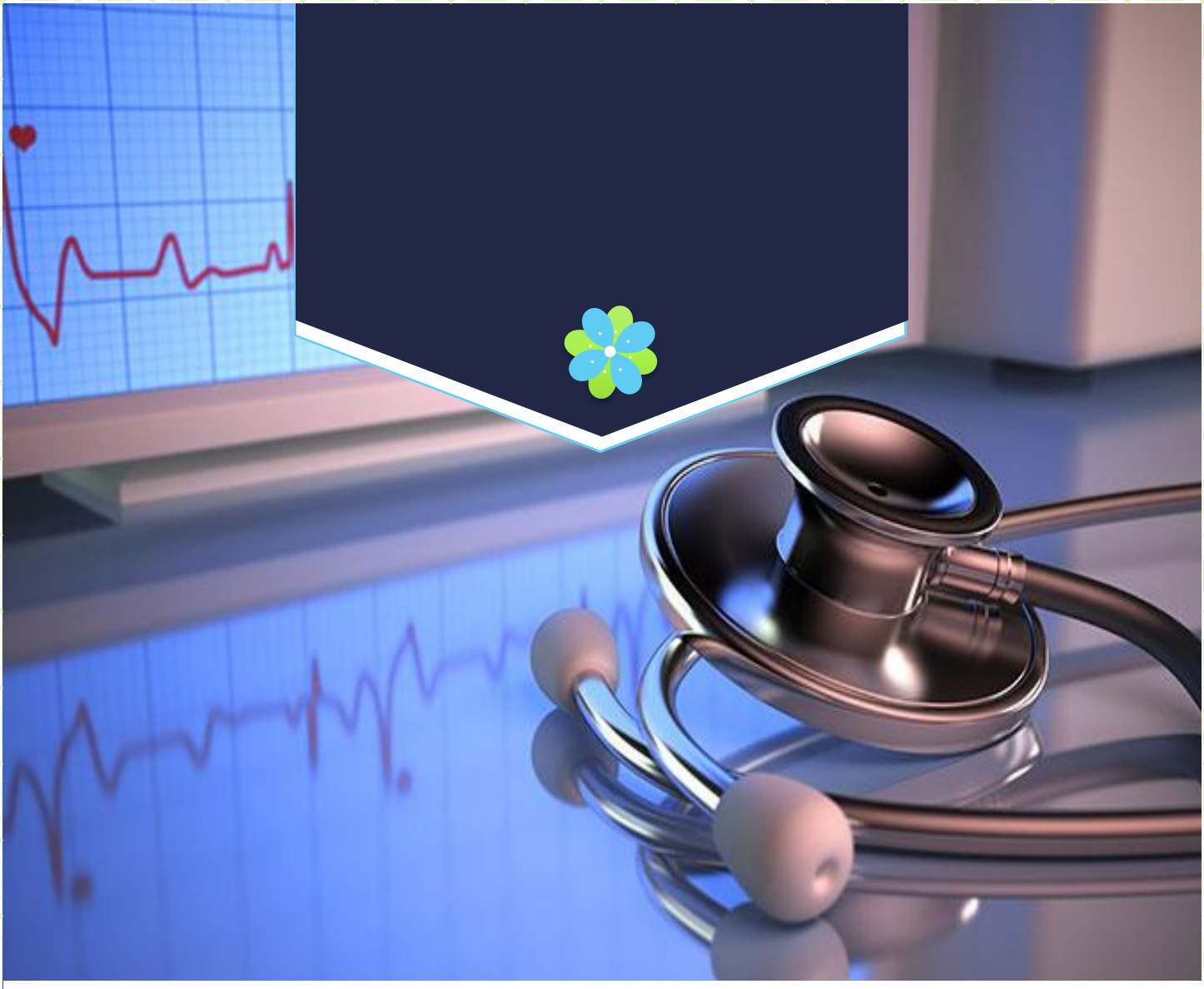

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WHAT IS FAMILY MEDICINE

- **The specialty of family medicine was created in 1969 to fulfill the generalist function in medicine, which suffered with the growth of sub-specialization after World War II.**
- **Family practice is the medical specialty that provides continuing and comprehensive health care in a personalized manner to all ages and families regardless of the presence of disease or nature of the presenting complaint.**

WHY FAMILY MEDICINE?

1. **The recent changes in medicine.**
2. **The growth of specialization.**
3. **The fragmentation of the health care delivery system.**
4. **The social changes.**
5. **The appearance of a new pattern of illness.**
6. **The need for better doctor-patient relationship.**
7. **The high cost of inpatient care.**
8. **The limitation of resources.**

FAMILY MEDICINE PRINCIPLES

1) Family physicians do not just treat patient, they care for people. The care of the patient **(the whole patient)** is the primary goal
2) Family physicians provide **continuous health care**, the care is not terminated by cure of illness, the end of treatment or the incurability of an illness.
3) Family physician provides **comprehensive health care** and is available for any health problem in a person of either sex and of any age.
4) Family physician attaches importance to the **subjective aspects** of medicine.
5) Family physician is a manager of resources.

- ***Family physician must often diagnose what things are not rather than what they are.***

6) Family physician provides cost-effective health care
7) Family physician sees every contact with patients as an opportunity for **prevention or health education**
8) Family physician sees a patient at the office, in their houses and in the hospital.
9) Family physician develops special relationship with the patient as a result of both duration and intensity of care thus achieving patient satisfaction.
10) Family physician sees himself as part of a community wide networks of supportive and health care agencies.

THIS WILL RESULT IN :

- ✓ ***Continuing, comprehensive, personalized care***
- ✓ ***Early detection and management of illness***
- ✓ ***Prevention of diseases and maintenance of health***
- ✓ ***Improving quality of primary care.***

THE SKILLS OF FAMILY PHYSICIAN

1) **The solution of undifferentiated problems** in the context of continuing relationship with family. The symptoms present tend to

be unorganized and undifferentiated while those encountered in hospital tend to be medicalized and more differentiated.

2) **Preventive Skills**: The identification of risks & early deviation from normality who are known to physician.

3) **Therapeutic Skills**: The aim of doctor – patient relationship is to maximize the effectiveness of all kinds of therapy.

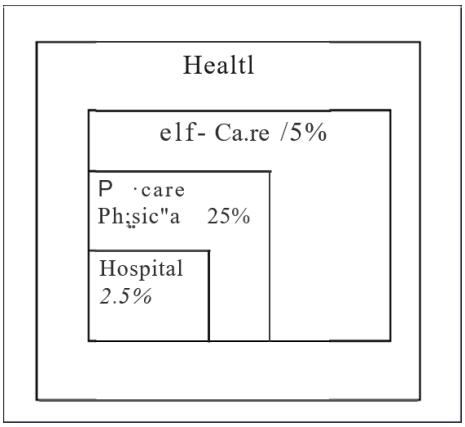
4) **Resource management skills**: employment of resources of the community and health care system for the benefit of the patient. This includes the skills of management, consultation & referral.

PRIMARY HEALTH CARE

- The quality of health care in any country is associated with primary care performance.
- Primary health care is an essential part of every health care system in the world.
- The higher the primary care physician – to – population ratio the better most health care outcomes are.
- Primary care has to be the keystone base in an integrated system. It has to be part of the national, social and economic structure

IT SHOULD BE :

1. **Available** (when patient has urgent or chronic complaints)
2. **Accessible** (Geographically)
3. **Affordable** (financially)
4. **Appreciated** and understood
5. **Comprehensive:** Provides broad range of services including acute and chronic diseases management, prevention and psychological management, Care in clinic, hospital and nursing home or via telephone.
6. **Coordinated:** Aware of pt entire list of problems and is central source of information about patient care. Control pt. referral to specialists, manage care delivered by team of health care workers and translate specially advice for pt.
7. **Continuous:** Develop long term relationships with pt. maintain longitudinal record of pt. problems and promote health over the long term.
8. **Accountable:** Responsible for broad range of health issues and outcome and is pt. advocates in health care system. Educate pt. about treatment outcome, prognosis and understand pt. preferences.



Primary health care priorities:

BREAKING BAD NEWS

When breaking bad news, you should follow the SPIKES protocol.

SPIKES PROTOCOL

S – SETTING:

Ensure you are in a comfortable confidential room where you will not be interrupted

P - PERCEPTION

- **Events that have led up to now**
- **Ask them what they already know/expect**
- **Spend some time trying to get them to say what the diagnosis is**

- *"Could you tell me what's happened so far?"*
- *"Do you have any ideas as to what the problem might be?"*
- *"Is there anything you have been worried about?"*

I - INVITATION

CHECK IF THE PATIENT

1. **Wants to know the result now**
2. **Would like a family member to be present**

"I do have the result here today, would you like me to explain it to you now?" "Would you prefer if a family member/ friend is present?"

K - KNOWLEDGE

GIVING THE DIAGNOSIS:

- Build up to the result – give a warning shot
- Chunk the diagnosis (stepped approach)
- After every statement you say, pause & wait for the patient to ask the next question (silence is the best thing at this point – there are a million thoughts going around in their head)
- If the silence is very awkward, you can ask a question about how they are feeling (see E)

Explaining:

- DO NOT launch in to explain anything – during K and afterwards, the patient must lead the consult – only answer questions they ask (they will not remember anything else you say)
- Chunk & check any requested explanations

"As you know, we took a biopsy and, unfortunately, the results are not as we hoped." PAUSE & WAIT "I'm afraid / unfortunately / I'm sorry to tell you it is a tumour"

E – EMOTIONS AND EMPATHY

- Acknowledge and reflect their emotions back (including body language)
- Don't try to solve their problems or reassure them, just listen and summarise/ bounce their concerns back to them and expand on them (it shows you are listening and conveys empathy)
- If there is a lot of silence, you can ask about their emotions

"I can see this news was a huge shock" PAUSE & WAIT

"You appear very anxious" PAUSE & WAIT

"So you've told me that you biggest worries are telling your children and losing your hair?" "How are you feeling about hearing the news?"

"You're very quiet, can I ask what's going though your mind?" "What's upsetting you the most?"

2. Empathise

3. Explore it: find the content of the cue e.g. *"would it be OK if I asked more about that?"*

4. ONLY reply/ try to solve the problem if you have to – most the time, DON'T!
e.g. "I'm dying, what does it matter?"

"You're dying? What's going through your mind when you say that phrase?" PAUSE & WAIT

"I'm really sorry about that. I can't imagine what it's like to feel like that" MASSIVE PAUSE & WAIT FOR PATIENT

S – STRATEGY AND SUMMARY

- Agree on a plan
- Summarise concerns
- Ask how they are left feeling

COMMUNICATING DURING THE CONSULTATION

BREAKING THE NEWS

Stepped approach (you need permission to move on from each step):

1. "I'm afraid it's not good news Julie" PAUSE & WAIT FOR PATIENT TO ASK

2. "Unfortunately the lump is a problem" PAUSE & WAIT FOR PATIENT TO ASK

3. "Yes, I'm so sorry to have to tell you, it is a cancer" PAUSE & WAIT (FOR AGES!) FOR PATIENT TO ASK

☐ Next: Don't say anything. It's difficult but the most effective way to communicate from now onwards is not to say anything until asked. If it really gets awkward, reflect the fact that they are quiet/shocked, pause, then as what's going through their mind.

DON'T START GIVING INFORMATION UNTIL IT IS REQUESTED

- ❑ Patients have concerns in their head and therefore won't listen to anything else you say. You need to get the concerns out first.
- ❑ Prompt if need to e.g. "you're very quiet, can I ask what's going though your mind?"
- ❑ Summarise all concerns back to them and expand on them as above

RESPONDING TO CUES/ QUESTIONS

• Cue = verbal/non-verbal negative feeling (tip of the iceburg)

• Dealing with a cue

1. Bounce it back (you must show you heard it)

APPROACH TO FATIGUE

CASE

- A 23 year old male patient , medically free, university student , presented to the clinic with fatigue of 6 month duration , increasing in the last 2 months .
- He described his fatigue that he is no more able to do things that he used to do before such as going to the gym , going out with his friends.
- prevalence in population-based surveys in Britain and the United States of between 6.0 and 7.5%
- A cross-sectional survey of United States workers found the *two-week period prevalence of fatigue to be 38 %* , with an estimated annual cost to employers exceeding $136 billion in lost productive work time.
- 21 to 33 % of patients seeking attention in primary care settings report significant fatigue.

INTRODUCTION

Fatigue is a common complaint, with a prevalence in population-based surveys in Britain and the United States of between 6.0 and 7.5 percent. A cross-sectional survey of US workers found the two week period prevalence of fatigue to be 38 percent, with an estimated annual cost to employers exceeding $136 billion in lost productive work time . In addition, an

Major causes of chronic fatigue

Psychologic	**Infectious**
Depression	Endocarditis
Anxiety	Tuberculosis
Somatization disorder	Mononucleosis
Malnutrition or drug addiction	Hepatitis
Pharmacologic	Parasitic disease
Hypnotics	HIV infection
Antihypertensives	Cytomegalovirus
Antidepressants	**Cardiopulmonary**
Drug abuse and drug withdrawal	Chronic heart failure
Endocrine-metabolic	Chronic obstructive pulmonary disease
Hypothyroidism	**Connective tissue disease**
Diabetes mellitus	Rheumatoid disease
Apathetic hyperthyroidism	**Disturbed sleep**
Pituitary insufficiency	Sleep apnea
Hypercalcemia	Esophageal reflux
Adrenal insufficiency	Allergic rhinitis
Chronic renal failure	Psychologic causes (see above)
Hepatic failure	**Idiopathic (diagnosis by exclusion)**
Neoplastic-hematologic	Idiopathic chronic fatigue
Occult malignancy	Chronic fatigue syndrome
Severe anemia	Fibromyalgia

estimated 21 to 33 percent of patients seeking attention in primary care settings report significant fatigue.

DEFINITION

Clinical fatigue incorporates three components, present to variable degrees in individual patients: **difficulty or inability to initiate activity** (perception of generalized weakness); **reduced capacity to maintain activity** (easy fatiguability); and **difficulty with concentration, memory, and emotional stability** (mental fatigue).

- Fatigue should be distinguished from somnolence, dyspnea, and muscle weakness, although these symptoms may also be associated with fatigue.

Three categories are used in referring to fatigue, based upon the duration of symptoms:

- **Recent fatigue —** symptoms lasting less than one month
- **Prolonged fatigue —** symptoms lasting for more than one month
- **Chronic fatigue** — symptoms lasting over six months, but does not necessarily imply the presence of the chronic fatigue syndrome

Epidemiology and etiology

Fatigue as a major symptom is found in all populations and is associated with multiple factors (table 1). The prevalence of fatigue is generally higher in women than in men. Females comprise 75 percent or more of most series of patients with chronic fatigue syndrome (CFS), also known as systemic exertion intolerance disease (SEID).

- Medical or psychiatric diagnoses can explain fatigue in approximately two-thirds of patients with complaints of chronic fatigue.
- Psychiatric illness is present in 60 to 80 percent of patients with chronic fatigue .
- In one study, for example, a psychiatric diagnosis was found in 74 percent of over 400 patients who presented to a chronic fatigue clinic with at least one month of fatigue.
- The three major psychiatric illnesses were major depression (58%), panic disorder (14 %), and somatization disorder (10 %).
- There is debate, however, whether depression in individual patients is the cause or consequence of symptoms of chronic fatigue.
- Drugs are another important cause of chronic fatigue symptom, Drugs causing fatigue:
  1) Hypnotics
  2) muscle relaxants
  3) antidepressants
  4) first generation antihistamines
  5) beta blockers
  6) opioids

Chronic fatigue syndrome/ systemic exertion intolerance disease (CFS/SEID)

- Chronic fatigue syndrome (CFS), also known as systemic exertion intolerance disease (SEID), It is important to distinguish between the chronic fatigue syndrome (CFS/SEID), an uncommon cause of chronic fatigue symptoms, and idiopathic chronic fatigue.

2015 IOM diagnostic criteria for CFS/SEID

Diagnosis requires that the patient have the following three symptoms:
1. A substantial reduction or impairment in the ability to engage in pre-illness levels of occupational, educational, social, or personal activities that persists for more than six months and is accompanied by fatigue, which is often profound, is of new or definite onset (not lifelong), is not the result of ongoing excessive exertion, and is not substantially alleviated by rest; 2. Post-exertional malaise;* and 3. Unrefreshing sleep*
At least one of the two following manifestations is also required:
1. Cognitive impairment*; or 2. Orthostatic intolerance±

CFS/SEID: chronic fatigue syndrome/systemic exertion intolerance disease.
* Frequency and severity of symptoms should be assessed. The diagnosis of CFS/SEID should be questioned if patients do not have these symptoms at least half of the time with moderate, substantial, or severe intensity.
± Onset of symptoms when standing upright that are improved by lying back down.

- In 2015, the Institute of Medicine (IOM) redefined the diagnostic criteria for CFS/SEID (table) and suggested the name change to systemic exertion intolerance disease . The IOM diagnostic criteria focus on the most specific features of the disease. As with previous definitions, symptoms should be present for at least six months and have moderate, substantial, or severe intensity at least one-half of the time. Other criteria include: post-exertional malaise, sleep problems, cognitive impairment, and orthostatic-related symptoms.
- CFS/SEID represents a small subset of those who complain of chronic fatigue, accounting for 1 to 9 % of patients in a population with fatigue of at least six months' duration.

EVALUATION

The history is the most important component of the evaluation of chronic fatigue. The physical examination and laboratory studies provide supporting data.

HISTORY

- Fatigue that is due to an underlying medical or psychiatric disorder usually presents as one of **several reported symptoms**.
- A specific etiology for fatigue is found less frequently when fatigue itself is the principal concern, and the patient presents with few or no other symptoms.
- The clinician should rely upon **open-ended questions**, encouraging the patient to describe the fatigue in his or her own words. Questions and comments such as "What do you mean by fatigue?" or "Please describe what you mean".
- Patients with organ-based medical illness often associate their **fatigue with activities** they are unable to complete. In contrast, patients with fatigue that is not organ-based are tired all the time; their **fatigue is not necessarily related to exertion, nor does it improve with rest**.

THE HISTORY SHOULD DETERMINE THE SEVERITY AND TEMPORAL PATTERN OF FATIGUE AS FOLLOWS:

1) **Onset -** abrupt or gradual, related to event or illness?
2) **Course -** stable, improving or worsening?
3) **Duration and daily pattern**
4) **Factors that alleviate or exacerbate symptoms**
5) **Impact on daily life** - ability to work, socialize, participate in family activities
6) **Accommodations** that patient/family has made to adjust to fatigue symptoms

- Symptoms suggesting underlying occult medical illness should be explored in a detailed review of systems, including presence of weight loss or night sweats. The history should also include questions screening for psychiatric disorders (particularly depression, anxiety disorders, somatoform disorders, and substance abuse). Patients who are victims of domestic violence may present with symptoms of fatigue.

- At least 50% of patients with chronic fatigue, in a large primary study of fatigue in Britain, attributed their fatigue to mainly psychological causes.

- The quantity and quality of the patient's sleep should be assessed to determine whether or not sleep improves the patient's symptoms. Such improvement may suggest a primary sleep disorder or disturbed sleep as an etiology for the patient's fatigue.

- A thorough evaluation of medications, both prescribed and over the counter, should be undertaken. Recreational drug use, including alcohol, should be carefully explored in any patient with fatigue.

LABORATORY STUDIES

- Extensive laboratory evaluation in the absence of a positive history or physical examination are of little diagnostic utility in the evaluation of the fatigued patient .
- As an example, in a prospective study of 100 adults with the chief complaint of fatigue for at least one month, laboratory studies clarified the cause of fatigue in only 5 percent .

Laboratory Testing for Patients with Unexplained Fatigue

*TEST**	*POSSIBLE CONDITIONS*	*COMMENTS*
Complete blood count	Anemia	Should be performed in most patients with a two-week history of fatigue; results change management in 5 percent of patients[12]
Erythrocyte sedimentation rate	Inflammatory state	
Chemistry panel	Liver disease, renal failure, protein malnutrition	
Thyroid function tests	Hypothyroidism	
Human immunodeficiency virus antibodies	Chronic infection, if not previously tested	
Pregnancy test, if indicated	Pregnancy, breathlessness due to progestins	
Chest radiography	Adenopathy, cancer	Rarely useful; consider only if indicated by physical findings or abnormal baseline blood test results
Tuberculin skin test	Tuberculosis, chronic infection	
Electrocardiography	Congestive heart failure, arrhythmia	
Pulmonary function tests	Chronic obstructive pulmonary disease, cancer	
Toxicology screen	Substance abuse	
Lyme titers	Chronic Lyme disease	
Rapid plasma reagin	Syphilis infection	
Brain magnetic resonance imaging	Multiple sclerosis	
Echocardiography	Valvular heart disease, congestive heart failure	
Specialized blood testing (e.g., ferritin, iron, vitamin B_{12}, and folate levels; iron-binding capacity; direct antiglobulin test)	Iron deficiency, Addison disease, celiac disease, myasthenia gravis, poisoning	

PHYSICAL EXAMINATION

The physical examination is important to exclude some specific causes of fatigue and also helps to establish the doctor-patient relationship, assuring the patient that his or her complaint is a concern worth investigating. The physical examination should note:

1) **General appearance**: level of alertness, psychomotor agitation or retardation, grooming
2) **Presence of lymphadenopathy**
3) **Evidence of thyroid disease**: goiter, thyroid nodule, ophthalmologic changes .
4) **Cardiopulmonary examination**: signs of congestive heart failure and chronic lung disease
5) **Neurologic examination**: muscle bulk, tone, and strength; deep tendon reflexes; sensory and cranial nerve evaluation.

Reasonable initial laboratory studies to obtain include:

1) Complete blood count with differential
2) Erythrocyte sedimentation rate, ferritin
3) Chemistry screen (including electrolytes, glucose, renal and liver function tests)
4) Thyroid stimulating hormone
5) Creatine kinase, if pain or muscle weakness present
6) Testing for latent tuberculosis should be considered if appropriate based upon the history and risk factors.
7) ESR >> older patients with symptoms consistent with polymyalgia rheumatica or giant cell (temporal) arteritis.
8) screening for occult Hepatitis C viral infection for those born (1945-1965)
9) Screening for human immunodeficiency virus (HIV) is recommended for all adults.

- ❑ HIV testing and PPD placement should be considered if appropriate based upon the history.
- ❑ In the absence of suggestive history or symptoms, we do not suggest routine testing for infection (eg, Epstein-Barr virus [EBV], cytomegalovirus [CMV], or Lyme titers), immunological deficiency (eg, immunoglobulins), inflammatory disease (eg, antinuclear antibodies [ANA] or rheumatoid factor), vitamin deficiencies, or antibody studies for celiac disease.

Physiologic fatigue

- ❖ Physiologic fatigue is initiated by inadequate rest, physical effort, or mental strain unrelated to an underlying medical condition. Diminished motivation and boredom also play a role. Physiologic fatigue is most common in adolescents and older persons.
- ❖ During intense training, well-conditioned athletes occasionally misinterpret fatigue as illness or depression. Conversely, fatigue and depression can emerge in a physically fit athlete after as little as one week with no exercise.

MANAGEMENT

- ❖ Adequate sleep (i.e., generally seven to eight hours per night for adults) decreases tension and improves mood.
- ❖ Patients should be instructed to restructure their daily activities to get the sleep they need, and to practice good sleep hygiene.

GOOD SLEEP HYGIENE MEASURES:

- ➢ maintaining a regular morning rising time
- ➢ increasing activity level in the afternoon
- ➢ avoiding exercise in the evening or before bedtime
- ➢ increasing daytime exposure to bright light
- ➢ taking a hot bath within the two hours before bedtime
- ➢ avoiding caffeine, nicotine, alcohol, and excessive food or fluid intake in the evening
- ➢ using the bedroom only for sleep
- ➢ practicing a bedtime routine that includes minimizing light and noise exposure and turning off the television.
- ➢ Naps may help, but should be limited to less than one hour in the early afternoon.
- ➢ Stimulants improve short-term performance (caffeine, modafinil)
- ➢ Physical fitness also improves energy levels. One study showed that truck drivers who engaged in 30-minute exercise sessions more than once a week had fewer traffic incidents. Another study showed that 10 weeks of supervised exercise increased energy levels among persons with fatigue, regardless of the underlying cause.

TREATMENT

□ Treat underlying cause

□ Systematic reviews of CFS/SEID have determined effectiveness for only two treatments: cognitive behavioral therapy (CBT) and graded exercise therapy (GET)

□ THE DOCTOR-PATIENT RELATIONSHIP IS OF PROFOUND IMPORTANCE

The physician should acknowledge the patient's complaints as real and potentially debilitating.
The patient should have confidence that the physician will take a rational, stepwise approach to the evaluation, and that the physician will act as a guide in establishing therapeutic goals.

THESE GOALS SHOULD INCLUDE:

A. Accomplishing the activities of daily living
B. Returning to work
C. Maintaining interpersonal relationships

D. Performing some form of daily exercise

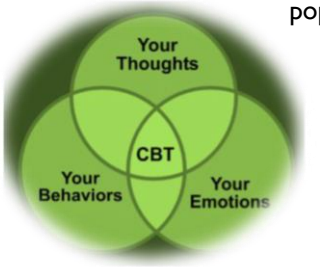
- Brief regularly scheduled appointments can be used to monitor progress in these areas and are preferred to having the patient being seen on an "as needed" basis.

ANTIDEPRESSANTS

☐ A trial of antidepressant drugs (eg, SSRI OR SNRI) should be offered to patients whose illness has features of depression, regardless of whether strict criteria for depression have been met.

COGNITIVE BEHAVIORAL THERAPY

☐ is effective in patients with CFS/SEID and may be useful in those with idiopathic chronic fatigue.



☐ A series of one-hour sessions designed to alter beliefs and behaviors that might delay recovery.

CBT COMPONENTS INCLUDE:

1) explanation of the model for chronic fatigue
2) challenging beliefs and awareness of fatigue and reorienting these beliefs
3) achievement of physical activity goals and other personal activity goals
4) helping the patient attain control over symptoms.

GRADED EXERCISE THERAPY

☐ GET is based on a physiological model of deconditioning.

☐ GET does not address cognition.

☐ improvement >> 55%, CBT >> 70 %.

OTHER MEASURES

Other measures that may be useful include:

- Provision of general sleep hygiene advice and discouraging over-sleeping
- Provision of patient education brochures and other materials, discussion of various aspects of chronic fatigue, and referral to support groups
- Iron therapy in nonanemic patients with low serum ferritin may improve symptoms of fatigue

PROGNOSIS

☐ In both (CFS) and idiopathic chronic fatigue are not generally favorable for full return to premorbid status.

☐ No resulted organ failure or death.

☐ Mortality from suicide was higher than the general population in patients with chronic fatigue, but not CFS/SEID

POOR PROGNOSIS IN:

1) older age, more chronic illness, a comorbid psychiatric disorder, and a firm belief that physical causes were responsible for the fatigue.
2) More than eight medically unexplained physical symptoms (excluding symptoms in the case criteria for CFS/SEID)
3) A lifetime history of dysthymic disorder
4) More than 1.5 years of chronic fatigue
5) less than 16 years of formal education
6) Age exceeding 38 years at presentation

Summary and recommendations

- Clinical fatigue (generalized weakness, easy fatiguability, and mental fatigue) should be distinguished from somnolence, dyspnea, and muscle weakness. Chronic fatigue refers to symptoms lasting over six months, but does not imply the presence of the chronic fatigue syndrome.

- Fatigue is related to medical or psychiatric diagnosis in about two-thirds of patients with chronic symptoms .Chronic fatigue syndrome (CFS) with a defined set of criteria is found in less than 10 percent of patients with chronic fatigue.

- The history is the most important part of the evaluation for patients with chronic fatigue. Assessment should focus on the temporal features of fatigue, its impact on

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES	COMMENTS
patients with fatigue, regardless of etiology.		43, 44, 46	evidence that exercise therapy worsens outcomes.
Selective serotonin reuptake inhibitors, such as fluoxetine (Prozac), paroxetine (Paxil), or sertraline (Zoloft), may be helpful for patients with fatigue in whom depression is suspected.	B	22, 49	A six-week trial is recommended to evaluate effectiveness.
Cognitive behavior therapy is an effective treatment for adult outpatients with chronic fatigue syndrome.	A	22, 47, 48	—
Stimulants seldom return patients to predisease performance.	B	21, 45	Stimulants are associated with headaches, restlessness, insomnia, and dry mouth.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to https://www.aafp.org/afpsort.xml.

the patient's life, potential underlying psychiatric illness or sleep disturbance, and medications. Laboratory studies should be limited, in the absence of history or physical findings suggesting a particular illness.

- Establishing a trusting clinician-patient relationship and defining appropriate therapeutic goals is important. For patients with CFS, we recommend treatment with cognitive behavioral therapy (CBT) or graded exercise therapy (GET). For patients with chronic unexplained fatigue who do not meet criteria for CFS, we suggest treatment with CBT or GET.

- He hates his father for being aggressive to his mother.
- Thinks of death but never tried to hurt himself or thought of suicide.
- No change in bowel habit , no fever , no weight loss no palpitations , no chest pain , no SOB.
- Systemic review >> free
- Physical examination >> free
- Labs >> all within normal

BACK TO THE CASE

- He is smoker and feels guilty for that , not on medications , doesn't drink alcohol.
- He has decreased appetite , difficulty sleeping at night , no documented weight loss.
- Decreased concentration , low mood , loss of interest in things he used to enjoy.
- he spends his time sitting alone in his room.

APPROACH TO DIZZINESS

INTRODUCTION

- Dizziness is a common yet imprecise symptom often encountered by family physicians.
- Dizziness' is an ambiguous term, so the first thing to do is clarify what patients are talking about by asking them to describe what they're feeling. Then, based upon their description, you can tell if they are talking about lightheadedness, vertigo, presyncope, or gait problems.
- Primary care physicians see at least one-half of the patients who present with dizziness. Yet because patients use the terms "dizziness" and "vertigo" to describe a broad range of sensations, the symptom can pose a diagnostic challenge.

WHAT ARE THE KEY DIFFERENCES BETWEEN DIZZINESS AND VERTIGO?

- vertigo only refers to the illusion of motion, such as spinning when none is present, and many times the inner ear is the culprit for these symptoms.
- Dizziness, on the other hand, is a vaguer term that, while it includes vertigo, may also refer to sensations of discomfort in the head, such as lightheadedness, wooziness, disorientation, disequilibrium or imbalance. Many of these forms of dizziness aren't from the inner ear, but rather come from the brain or other parts of the body, such as with blood pressure changes.

- Dizziness was traditionally classified into four categories based on the patient's description:
  1) vertigo
  2) presyncope
  3) disequilibrium
  4) light-headedness

- Patients often have difficulty describing their symptoms
- Symptom quality does not reliably predict the cause of dizziness.
- SO, Attention to the timing and triggers of dizziness is preferred over the symptom type because patients more consistently report this information.eg:
  - **Episodic vertigo** triggered by **head motion** may be due to benign paroxysmal positional vertigo**(BPPV).**
  - Vertigo with **unilateral hearing loss** suggests **Meniere disease.**
  - **Episodic vertigo not associated** with any trigger may be a symptom of **vestibular neuritis.**
- Evaluation focuses on determining whether the etiology is peripheral or central. Peripheral etiologies are usually benign. Central etiologies often require urgent treatment.

Table 1. Differential Diagnosis of Dizziness and Vertigo: Common Causes

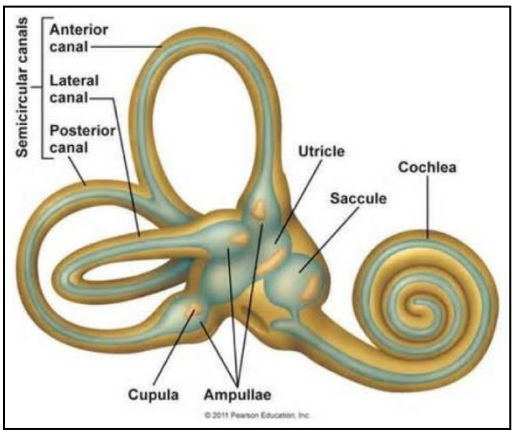
Cause (most to least frequent)	*Clinical description*
Peripheral causes	
Benign paroxysmal positional vertigo	Transient triggered episodes of vertigo caused by dislodged canaliths in the semicircular canals
Vestibular neuritis	Spontaneous episodes of vertigo caused by inflammation of the vestibular nerve or labyrinthine organs, usually from a viral infection
Meniere disease	Spontaneous episodes of vertigo associated with unilateral hearing loss caused by excess endolymphatic fluid pressure in the inner ear
Otosclerosis	Spontaneous episodes of vertigo caused by abnormal bone growth in the middle ear and associated with conductive hearing loss
Central causes	
Vestibular migraine	Spontaneous episodes of vertigo associated with migraine headaches
Cerebrovascular disease	Continuous spontaneous episodes of vertigo caused by arterial occlusion or insufficiency, especially affecting the vertebrobasilar system
Cerebellopontine angle and posterior fossa meningiomas	Continuous spontaneous episodes of dizziness caused by vestibular schwannoma (i.e., acoustic neuroma), infratentorial ependymoma, brainstem glioma, medulloblastoma, or neurofibromatosis
Other causes	
Psychiatric	Initially episodic, then often continuous episodes of dizziness without another cause and associated with psychiatric condition (e.g., anxiety, depression, bipolar disorder)
Medication induced	Continuous episodes of dizziness without another cause and associated with a possible medication adverse effect
Cardiovascular/ metabolic	Acute episodic symptoms that are not associated with any triggers
Orthostatic	Acute episodic symptoms associated with a change in position from supine or sitting to standing

Peripheral Etiologies

- Peripheral causes of dizziness arise from abnormalities in the peripheral vestibular system.
- The vestibular system is situated in the petrous part of the temporal bone, in close proximity to the cochlea. The vestibular system responds to movement of the head relative to space and gravity, using inertial-sensing receptors.

THE VESTIBULAR SYSTEM CONSISTS OF TWO TYPES OF SENSORS:

1. the two otolith organs (the saccule and utricle) , which sense linear movement (translation).
2. a set of three semicircular canals, arranged at right angles to each other, sensing rotation movement in three planes.
- and the vestibular nerve.



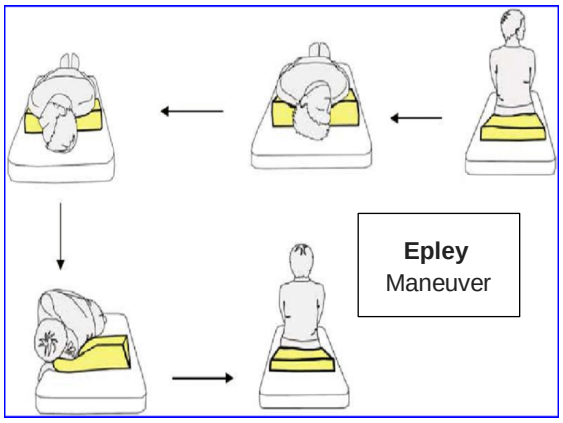
Common Peripheral Causes Of Dizziness/Vertigo Include:

1) BPPV
2) vestibular neuritis (i.e., vestibular neuronitis)
3) Meniere disease.

1. BENIGN PAROXYSMAL POSITIONAL VERTIGO

- BPPV in which calcium carbonate crystals, otoconia/canaliths, that normally reside in the utricle of the ear are dislodged and end up in the semicircular canals, usually the posterior canal.
- at any age, but is most common between 50 and 70 years.
- No obvious cause is found in 50% to 70% of older patients, but head trauma is a possibility in younger persons.

There are two main techniques used in the assessment and management of benign paroxysmal positional vertigo (BPPV) – the **Dix-Hallpike test** and the **Epley** Manoeuvre.
The **Dix-Hallpike Test** is used for the diagnosis of BPPV, whilst the **Epley** Manoeuvre can be used for its treatment once diagnosed.



TREATMENT:

1) canalith repositioning procedure such as the **Epley maneuver** which repositions the canalith from the semicircular canal into the vestibule.

2) *The success rate is approximately 70% 1st attempt, and 100% on successive maneuvers.*

3) **Home treatment: Brandt-Daroff exercises** can also be successful.

4) If there is no improvement with repeated repositioning maneuvers, or if atypical or ongoing nystagmus with nausea is present, another cause should be considered

5) Pharmacologic treatment has no role in the treatment of BPPV.

DIX-HALLPIKE TEST

1) Position the patient sitting upright on the bed, such that when supine their head will hang over the edge.

2) Stand behind the patient, and turn their head to 45° to one side. Warn the patient about what you are going to do.

3) Supporting the neck, lay the patient flat in one quick smooth movement, ensuring the head hangs over the end of the bed.

4) Ask the patient to keep their eyes open and observe for any abnormal eye movements. You should observe for at least 30 seconds.

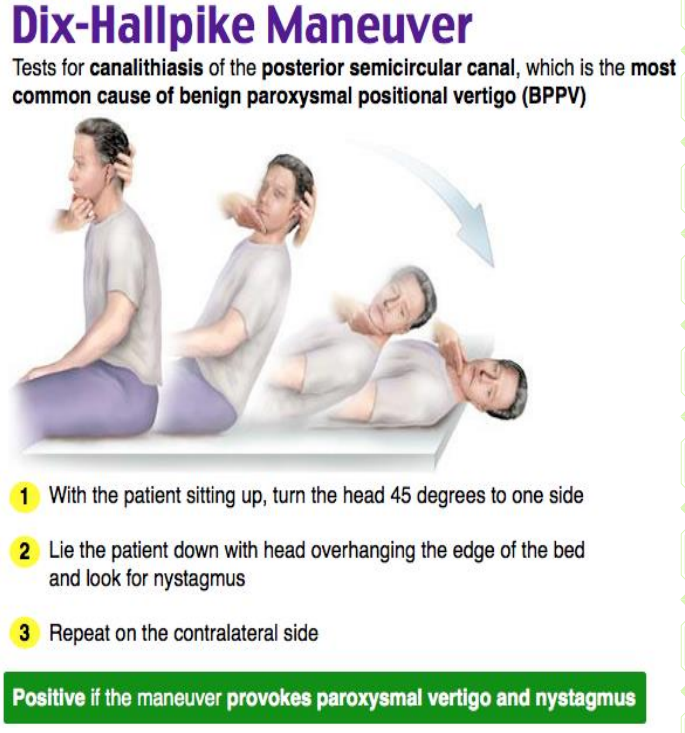
5) If positive, patient will experience vertigo and you will observe nystagmus:

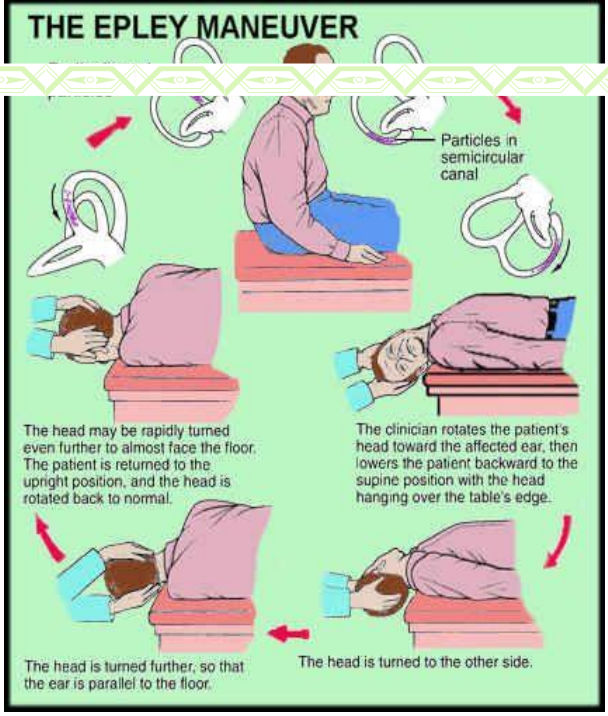
    - Rotatory nystagmus is seen if the superior semicircular canal is involved (most common)
    - Horizontal nystagmus is seen if the lateral semicircular canal is involved

6) Repeat the test for the other side

EPLEY MANOEUVRE

1) Continuing from the Dix-Hallpike Test, keep the patient supine and turn the head to the neutral position, still hanging over the end of the bed. Pause in this position for 30 seconds
2) Next, turn the head to the contralateral side, approximately 30° past the midline.
3) Maintaining the position of the head, ask the patient to roll onto their shoulder.
4) Turn the head so that it is facing the floor (the chin should be near to the shoulder), and pause in this position for 30 seconds.
5) Gently bring the patient to sitting position, ensuring the head position does not change relative to the trunk (their chin should remain near or on the shoulder). Pause for 30 seconds.
6) Turn the head to the center, and flex the neck – placing the chin onto the chest. Pause for 30 seconds.





2. VESTIBULAR NEURITIS

- This is inflammation of the vestibular nerve, possibly due to **viral infection**.
- This inflammation disrupts the transmission of sensory information from the ear to the brain.
- Vertigo Associated with **severe nausea and vomiting**
- vertigo = Longer than one day.
- the second most common cause of vertigo
- is thought to be of viral origin.
- It most commonly affects persons 30 to 50 years of age.
- Men = women
- diagnosed on the basis of the clinical history and physical examination.
- It can cause severe rotatory vertigo with nausea and apparent movement of objects in the visual field (oscillopsia), horizontally rotating spontaneous nystagmus to the nonaffected side, or an abnormal gait with a tendency to fall to the affected side.
- Hearing is not impaired in this condition.
- The Dix-Hallpike maneuver is not useful because patients with vestibular neuritis do not have episodic positionally triggered symptoms.
- As vestibular compensation occurs, the patient's vertigo resolves slowly over a few days.
- In 50% of patients, the underlying nerve damage may take two months to resolve. disequilibrium may persist for months
- If the attacks do not become successively shorter, another diagnosis should be considered.
- Reassurance, explanation, and advice are essential, in combination with symptomatic treatment for the first few days.
- The prognosis is excellent, but development of BPPV after an attack of vestibular neuritis may occur in 15% of patients.

TREATMENT:

- medications and vestibular rehabilitation
- Antiemetics and antinausea medications should be used for no more than three days because of their effects in blocking central compensation.

- Vertigo and associated nausea or vomiting can be treated with a combination of antihistamine, antiemetic, or benzodiazepine.
- systemic corticosteroids ? insufficient evidence for their routine use.
- Antiviral medications ? ineffective

3.MENIERE DISEASE

- vertigo and unilateral hearing loss.
- It caused by increased volume of endolymph in the semicircular canals. excess fluid pressure leading to inner ear dysfunction; however, the exact cause is unknown.
- Although it can develop at any age, it is more common between 20 and 60 years.
- psychological factors such as stress can act as a trigger mechanism for an attacks.
- Vertigo = less than one day and more than one minute.
- Associated symptoms : Tinnitus, fluctuating hearing loss worsened during an attack.(unilateral), Nausea & vomiting, loss of balance and headache

Diagnostic criteria for Meniere disease :

- 1. episodic vertigo (at least two episodes lasting at least 20 minutes)
- 2. associated with documented low- to medium-frequency sensorineural hearing loss by audiometric testing in the affected ear
- 3.tinnitus or aural fullness in the affected ear. initially unilateral
- The auditory symptoms are initially unilateral.

TREATMENT:

1) 1st line : lifestyle changes, including limiting dietary salt intake to less than 2,000 mg per day, reducing caffeine intake, and limiting alcohol to one drink per day.
2) Daily thiazide diuretic therapy can be added if vertigo is not controlled with lifestyle changes.
3) Transtympanic injections of glucocorticoids and gentamicin ?? can improve vertigo.
4) Vestibular suppressant medications may be used for acute attacks.
5) Surgery
6) Vestibular exercises

Central Etiologies

- The vestibular nuclei, cerebellum, brainstem, spinal cord, and vestibular cortex make up the central vestibular system.

- cause approximately 25% of dizziness experienced by patients.
- may present with disequilibrium and ataxia rather than true vertigo. However, vertigo can be a presenting symptom of an impending cerebrovascular event.
- may mimic a more benign peripheral disorder, and a stroke may present with no focal neurologic signs.
- CT does not have adequate sensitivity to distinguish stroke from benign causes of acute vestibular syndrome.
- The **HINTS examination** is highly sensitive and specific in identifying stroke in patients with acute vestibular syndrome, and it is superior to diffusion-weighted magnetic resonance imaging in ruling out stroke!

1. VESTIBULAR MIGRAINE

- Episodic vertigo in a patient with a history of migraine headaches suggests vestibular migraine.

2. VERTEBROBASILAR ISCHEMIA

- The blood supply to the brainstem, cerebellum, and inner ear is derived from the vertebrobasilar system.
- Diagnosis usually relies on a history of brainstem symptoms, such as diplopia, dysarthria, weakness, or clumsiness of the limbs.
- Vertigo is the initial symptom in 48% of patients, although fewer than one-half will have an associated neurologic finding.

General Approach

A. History: Timing, Triggers, And Medications

- Questions regarding:

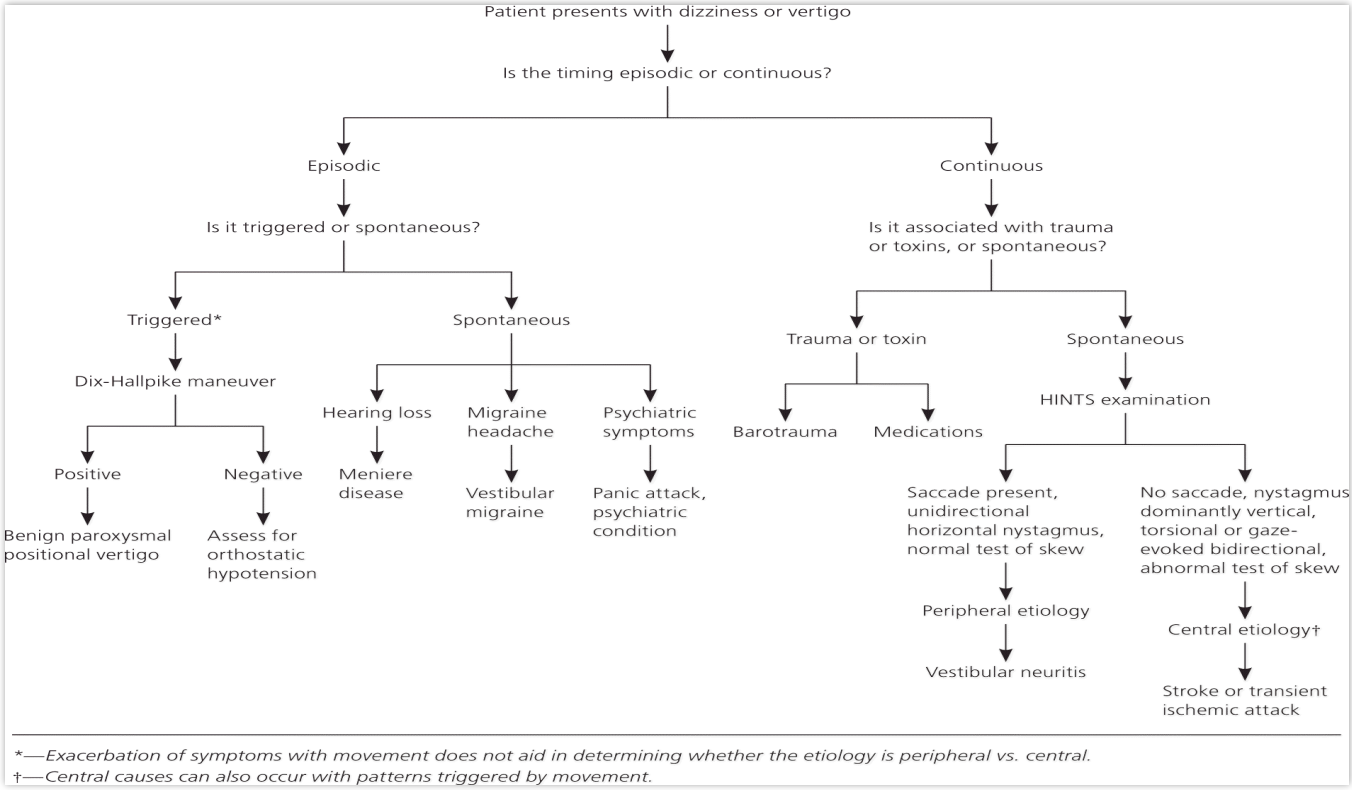
1. **Timing** (onset, duration, and evolution of dizziness)

2. **Triggers** (actions, movements, or situations) that provoke dizziness

CAN CATEGORIZE THE DIZZINESS AS MORE LIKELY TO BE :

1. peripheral
2. central in etiology.

- Findings from the physical examination can help confirm a probable diagnosis.



TITRATE THE EVALUATION

- TiTrATE is a novel diagnostic approach to determining the probable etiology of dizziness or vertigo.
- The approach uses the Timing of the symptom, the Triggers that provoke the symptom, And a Targeted Examination.
- The responses place the dizziness into one of three clinical scenarios :
  1) episodic triggered
  2) spontaneous episodic
  3) continuous vestibular

1. EPISODIC TRIGGERED SYMPTOMS

- Brief episodes of intermittent dizziness lasting seconds to hours.
- Common triggers are head motion on change of body position (e.g., rolling over in bed).
- Consistent with a diagnosis of benign paroxysmal positional vertigo (BPPV).

2. SPONTANEOUS EPISODIC SYMPTOMS

- (no trigger)
- patients have episodes of dizziness lasting seconds to days

- the patient's history establishes the diagnosis.
- Common diagnostic considerations for spontaneous episodic symptoms include Meniere disease, vestibular migraine, and psychiatric diagnoses such as anxiety disorders.
- Symptoms associated with lying down are more likely vestibular.

3. CONTINUOUS VESTIBULAR SYMPTOMS

- Patients have persistent dizziness lasting days to weeks.
- The symptoms may be due to traumatic or toxic exposure.
- Classic vestibular symptoms include continuous dizziness or vertigo associated with nausea, vomiting, nystagmus, gait instability, and head-motion intolerance.
- If no trauma or exposures → these findings are most consistent with vestibular neuritis or central etiologies.
- *Central causes can also occur with patterns triggered by movement !!*

Is It Triggered By A Specific Position Or Change In Position?

- BPPV is triggered with sudden changes in position, such as a quick turn of the head on awakening or tipping the head back in the shower.
- Orthostatic hypotension occurs with movement to the upright position.
- *dizziness that worsens with movement does not aid in determining whether the etiology is peripheral vs. central (benign vs. dangerous).*
- Medications were implicated in 23% of cases of dizziness in older adults in a primary care setting.
- The use of five or more medications is associated with an increased risk of dizziness.
- Older patients are more susceptible to medication adverse effects.

Medications Associated with Dizziness

MEDICATION	CAUSAL MECHANISM
Alcohol	Cardiac effects: hypotension, postural hypotension, torsades de pointes, other arrhythmias
Antiarrhythmics, class 1a	
Antidementia agents	
Antiepileptics	
Antihistamines (sedating)	
Antihypertensives	
Anti-infectives: anti-influenza agents, antifungals, quinolones	
Antiparkinsonian agents	
Attention-deficit/hyperactivity disorder agents	
Digitalis glycosides	
Dipyridamole	
Narcotics	
Nitrates	
Phosphodiesterase type 5 inhibitors	
Skeletal muscle relaxants	
Sodium–glucose cotransporter-2 inhibitors	
Urinary anticholinergics	
Skeletal muscle relaxants	
Sodium–glucose cotransporter-2 inhibitors	
Urinary anticholinergics	
Skeletal muscle relaxants	Central anticholinergic effects
Urinary and gastrointestinal antispasmodics	
Antiepileptics	Cerebellar toxicity
Benzodiazepines	
Lithium	
Antidiabetic agents	Hypoglycemia
Beta adrenergic blockers	
Aminoglycosides	Ototoxicity
Antirheumatic agents	
Anticoagulants	Bleeding complications (anticoagulants), bone marrow suppression (antithyroid agents)
Antithyroid agents	

B. Physical Examination

- Findings from the physical examination—including a cardiac and neurologic assessment, with attention to the head, eye, ear, nose, and throat examination—are usually normal in patients presenting with dizziness.
- Blood pressure should be measured while the patient is standing and in the supine position. Orthostatic hypotension ?
- A full Neurologic examination should be performed in patients with orthostatic dizziness but no hypotension or BPPV.
- The use of the **HINTS (head-impulse, nystagmus, test of skew) examination** can help distinguish a possible stroke (central cause) from acute vestibular syndrome (peripheral cause)

HINTS EXAMINATION

1) ***Head-Impulse.*** While the patient is sitting, the head is thrust 10 degrees to the right and then to the left while the patient's eyes remain fixed on the examiner's nose. If a saccade (rapid movement of both eyes) occurs, the etiology is likely peripheral. No eye movement strongly suggests a central etiology.

2) ***Nystagmus.*** The patient should follow the examiner's finger as it moves slowly left to right. Spontaneous unidirectional horizontal nystagmus that worsens when gazing in the direction of the nystagmus suggests a peripheral cause (vestibular neuritis).
- Spontaneous nystagmus that is dominantly vertical or torsional, or that changes direction with the gaze (gaze-evoked bidirectional) → central etiology

3) ***Test of Skew.*** Test of skew is assessed by asking the patient to look straight ahead, then cover and uncover each eye. Vertical

deviation of the covered eye after uncovering is an abnormal result which is fairly specific for brainstem involvement.

- BPPV is diagnosed with the Dix-Hallpike maneuver .
- Transient upbeat-torsional nystagmus during the maneuver is diagnostic of BPPV if the timing and trigger are consistent with BPPV.
- Nystagmus may not develop immediately, and a sense of vertigo may occur and last for one minute.
- A negative result does not rule out BPPV if the timing and triggers are consistent with BPPV.
- Nystagmus with the maneuver may be due to a central etiology, especially if the timing and trigger are not consistent with BPPV.

C. LABORATORY TESTING AND IMAGING

- Most patients presenting with dizziness do not require laboratory testing.
- Patients with chronic medical conditions (e.g., diabetes mellitus, hypertension) may require blood glucose and electrolyte measurements.
- Patients with symptoms suggestive of cardiac disease should undergo electrocardiography, Holter monitoring, and possibly carotid Doppler testing.
- *However, in a summary analysis of multiple studies that included 4,538 patients, only 26 (0.6%) had a laboratory result that explained their dizziness.*
- Routine imaging is not indicated. However, any abnormal neurologic finding, including asymmetric or unilateral hearing loss, requires CT or MRI to evaluate for cerebrovascular disease.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Vertigo associated with unilateral hearing loss should raise suspicion for Meniere disease.	C	41
The physical examination in patients with dizziness should include orthostatic blood pressure measurement, nystagmus assessment, and the Dix-Hallpike maneuver for triggered vertigo.	C	16
The HINTS (head-impulse, nystagmus, test of skew) examination can help differentiate a peripheral cause of vestibular neuritis from a central cause.	C	20
Laboratory testing and imaging are not recommended when no neurologic abnormality is found on examination.	C	1
Benign paroxysmal positional vertigo is treated with a canalith repositioning procedure (e.g., Epley maneuver).	A	30
Vestibular neuritis symptoms may be relieved with medication and vestibular rehabilitation.	C	20
Meniere disease may improve with a low-salt diet and diuretic use.	B	41

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

Adult Vaccination

"When meditating over a disease, I never think of finding a remedy for it, but instead, a means of prevention."

Recommended Adult Immunization Schedule
for ages 19 years or older

How to use the adult immunization schedule

1 Determine recommended vaccinations by age **(Table 1)**

2 Assess need for additional recommended vaccinations by medical condition and other indications **(Table 2)**

3 Review vaccine types, frequencies, and intervals, and considerations for special situations **(Notes)**

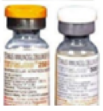
Vaccines in the Adult Immunization Schedule*

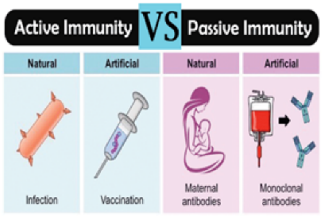
Vaccines	Abbreviations	Trade names
Haemophilus influenzae type b vaccine	Hib	ActHIB Hiberix
Hepatitis A vaccine	HepA	Havrix Vaqta
Hepatitis A and hepatitis B vaccine	HepA-HepB	Twinrix
Hepatitis B vaccine	HepB	Engerix-B Recombivax HB Heplisav-B
Human papillomavirus vaccine	HPV vaccine	Gardasil 9
Influenza vaccine, inactivated	IIV	Many brands
Influenza vaccine, live attenuated	LAIV	FluMist Quadrivalent
Influenza vaccine, recombinant	RIV	Flublok Quadrivalent
Measles, mumps, and rubella vaccine	MMR	M-M-R II
Meningococcal serogroups A, C, W, Y vaccine	MenACWY	Menactra Menveo
Meningococcal serogroup B vaccine	MenB-4C MenB-FHbp	Bexsero Trumenba
Pneumococcal 13-valent conjugate vaccine	PCV13	Prevnar 13
Pneumococcal 23-valent polysaccharide vaccine	PPSV23	Pneumovax
Tetanus and diphtheria toxoids	Td	Tenivac Td vaccine
Tetanus and diphtheria toxoids and acellular pertussis vaccine	Tdap	Adacel Boostrix
Varicella vaccine	VAR	Varivax
Zoster vaccine, recombinant	RZV	Shingrix
Zoster vaccine live	ZVL	Zostavax

*Administer recommended vaccines if vaccination history is incomplete or unknown. Do not restart or add doses to vaccine series for extended intervals between doses. The use of trade names is for identification purposes only and does not imply endorsement by the ACIP or CDC.

Immunization refers to the artificial induction of immunity. It can be by

- Active Immunization: the use of live attenuated infectious agents or inactivated toxins, or antigens obtained by genetic recombination OR
- Passive Immunization: temporary immunity obtained by the administration of immunoglobulins or antitoxins.





ACTIVE	PASSIVE
Immunogenic antigen is given then the body forms its own protective antibodies.	Ready-made immune globulin (antibodies) from human or animal sources are given to the body.
Long term protection (Sometimes life long)	Temporary immunity that decreases with time (turnover of the administered immunoglobulin)
Examples: **Natural**: Infection **Artificial**: Vaccination	Examples: **Natural**: Mother's Ig to infant (transplacental/breast milk) effective for about 6 months. **Artificial**: Adminestration of antibodies (e.g: Hepatitis B IG, Varicella IG)

Table 1 Recommended Adult Immunization Schedule by Age Group United States, 2019

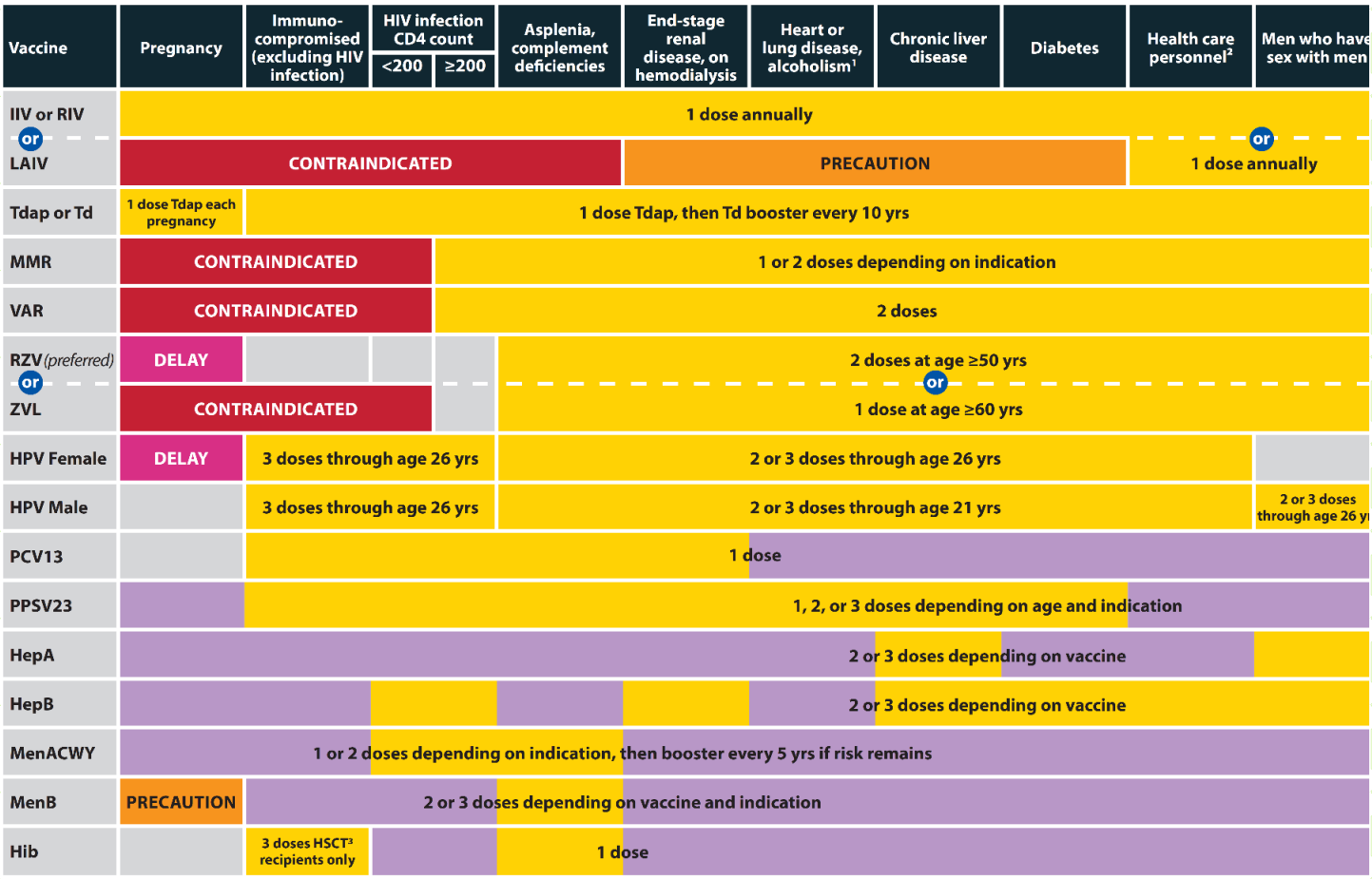
Vaccine	19–21 years	22–26 years	27–49 years	50–64 years	≥65 years
Influenza inactivated (IIV) or **Influenza recombinant** (RIV)	1 dose annually				
or					
Influenza live attenuated (LAIV)	1 dose annually				
Tetanus, diphtheria, pertussis (Tdap or Td)	1 dose Tdap, then Td booster every 10 yrs				
Measles, mumps, rubella (MMR)	1 or 2 doses depending on indication (if born in 1957 or later)				
Varicella (VAR)	2 doses (if born in 1980 or later)				
Zoster recombinant (RZV) *(preferred)*				2 doses	
or				or	
Zoster live (ZVL)					1 dose
Human papillomavirus (HPV) **Female**	2 or 3 doses depending on age at initial vaccination				
Human papillomavirus (HPV) **Male**	2 or 3 doses depending on age at initial vaccination				
Pneumococcal conjugate (PCV13)	1 dose				
Pneumococcal polysaccharide (PPSV23)	1 or 2 doses depending on indication				1 dose
Hepatitis A (HepA)	2 or 3 doses depending on vaccine				
Hepatitis B (HepB)	2 or 3 doses depending on vaccine				
Meningococcal A, C, W, Y (MenACWY)	1 or 2 doses depending on indication, then booster every 5 yrs if risk remains				
Meningococcal B (MenB)	2 or 3 doses depending on vaccine and indication				
***Haemophilus influenzae* type b** (Hib)	1 or 3 doses depending on indication				

Recommended vaccination for adults who meet age requirement, lack documentation of vaccination, or lack evidence of past infection

Recommended vaccination for adults with an additional risk factor or another indication

No recommendation

Table 2 Recommended Adult Immunization Schedule by Medical Condition and Other Indications United States, 2019



Haemophilus influenzae type b vaccination

Special situations

- **Anatomical or functional asplenia (including sickle cell disease)**: 1 dose Hib if previously did not receive Hib; if elective splenectomy, 1 dose Hib, preferably at least 14 days before splenectomy
- **Hematopoietic stem cell transplant** (HSCT): 3-dose series Hib 4 weeks apart starting 6–12 months after successful transplant, regardless of Hib vaccination history

Hepatitis A vaccination

Routine vaccination

- **Not at risk but want protection from hepatitis A** (identification of risk factor not required): 2-dose series HepA (Havrix 6–12 months apart or Vaqta 6–18 months apart [minimum interval: 6 months]) or 3-dose series HepA-HepB (Twinrix at 0, 1, 6 months [minimum intervals: 4 weeks between doses 1 and 2, 5 months between doses 2 and 3])

Special situations

- **At risk for hepatitis A virus infection**: 2-dose series HepA or 3-dose series HepA-HepB as above
  - **Chronic liver disease**
  - **Clotting factor disorders**
  - **Men who have sex with men**
  - **Injection or non-injection drug use**
  - **Homelessness**
  - **Work with hepatitis A virus** in research laboratory or nonhuman primates with hepatitis A virus infection
  - **Travel in countries with high or intermediate endemic hepatitis A**
  - **Close personal contact with international adoptee** (e.g., household, regular babysitting) in first 60 days after arrival from country with high or intermediate endemic hepatitis A (administer dose 1 as soon as adoption is planned, at least 2 weeks before adoptee's arrival)

Hepatitis B vaccination

Routine vaccination

- **Not at risk but want protection from hepatitis B** (identification of risk factor not required): 2- or 3-dose series HepB (2-dose series Heplisav-B at least 4 weeks apart [2-dose series HepB only applies when 2 doses of Heplisav-B are used at least 4 weeks apart] or 3-dose series Engerix-B or Recombivax HB at 0, 1, 6 months [minimum intervals: 4 weeks between doses 1 and 2, 8 weeks between doses 2 and 3, 16 weeks between doses 1 and 3]) or 3-dose series HepA-HepB (Twinrix at 0, 1, 6 months [minimum intervals: 4 weeks between doses 1 and 2, 5 months between doses 2 and 3])

Special situations

- **At risk for hepatitis B virus infection**: 2-dose (Heplisav-B) or 3-dose (Engerix-B, Recombivax HB) series HepB, or 3-dose series HepA-HepB as above
  - **Hepatitis C virus infection**
  - **Chronic liver disease** (e.g., cirrhosis, fatty liver disease, alcoholic liver disease, autoimmune hepatitis, alanine aminotransferase [ALT] or aspartate aminotransferase [AST] level greater than twice upper limit of normal)
  - **HIV infection**
  - **Sexual exposure risk** (e.g., sex partners of hepatitis B surface antigen (HBsAg)-positive persons; sexually active persons not in mutually monogamous relationships, persons seeking evaluation or treatment for a sexually transmitted infection, men who have sex with men)
  - **Current or recent injection drug use**
  - **Percutaneous or mucosal risk for exposure to blood** (e.g., household contacts of HBsAg-positive persons; residents and staff of facilities for developmentally disabled persons; health care and public safety personnel with reasonably anticipated risk for exposure to blood or blood-contaminated body fluids; hemodialysis, peritoneal dialysis, home dialysis, and predialysis patients; persons with diabetes mellitus age younger than 60 years and, at discretion of treating clinician, those age 60 years or older)
  - **Incarcerated persons**
  - **Travel in countries with high or intermediate endemic hepatitis B**

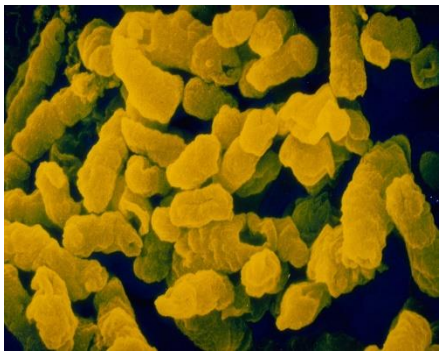
Human papillomavirus vaccination

Routine vaccination

- **Females through age 26 years and males through age 21 years:** 2- or 3-dose series HPV vaccine depending on age at initial vaccination; males age 22 through 26 years may be vaccinated based on individual clinical decision (HPV vaccination routinely recommended at age 11–12 years)
- **Age 15 years or older at initial vaccination**: 3-dose series HPV vaccine at 0, 1–2, 6 months (minimum intervals: 4 weeks between doses 1 and 2, 12 weeks between doses 2 and 3, 5 months between doses 1 and 3; repeat dose if administered too soon)
- **Age 9 through 14 years at initial vaccination and received 1 dose, or 2 doses less than 5 months apart**: 1 dose HPV vaccine
- **Age 9 through 14 years at initial vaccination and received 2 doses at least 5 months apart**: HPV vaccination complete, no additional dose needed
- If completed valid vaccination series with any HPV vaccine, no additional doses needed

Special situations

- **Immunocompromising conditions (including HIV infection) through age 26 years**: 3-dose series HPV vaccine at 0, 1–2, 6 months as above
- **Men who have sex with men and transgender persons through age 26 years**: 2- or 3-dose series HPV vaccine depending on age at initial vaccination as above
- **Pregnancy through age 26 years**: HPV vaccination not recommended until after pregnancy; no intervention needed if vaccinated while pregnant; pregnancy testing not needed before vaccination



Notes

Recommended Adult Immunization Schedule United States, 2019

Influenza vaccination

Routine vaccination

- **Persons age 6 months or older**: 1 dose IIV, RIV, or LAIV appropriate for age and health status annually
- For additional guidance, see www.cdc.gov/flu/professionals/index.htm

Special situations

- **Egg allergy, hives only**: 1 dose IIV, RIV, or LAIV appropriate for age and health status annually
- **Egg allergy more severe than hives** (e.g., angioedema, respiratory distress): 1 dose IIV, RIV, or LAIV appropriate for age and health status annually in medical setting under supervision of health care provider who can recognize and manage severe allergic conditions
- **Immunocompromising conditions (including HIV infection), anatomical or functional asplenia, pregnant women, close contacts and caregivers of severely immunocompromised persons in protected environment, use of influenza antiviral medications in previous 48 hours, with cerebrospinal fluid leak or cochlear implant**: 1 dose IIV or RIV annually (LAIV not recommended)
- **History of Guillain-Barré syndrome within 6 weeks of previous dose of influenza vaccine**: Generally should not be vaccinated

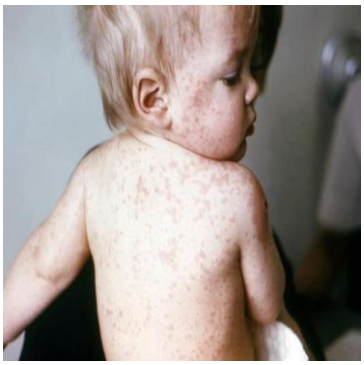
Measles, mumps, and rubella vaccination

Routine vaccination

- **No evidence of immunity to measles, mumps, or rubella**: 1 dose MMR
  - Evidence of immunity: Born before 1957 (except health care personnel [see below]), documentation of receipt of MMR, laboratory evidence of immunity or disease (diagnosis of disease without laboratory confirmation is not evidence of immunity)

Special situations

- **Pregnancy with no evidence of immunity to rubella**: MMR contraindicated during pregnancy; after pregnancy (before discharge from health care facility), 1 dose MMR
- **Non-pregnant women of childbearing age with no evidence of immunity to rubella**: 1 dose MMR
- **HIV infection with CD4 count ≥200 cells/µL for at least 6 months and no evidence of immunity to measles, mumps, or rubella**: 2-dose series MMR at least 4 weeks apart; MMR contraindicated in HIV infection with CD4 count <200 cells/µL
- **Severe immunocompromising conditions**: MMR contraindicated
- **Students in postsecondary educational institutions, international travelers, and household or close personal contacts of immunocompromised persons with no evidence of immunity to measles, mumps, or rubella**: 1 dose MMR if previously received 1 dose MMR, or 2-dose series MMR at least 4 weeks apart if previously did not receive any MMR
- **Health care personnel born in 1957 or later with no evidence of immunity to measles, mumps, or rubella**: 2-dose series MMR at least 4 weeks apart for measles or mumps, or at least 1 dose MMR for rubella; if born before 1957, consider 2-dose series MMR at least 4 weeks apart for measles or mumps, or 1 dose MMR for rubella



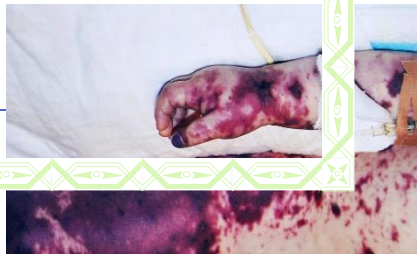
Meningococcal vaccination

Special situations for MenACWY

- **Anatomical or functional asplenia (including sickle cell disease), HIV infection, persistent complement component deficiency, eculizumab use**: 2-dose series MenACWY (Menactra, Menveo) at least 8 weeks apart and revaccinate every 5 years if risk remains
- **Travel in countries with hyperendemic or epidemic meningococcal disease, microbiologists routinely exposed to *Neisseria meningitidis***: 1 dose MenACWY and revaccinate every 5 years if risk remains
- **First-year college students who live in residential housing (if not previously vaccinated at age 16 years or older) and military recruits**: 1 dose MenACWY

Special situations for MenB

- **Anatomical or functional asplenia (including sickle cell disease), persistent complement component deficiency, eculizumab use, microbiologists routinely exposed to *Neisseria meningitidis***: 2-dose series MenB-4C (Bexsero) at least 1 month apart, or 3-dose series MenB-FHbp (Trumenba) at 0, 1–2, 6 months (if dose 2 was administered at least 6 months after dose 1, dose 3 not needed); MenB-4C and MenB-FHbp are not interchangeable (use same product for all doses in series)
- **Pregnancy**: Delay MenB until after pregnancy unless at increased risk and vaccination benefit outweighs potential risks
- **Healthy adolescents and young adults age 16 through 23 years (age 16 through 18 years preferred) not at increased risk for meningococcal disease**: Based on individual clinical decision, may receive 2-dose series MenB-4C at least 1 month apart, or 2-dose series MenB-FHbp at 0, 6 months (if dose 2 was administered less than 6 months after dose 1, administer dose 3 at least 4 months after dose 2); MenB-4C and MenB-FHbp are not interchangeable (use same product for all doses in series)



Notes

Recommended Adult Immunization Schedule United States, 2019

Pneumococcal vaccination

Routine vaccination

- **Age 65 years or older** (immunocompetent): 1 dose PCV13 if previously did not receive PCV13, followed by 1 dose PPSV23 at least 1 year after PCV13 and at least 5 years after last dose PPSV23
  - Previously received PPSV23 but not PCV13 at age 65 years or older: 1 dose PCV13 at least 1 year after PPSV23
  - When both PCV13 and PPSV23 are indicated, administer PCV13 first (PCV13 and PPSV23 should not be administered during same visit)

Special situations

- **Age 19 through 64 years with chronic medical conditions (chronic heart [excluding hypertension], lung, or liver disease; diabetes), alcoholism, or cigarette smoking**: 1 dose PPSV23
- **Age 19 years or older with immunocompromising conditions (congenital or acquired immunodeficiency [including B- and T-lymphocyte deficiency, complement deficiencies, phagocytic disorders, HIV infection], chronic renal failure, nephrotic syndrome, leukemia, lymphoma, Hodgkin disease, generalized malignancy, iatrogenic immunosuppression [e.g., drug or radiation therapy], solid organ transplant, multiple myeloma) or anatomical or functional asplenia (including sickle cell disease and other hemoglobinopathies)**: 1 dose PCV13 followed by 1 dose PPSV23 at least 8 weeks later, then another dose PPSV23 at least 5 years after previous PPSV23; at age 65 years or older, administer 1 dose PPSV23 at least 5 years after most recent PPSV23 (note: only 1 dose PPSV23 recommended at age 65 years or older)
- **Age 19 years or older with cerebrospinal fluid leak or cochlear implant**: 1 dose PCV13 followed by 1 dose PPSV23 at least 8 weeks later; at age 65 years or older, administer another dose PPSV23 at least 5 years after PPSV23 (note: only 1 dose PPSV23 recommended at age 65 years or older)

Tetanus, diphtheria, and pertussis vaccination

Routine vaccination

- **Previously did not receive Tdap at or after age 11 years**: 1 dose Tdap, then Td booster every 10 years

Special situations

- **Previously did not receive primary vaccination series for tetanus, diphtheria, and pertussis**: 1 dose Tdap followed by 1 dose Td at least 4 weeks after Tdap, and another dose Td 6–12 months after last Td (Tdap can be substituted for any Td dose, but preferred as first dose); Td booster every 10 years thereafter
- **Pregnancy**: 1 dose Tdap during each pregnancy, preferably in early part of gestational weeks 27–36
- For information on use of Tdap or Td as tetanus prophylaxis in wound management, see www.cdc.gov/mmwr/volumes/67/rr/rr6702a1.htm

Varicella vaccination

Routine vaccination

- **No evidence of immunity to varicella**: 2-dose series VAR 4–8 weeks apart if previously did not receive varicella-containing vaccine (VAR or MMRV [measles-mumps-rubella-varicella vaccine] for children); if previously received 1 dose varicella-containing vaccine: 1 dose VAR at least 4 weeks after first dose
  - Evidence of immunity: U.S.-born before 1980 (except for pregnant women and health care personnel [see below]), documentation of 2 doses varicella-containing vaccine at least 4 weeks apart, diagnosis or verification of history of varicella or herpes zoster by a health care provider, laboratory evidence of immunity or disease

Special situations

- **Pregnancy with no evidence of immunity to varicella**: VAR contraindicated during pregnancy; after pregnancy (before discharge from health care facility), 1 dose VAR if previously received 1 dose varicella-containing vaccine, or dose 1 of 2-dose series VAR (dose 2: 4–8 weeks later) if previously did not receive any varicella-containing vaccine, regardless of whether U.S.-born before 1980
- **Health care personnel with no evidence of immunity to varicella**: 1 dose VAR if previously received 1 dose varicella-containing vaccine, or 2-dose series VAR 4–8 weeks apart if previously did not receive any varicella-containing vaccine, regardless of whether U.S.-born before 1980
- **HIV infection with CD4 count ≥200 cells/μL with no evidence of immunity**: Consider 2-dose series VAR 3 months apart based on individual clinical decision; VAR contraindicated in HIV infection with CD4 count <200 cells/μL
- **Severe immunocompromising conditions**: VAR contraindicated

Zoster vaccination

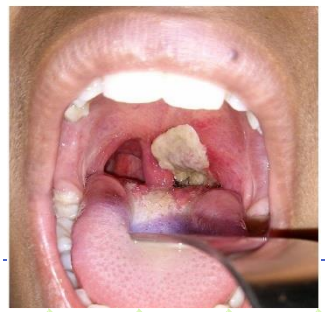
Routine vaccination

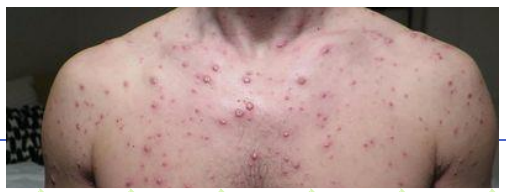
- **Age 50 years or older**: 2-dose series RZV 2–6 months apart (minimum interval: 4 weeks; repeat dose if administered too soon) regardless of previous herpes zoster or previously received ZVL (administer RZV at least 2 months after ZVL)
- **Age 60 years or older**: 2-dose series RZV 2–6 months apart (minimum interval: 4 weeks; repeat dose if administered too soon) or 1 dose ZVL if not previously vaccinated (if previously received ZVL, administer RZV at least 2 months after ZVL); RZV preferred over ZVL

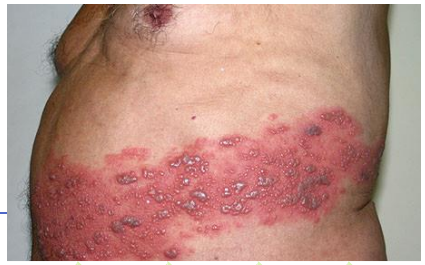
Special situations

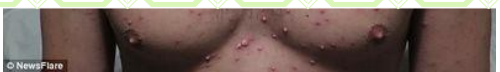
- **Pregnancy**: ZVL contraindicated; consider delaying RZV until after pregnancy if RZV is otherwise indicated
- **Severe immunocompromising conditions (including HIV infection with CD4 count <200 cells/μL)**: ZVL contraindicated; recommended use of RZV under review

Figure 1. A Strongly Adherent Pseudomembrane Of Diphtheria









APPROACH TO ACUTE HEADACHE IN ADULTS

INTRODUCTION

- Approximately one-half of the adult population worldwide is affected by a headache disorder. The International Headache Society classification and diagnostic criteria can help physicians differentiate primary headaches (e.g., tension, migraine, cluster) from secondary headaches (e.g., those caused by infection or vascular disease). A thorough history and physical examination, and an understanding of the typical features of primary headaches, can reduce the need for neuroimaging, lumbar puncture, or other studies.

- Some red flag signs and symptoms identified in the history or during a physical examination can indicate serious underlying pathology and will require neuroimaging or other testing to evaluate the cause of headache.

- Red flag signs and symptoms include focal neurologic signs, papilledema, neck stiffness, an immunocompromised state, sudden onset of the worst headache in the patient's life, personality changes, headache after trauma, and headache that is worse with exercise.

- If an intracranial hemorrhage is suspected, head computed tomography without contrast media is recommended. For most other dangerous causes of headache, magnetic resonance imaging or computed tomography is acceptable.

- It is important for physicians evaluating adult patients with acute headache to determine whether the condition is benign or if it indicates dangerous neurologic or systemic pathology. The most common types of headaches are tension-type headaches, migraines, and cluster headaches, which affect approximately 40, 10, and 1 % of the adult population, respectively.

- Most headache diagnoses are based entirely on the patient history. Only rarely does physical examination provide clues to the diagnosis.

- The International Headache Society has published a system of classification and operational diagnostic criteria for. Classifying headaches into primary (tension, migraine, or cluster) and secondary types (e.g., those caused by infection or vascular disease) is also useful to differentiate headaches that, although perhaps recurrent and temporarily disabling, have no dangerous underlying cause from those that may be a sign of significant pathology, because they represent an underlying systemic or neurologic disorder (Table 1).

Table 1. International Classification of Headache Disorders, 2nd ed. (ICHD-2)

Primary headaches
Migraine
Tension-type
Cluster
Other (e.g., cold stimulus headache)
Secondary headaches
Headache attributed to any of the following: head or neck trauma, cranial or cervical vascular disorder, nonvascular intracranial disorder, substance use or withdrawal, infection, disturbance of homeostasis, psychiatric disorder
Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures

Primary Headaches

Patients with a history of headache who do not have red flag signs and symptoms are at low risk of serious headache. Additionally, they should have

primary headache characteristics. Criteria for low-risk headaches are listed in Table 6. Patients at low risk of serious headache do not require neuroimaging.

Table 6. Criteria for Low-Risk Headaches

Age younger than 30 years
Features typical of primary headaches (Tables 1 through 5)
History of similar headache
No abnormal neurologic findings
No concerning change in usual headache pattern
No high-risk comorbid conditions (e.g., human immunodeficiency virus infection)
No new, concerning historical or physical examination findings (Table 7)

1. Tension-Type Headache

- Tension-type headache is the most common form of headache, and affects more than 40 percent of the adult population worldwide.
- It is characterized by bilateral mild to moderate pressure without other associated symptoms.
- Women are affected slightly more often than men.
- Nociceptors in the peri-cranial myofascial tissues are a likely source of tension headaches.
- Several studies have found that individuals who experience chronic tension-type headaches have increased sensitivity to pressure, electrical stimuli, and thermal stimuli in the peri-cranial myofascial tissue, and can find even normally harmless stimuli painful.
- Individuals who meet the criteria for tension-type headache but who have normal neurologic examination results require no additional laboratory testing or neuroimaging.
- Classification criteria for tension-type headaches are listed in Table 2.

Table 2. ICHD-2 Diagnostic Criteria for Episodic Tension-Type Headache

Infrequent
At least 10 episodes occurring fewer than one day per month on average (fewer than 12 days per year) and fulfilling the following criteria:
Headache lasts 30 minutes to seven days
Headache has at least two of the following features: bilateral location, pressing or tightening (nonpulsating) quality, mild or moderate intensity, not aggravated by routine physical activity such as walking or climbing stairs
Both of the following: no nausea or vomiting (anorexia may occur), either photophobia or phonophobia
Headache is not attributed to another disorder
Frequent
At least 10 episodes occurring on more than one but fewer than 15 days per month for at least three months and fulfilling all of the criteria for infrequent episodic tension-type headache

ICHD-2 = International Classification of Headache Disorders, 2nd ed.

2. Migraine Headaches

- Useful clinical criteria from the history and physical examination for distinguishing migraine from tension-type headache include nausea, photophobia (sensitivity to light), and phonophobia (sensitivity to sound). Physical activity often exacerbates migraine headache.

- Combined findings useful for distinguishing migraine can be summarized by the **POUND mnemonic** (pulsatile quality, duration of four to 72 hours, unilateral location, nausea or vomiting, and disabling intensity).
- Patients who meet at least four of these criteria are most likely to have a migraine.

- Aura may be present in some cases of migraine. Aura consists of visual, sensory, or speech symptoms that appear gradually, last no longer than 60 minutes, and are completely reversible. Table 3 lists criteria for migraine with aura; Table 4 lists criteria for migraine without aura.

Table 3. ICHD-2 Diagnostic Criteria for Migraine with Typical Aura

At least two episodes fulfilling the following criteria:

- Aura consisting of at least one of the following, but no motor weakness: fully reversible visual symptoms including positive features (e.g., flickering lights, spots or lines) and/or negative features (i.e., loss of vision); fully reversible sensory symptoms including positive features (i.e., pins and needles) and/or negative features (i.e., numbness); fully reversible dysphasic speech disturbance
- At least two of the following: homonymous visual symptoms and/or unilateral symptoms; at least one aura symptom develops gradually over five or more minutes and/or different aura symptoms occur in succession over five or more minutes; each symptom lasts at least five minutes, but no longer than 60 minutes
- A headache that fulfills the criteria for migraine without aura (Table 4), and begins during the aura or follows the aura within 60 minutes

Headache not attributed to another disorder

Table 4. ICHD-2 Diagnostic Criteria for Migraine Without Aura

At least five episodes fulfilling the following criteria:

- Headache episodes lasting four to 72 hours (untreated or unsuccessfully treated)
- Headache has at least two of the following characteristics: unilateral location, pulsating quality, moderate or severe pain intensity, aggravated by (or causes avoidance of) routine physical activity such as walking or climbing stairs
- During the headache, the patient experiences at least one of the following: nausea or vomiting; and photophobia and phonophobia

Headache is not attributed to another disorder

3. Cluster Headaches

- Cluster headaches are relatively rare, and are characterized by brief (15 to 180 minutes) episodes of severe head pain with associated autonomic symptoms (Table 5).

- The age of onset of cluster headaches varies, with 70 % of patients reporting onset before 30 years of age.

- Patients with cluster headache most commonly describe the pain as sharp, but some report that it can also be pulsating and pressure-like.

- Although pain can occur on both sides of the head, most patients report unilateral pain. Pain most commonly occurs in the retro-orbital area, followed by the temporal region, upper teeth, jaw, cheek, lower teeth, and neck.
- Ipsilateral autonomic symptoms such as eyelid edema, nasal congestion, lacrimation, or

forehead sweating usually accompany the pain.

Table 5. ICHD-2 Diagnostic Criteria for Cluster Headache

At least five episodes fulfilling the following criteria:
- Severe or very severe unilateral orbital, supraorbital, or temporal pain lasting 15 to 180 minutes if untreated
- Headache is accompanied by at least one of the following ipsilateral autonomic symptoms: conjunctival injection or lacrimation, nasal congestion or rhinorrhea, eyelid edema, forehead and facial sweating, miosis or ptosis, restlessness or agitation
- Headache episodes occur from one every other day to eight per day

Not attributable to another disorder

Episodic cluster headache

Fulfills all of the above criteria

At least two cluster periods lasting seven to 365 days and separated by pain-free remissions of more than one month

Chronic cluster headache

Fulfills all of the above criteria

Episodes recur for more than one year without remission periods or with remission periods lasting less than one month

- There tend to be several (up to eight) episodes in the same day, with each episode lasting between 15 and 180 minutes.
  - **In the episodic form** (80 to 90% of cases), episodes occur daily for a number of weeks followed by a period of remission. On average, a period of cluster headaches lasts 6 to 12 weeks, with remission lasting up to 12 months.
  - **In the chronic form** (10 to 20 % of cases), episodes occur without significant periods of remission.

- Only 25 percent of patients with cluster headaches are diagnosed correctly within one year of symptom onset, and more than 40 per-cent report a delay in diagnosis of five years or longer.

- The most common incorrect diagnoses reported in one study were migraine (34%), sinusitis (21 %), and allergies (6 %).

- Family history appears to have a role in some cases.

- A number of comorbidities are associated with cluster headaches, including depression (24 %), sleep apnea (14 %), restless legs syndrome (11 %), and asthma (9 %).

- Depression is an important diagnosis, because many individuals who have cluster headaches report suicidal thoughts.

DANGEROUS HEADACHES

- Distinguishing dangerous headaches from benign or low-risk headaches is a significant challenge because the symptoms can overlap.
- Recommendations for differentiating dangerous from benign headaches are provided in **Table 7.**
- Patients with characteristics of secondary headache should be evaluated to determine whether the headache is dangerous.
- Computed tomography of the head is the most widely used imaging study for acute head trauma because of its availability, speed, and accuracy.
- However, magnetic resonance imaging(MRI) of the brain is more sensitive for detecting subdural hematoma, and is therefore

particularly important in identifying smaller lesions.

Table 7. Red Flag Signs and Symptoms in the Evaluation of Acute Headache

Danger sign or symptom	*Possible diagnoses*	*Tests*
First or worst headache of the patient's life	Central nervous system infection, intracranial hemorrhage	Neuroimaging
Focal neurologic signs (not typical aura)	Arteriovenous malformation, collagen vascular disease, intracranial mass lesion	Blood tests, neuroimaging
Headache triggered by cough or exertion, or while engaged in sexual intercourse	Mass lesion, subarachnoid hemorrhage	Lumbar puncture, neuroimaging
Headache with change in personality, mental status, level of consciousness	Central nervous system infection, intracerebral bleed, mass lesion	Blood tests, lumbar puncture, neuroimaging
Neck stiffness or meningismus	Meningitis	Lumbar puncture
New onset of severe headache in pregnancy or postpartum	Cortical vein/cranial sinus thrombosis, carotid artery dissection, pituitary apoplexy	Neuroimaging
Older than 50 years	Mass lesion, temporal arteritis	Erythrocyte sedimentation rate, neuroimaging
Papilledema	Encephalitis, mass lesion, meningitis, pseudotumor	Lumbar puncture, neuroimaging
Rapid onset with strenuous exercise	Carotid artery dissection, intracranial bleed	Neuroimaging
Sudden onset (maximal intensity occurs within seconds to minutes, thunderclap headache)	Bleeding into a mass or arteriovenous malformation, mass lesion (especially posterior fossa), subarachnoid hemorrhage	Lumbar puncture, neuroimaging
Systemic illness with headache (fever, rash)	Arteritis, collagen vascular disease, encephalitis, meningitis	Blood tests, lumbar puncture, neuroimaging, skin biopsy
Tenderness over temporal artery	Polymyalgia rheumatica, temporal arteritis	Erythrocyte sedimentation rate, temporal artery biopsy
Worsening pattern	History of medication overuse, mass lesion, subdural hematoma	Neuroimaging
New headache type in a patient with:		
Cancer	Metastasis	Lumbar puncture, neuroimaging
Human immunodeficiency virus infection	Opportunistic infection, tumor	Lumbar puncture, neuroimaging
Lyme disease	Meningoencephalitis	Lumbar puncture, neuroimaging

History and Physical Examination

HISTORY.

- Thunderclap headache, which is characterized by sudden-onset headache pain, with peak intensity occurring within several minutes, requires prompt evaluation. Subarachnoid hemorrhage, hypertensive emergencies, vertebral artery dissections, and acute angle–closure glaucoma can also present this way.
- Use of illicit drugs, including cocaine and methamphetamine, can increase the risk of intracranial bleeding or stroke.
- Prescription or over-the-counter medications such as aspirin, other nonsteroidal anti-inflammatory drugs, anticoagulants, and

glucocorticoids increase the risk of intracranial bleeding.

- A history of HIV infection or other immunosuppressive conditions in patients with headache may suggest a brain abscess, meningitis, or malignancy of the central nervous system (CNS).
- The presence of a coexisting infection in the lungs, sinuses, or orbital areas may precede and cause a CNS infection.
- A patient who reports the worst headache of his or her life, especially if the patient is older than 50 years, or who has a headache that occurs with exertion (including sexual intercourse) could be experiencing intracranial hemorrhage or carotid artery dissection.
- Prompt investigation is required for any headaches associated with neurologic findings, including changes in mental status, seizures, and visual disturbances. Additional red flag symptoms and signs are listed in Table 7.

PHYSICAL EXAMINATION

- Neurologic abnormalities require evaluation and are particularly concerning in association with acute headache. Abnormalities are one of the best predictors of CNS pathology.

- A focal neurologic deficit should not be attributed to migraine headache unless a similar pattern has occurred with a previous migraine. By definition, aura associated with migraine lasts 60 minutes or less.
- Therefore, headache with aura-like symptoms should not be assumed to be benign or a primary headache when aura-like symptoms are present for more than 60 minutes.

- Abnormal findings on examination can be pronounced, such as meningismus or unilateral vision loss, or subtle, such as extensor plantar response or unilateral pronator drift.
- Obtundation or confusion suggests a dangerous headache because these signs do not occur with benign or primary headache.

- Patients with headache and fever, papilledema, or severe hypertension (systolic pressure greater than 180 mm Hg or diastolic pressure greater than 120 mm Hg) require evaluation for CNS infection and increased intracranial pressure.
- Patients also should be evaluated to determine if their blood pressure should be lowered to safer levels to avoid intracranial hemorrhage from malignant hypertension.
-
- Contusions and facial or scalp lacerations increase the likelihood of associated intra-cranial hemorrhage .

Table 8. American College of Radiology Recommendations for Neuroimaging in Patients with Headache

Clinical features	*Recommended imaging modality*
Headache in immunocompromised patients	MRI of the head with and without contrast media
Headache in patients older than 60 years with suspected temporal arteritis	MRI of the head with and without contrast media
Headache with suspected meningitis	CT or MRI of the head without contrast media
Severe headache in pregnancy	CT or MRI of the head without contrast media
Severe unilateral headache caused by possible dissection of the carotid or arterial arteries	MRI of the head with and without contrast media, MRA of the head and neck, or CTA of the head and neck
Sudden onset or severe headache; worst headache of the patient's life	CT of the head without contrast media; CTA of the head with contrast media, MRA of the head with or without contrast media, or MRI of the head without contrast media

CT = computed tomography; CTA = computed tomographic angiography; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging.

Information from reference 28.

Periodic Health Examination And Screening

Strategy used in a population to identify the possible presence of an as-yet-undiagnosed disease in individuals without signs or symptoms.

Disease	Screening test	Age	gender
Breast cancer	1-Mammography	40-52 once every 2 yrs >52 once/year	Female
	2-Physical examination	20 yrs every 1-3 yrs	
	3-BRCA testing	family history of ovarian, tubual, peritoneal, breast CA	
	4-periodic manual examination	monthly after menses	
Abdominal aortic aneurysm	Abdominal Ultrasonography	65-75 yrs (one-time)	Male (smoker)
Hypertension	BP measurement	Adults >=18 yrs Children at 3 yrs of age Pregnancy	Both
Colon CA/polyps	1-Faecal occult blood test (3 stool samples)	50 yrs	Both
	2-Colonoscopy (most common)	*At 50 yrs (every 10 yrs) *fam. Hist.: 40 yrs or 10 yrs earlier than the youngest age (which is earlier) → every 5 yrs *FAP: at 10-12 yrs	
	3-Flexible sigmoidoscopy 4-double contrast barium enema 5-CT colonography	Every 5 years	
DM or preDM	HbA1c RBG/ FBG/ OGTT	*overweight+ 1 risk factor *45y regardless of weight	Both
Prostate CA/BPH	Digital rectal examination/ prostate specific antigen (PSA)	50 yrs	Male
Cervical CA	Pap test (Pap smear) HPV test	<65 yrs at 21yrs every 5 years at 30 yrs every 3 yrs + HPV test	Sexually active women
Osteoporosis	DEXA scan (frax score)	1-men >= 70 yrs with chronic disease: rhomatopid arthritis, chronic liver or renal disease. 2-women >= 65 yrs without HRT 3-post-menopausal women <65 yrs with RF 4- hyperthyroidism / hyperparathyroidism 5- on steroid for long duration	Both
Depression	Patient health questionnaire (PHQ 2 & 9)	*Adolescents 12-18 yrs *adults >18 including pregnant and postpartum women	Both
Sexually transmitted diseases	1-Chlamydia screening 2-Gonorrhoea screening 3-HIV screening	HIV screening (15-65 yrs)	Both
Lung CA	Low dose CT scan	55-80 yrs (smoker or ex-smoker within the last 15 yrs)	Both

PREVENTION:

1) ASPIRIN (LOW DOSE) PREVENTION

- use for the primary prevention of cardiovascular disease and colorectal cancer
- in adults aged 50 to 59 years
- have a 10% or greater 10-year cardiovascular risk
- not at increased risk for bleeding
- have a life expectancy of at least 10 years
- willing to take low-dose aspirin daily for at least 10 years

2)STATIN PREVENTION

adults without a history of cardiovascular disease (CVD) use a low- to moderate-dose statin
for prevention of CVD events and mortality when all of the following criteria are met:

1- They are ages 40 to 75 years.

2- they have 1 or more CVD risk factors (i.e., dyslipidemia, diabetes, hypertension, or smoking)

3- they have a calculated 10-year risk of a cardiovascular event of 10% or greater.

PREGNANCY:

1) Folic acid supplementation (0.4-0.8 mg daily / start 3 months before conception)
2) HBV screening
3) HIV screening
4) Syphilis screening
5) Rh incompatibility screening at first pregnancy 24-28 weeks of gestation (unless the father is Rh-) + give monoclonal antibodies immediately after birth if the baby is Rh+
6) Urine culture asymptomatic bacteriuria at 12-16 weeks of gestation or at the first visit if late
7) OGTT (oral glucose tolerance test) gestational diabetes
8) BP monitoring for preeclampsia/ eclampsia
9) PHQ 2 & 9 for depression
10) vaginal swab for GBS

CHILDREN:

1) BP at 3 yrs
2) Depression (PHQ 2 & 9) 12-18 yrs
3) Phenylketonuria screening newborns
4) Hypothyroidism to prevent cretinism at birth (take sample from the heel)
5) Dental caries prevention (dental chemoprophylaxis) infants and children up to 5 yrs
6) Hearing examination at birth
7) Vision examination

Preventive Service	Guideline
Abdominal Aortic Aneurysm	One-time screening for abdominal aortic aneurysm by ultrasonography in men age 65 to 75 years who have ever smoked.
Aspirin chemoprevention	Initiating low-dose aspirin use for the primary prevention of cardiovascular disease (CVD) and colorectal cancer (CRC) in adults aged 50 to 59 years who have a 10% or greater 10-year CVD risk, are not at increased risk for bleeding, have a life expectancy of at least 10 years, and are willing to take low-dose aspirin daily for at least 10 years. The decision to initiate aspirin in patients between 60 and 69 should be individualized. There is insufficient evidence to assess the balance of benefits and harms for patients younger than age 50 or older than age 70
Blood Pressure	Blood pressure should be measured at each visit beginning at age 18. "The USPSTF recommends annual screening for adults aged 40 years or older and for those who are at increased risk for high blood pressure. Persons at increased risk include those who have high-normal blood pressure (130 to 139/85 to 89 mm Hg), those who are overweight or obese, and African Americans. Adults aged 18 to 39 years with normal blood pressure (<130/85 mm Hg) who do not have other risk factors should be rescreened every 3 to 5 years. The USPSTF recommends rescreening with properly measured office blood pressure and, if blood pressure is elevated, confirming the diagnosis of hypertension with ABPM."
Breast Cancer Screen Self Breast Exam (SBE)	Beginning in their 20's, women should be told about the benefits and limitations of BSE, it is acceptable for women to choose not to do BSE, or to do it occasionally. The importance of promptly reporting changes to a physician is emphasized.
Clinical Breast Exam/Mammography	A clinical breast exam (CBE) may be performed though there is insufficient evidence to assess additional benefit beyond that of mammography. For women in their 40's, the decision to perform mammography and the frequency of mammograms should be individualized. For women ages 50-74, mammograms should be performed biennially. For women > 75 years of age, the decision to continue screening should be individualized. Screening should be discontinued for women with a life expectancy < 10 years. Women known to be at increased risk (Family history, Positive Gail risk screen) may benefit from earlier initiation of screening and/or referral to Breast Specialist.

Preventive Service	Guideline
Cervical Cancer Screening	Cervical cancer screening should begin at age 21 years (regardless of sexual history*). Screening before age 21 should be avoided because women less than 21 years old are at very low risk of cancer. Screening these women may lead to unnecessary and harmful evaluation and treatment .* • Women from ages 21 to 29 should be screened every three years, using either the standard Pap or liquid-based cytology. HPV co-testing (cytology + HPV test administered together) should **not be** used for women aged <30 years • Women ages 30-65 may be screened once every three years with either the standard Pap or liquid-based cytology OR every 5 years with HPV co-testing (cytology + HPV test administered together) • Women with certain risk factors may need more frequent screening, including those who have HIV, are immunosuppressed, were exposed to diethylstilbestrol (DES) in utero, and have been treated for cervical intraepithelial neoplasia (CIN) 2, CIN 3, or cervical cancer. • May discontinue screening >65 years(with adequate screening history)
Chlamydia & Gonorrhea Infection	Sexually active women aged 24 years and younger and other asymptomatic women at increased risk for infection.
Cholesterol Screening	• The age at which screening should begin should be based on an individual's other cardiac risk factors and desire to be screened. • Screening may begin in non-pregnant adults at any age but no later than age 40 (the age at which statin therapy for primary prevention is recommended). • 10-year risk should be re-evaluated every 4-6 yrs between 40-75 years old. • The development of diabetes or clinical ASCVD should prompt evaluation as well.
Colorectal Cancer Screening	Beginning at age 50, both men and women at *average risk* for developing colorectal cancer should use one of the screening tests below. The tests that are designed to find both early cancer and polyps are preferred if these tests are available and the patient is willing to have one of these more invasive tests. **Tests that find polyps and cancer** ▪ flexible sigmoidoscopy every 5 years* ▪ colonoscopy every 10 years ▪ CT colonography (virtual colonoscopy) every 5 years* (consider community availability) ▪ Combination Flex sig every 10 yrs with annual FIT testing **Tests that mainly find cancer** ▪ fecal occult blood test (FOBT) every year*,** ▪ fecal immunochemical test (FIT) every year*,** ▪ FIT- DNA test (sDNA), q 1 or 3 yrs *Colonoscopy should be done if test results are positive. Screening should be considered earlier and/or more often for individuals with any of the following colorectal cancer risk factors: personal Hx of colorectal cancer, a personal history of chronic inflammatory bowel disease (Crohns disease or ulcerative colitis), a strong family history of colorectal cancer or polyps (cancer or polyps in a first-degree relative [parent, sibling, or child] younger than 60 or in 2 or more first-degree relatives of any age), a known family history of hereditary colorectal cancer syndromes such as familial adenomatous polyposis (FAP) or hereditary non-polyposis colon cancer (HNPCC).

Preventive Service	Guideline
Diabetes Mellitus and Pre-Diabetes	All individuals 45 years and older should be screened. Testing should be considered in all adults who are overweight (BMI≥25 kg/m2 or ≥23 kg/m2 in Asian Americans) and have additional risk factors: • 1st degree relative with diabetes. • Physically inactive. • High-risk ethnic group (African American, Latino, Native or Asian Americans, Pacific Islanders). • Hypertension (≥140/90) or on therapy for hypertension. • PCOS, (polycystic ovary syndrome). • Plasma high-density lipoprotein cholesterol level <35 mg/dl or triglyceride level >250 mg/dl. • History of CVD. • Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans) Women with a history of gestational diabetes should have lifelong testing at least every 3 yrs Screening Methods: Fasting plasma glucose, 2 hr plasma glucose following 75 gm OGTT or A1C are all acceptable modalities. Re-screening should occur at a minimum every 3 yrs if results are normal. Individuals with pre-diabetes (impaired glucose tolerance (140-199 mg/dl) or impaired fasting glucose (100-125 mg/dl) or an A1C of 5.7-6.4% should be tested annually.
Depression	Screening for symptoms of depression should be at the initial visit for all new patients and then annually for existing patients. The patient may complete screening during the office visit with a patient self-reported questionnaire or using one of the various screening measures that have been specifically designed to detect depression. Physicians can choose the screening measures that are appropriate for their patients and practice setting, and for monitoring change in patients who are receiving treatment for depression.
Hepatitis C Screening	USPSTF & CDC recommend hepatitis C screening for all asymptomatic adults without known liver disease or functional abnormalities born between 945-1965. Other patients who should be screened include: • Those who have ever injected illegal drugs • Those who have received clotting factors made before 1987 • Those who have received blood/organs before July 1992 • Those who were ever on chronic hemodialysis • Those who have evidence of liver disease (elevated alanine aminotransferase [ALT] level) • Those who are infected with HIV

Preventive Service	Guideline
HIV Testing	Testing for HIV infection should be performed routinely for all patients aged 13--64 years in an "opt out" fashion. All persons likely to be at high risk should be screened at least annually (high risk includes: *injection-drug users and their sex partners, persons who exchange sex for money or drugs, sex partners of HIV-infected persons, men having sex with men or heterosexual persons who themselves or whose sex partners have had more than one sex partner since their most recent HIV test)*. No written consent required however documentation in the medical record of informed consent is necessary in the state of Maryland.
Lung Cancer Screening	The USPSTF recommends annual screening for lung cancer using low-dose CT scanning in adults aged 55-80 with a 30 pack year smoking history and who are current smokers or have quit within the past 15 yrs. Screening should be discontinued once a person has not smoked for 15 yrs, develops a health problem substantially limiting life expectancy, or is unable or unwilling to have curative lung surgery.
Osteoporosis Screening	Recommend BMD testing to all women aged 65 and older regardless of additional risk factors. In postmenopausal women and men over age 50, recommend BMD testing when you have concern based on their risk factor profile. The WHO FRAX tool can be used to estimate risk of osteoporosis. Routine screening of men age 70 and older regardless of additional risk factors is not recommended by the USPSTF but is by the NOF and other groups. Bone mineral density testing should be performed on all women who are postmenopausal with fractures to confirm the diagnosis of osteoporosis and determine the severity of disease (ACOG). The timing of repeat screening should be individualized based on baseline results but should occur no more often than every 2 yrs.
Prostate Cancer Screening	Offer and discuss risks and benefits of a PSA-based screening and digital rectal examinations to detect prostate cancer in men age 50 who are at average risk of prostate cancer and are expected to live at least 10 more years. Discussion should begin at age 45 for men at high risk (African-American men and men with a strong family history of one or more first-degree relatives [father, brothers] diagnosed before age 65. Men at even higher risk, due to multiple first-degree relatives affected at an early age, should be counseled at age 40 (ACS). Men who choose to be tested who have a PSA of less than 2.5 ng/ml, may only need to be retested every 2 years. Screening should be done yearly for men whose PSA level is 2.5 ng/ml or higher. • The USPSTF and American Academy of Family Physicians recommend against screening for prostate cancer (38, 42). • The American Cancer Society emphasizes informed decision making for prostate cancer screening: men at average risk should receive information beginning at age 50 years, and black men or men with a family history of prostate cancer should receive information at age 45 years (39). • The American College of Preventive Medicine recommends that clinicians discuss the potential benefits and harms of PSA screening with men aged 50 years or older, consider their patients' preferences, and individualize screening decisions (43).
Syphilis Screening	All pregnant patients and all non-pregnant patients at increased risk of syphilis exposure should be screened. Such patients include but may not be limited to men who have sex with men, HIV infected patients, commercial sex workers, patients who have been incarcerated, men younger than age 29 and patients living in areas of high prevalence.
Tuberculosis Screening	Screening for latent tuberculosis should be performed in groups at increased risk of exposure and increased risk of developing active disease including patients living in homeless shelters or correctional institutions, patients coming from countries with high prevalence of TB, immunosuppressed patients, patients with silicosis, and patients with TB exposure (household contacts or occupational exposure).

APPROACH TO CHRONIC COUGH

INTRODUCTION

- Chronic cough is a common problem in patients who visit family physicians, it is the fifth most common symptom for which patients seek care, resulting in approximately 30 million office visits per year.

- four conditions account for most cases of chronic cough:
  1) upper airway cough syndrome (postnasal drip)
  2) asthma
  3) gastroesophageal reflux, laryngopharyngeal reflux disease
  4) nonasthmatic eosinophilic bronchitis.

- Patients should be evaluated clinically (with spirometry, if indicated), and The initial treatment of patients with cough is often empiric and may involve a trial of decongestants, bronchodilators or histamine H2 antagonists, as monotherapy or in combination.
- Other potential causes include angiotensin-converting enzyme inhibitor use, environmental triggers, tobacco use, chronic obstructive pulmonary disease, and obstructive sleep apnea.
- If a therapeutic trial is not successful, sequential diagnostic testing including:
  - chest radiograph = rule out concerning infectious, inflammatory, and malignant thoracic conditions.
  - purified protein derivative test for tuberculosis
  - computed tomography of the sinuses
  - methacholine challenge test
  - barium swallow.

- By using a standard protocol for diagnosis and treatment, 90 % of patients with chronic cough can be managed successfully in the family physician's office.

- However, in some cases it may take three to five months to determine a diagnosis and effective treatment. For the minority of patients in whom this diagnostic approach is unsuccessful, consultation with a pulmonary specialist is appropriate.
- Cough occurs in association with acute upper respiratory infection, acute pharyngitis, acute bronchitis and chronic sinusitis, all of which rank among the top 10 reasons for visiting family physicians.

DEFINITION

- In adults, chronic cough is defined as symptoms lasting longer than eight weeks, whereas acute cough lasts less than three weeks and subacute cough from three to eight weeks.
- Cough caused by the common cold typically lasts one to three weeks and is self-limited.

ASSESSMENT OF CHRONIC COUGH

- The initial evaluation should focus on identifying potential triggers, such as the use of an angiotensin-converting enzyme (ACE) inhibitor, environmental exposures, smoking status, and chronic obstructive pulmonary disease (COPD).

- It should also rule out red flags (e.g., fever, weight loss, hemoptysis, hoarseness, excessive dyspnea or sputum production, recurrent pneumonia, smoking history of 20 pack-years, or smoker older than 45 years) that suggest a serious underlying cause of cough.

- Unless a likely cause is identified, chest radiography should be obtained to rule out most infectious, inflammatory, and malignant thoracic conditions.

- When physical examination findings are normal and no red flags are present, routine computed tomography of the chest and sinuses is not necessary, nor is initial bronchoscopy.

- The diagnostic approach should focus on detection and treatment of the four most common causes of chronic cough in adults: upper airway cough syndrome (UACS), asthma, nonasthmatic eosinophilic bronchitis, and gastroesophageal reflux disease (GERD)/laryngopharyngeal reflux disease.

- After evaluation and empiric management of these etiologies, less common causes should be considered (Tables 1 and 2).
- A suggested primary care approach to the evaluation of chronic cough for immunocompetent adults is shown in Figure 1.
- The following tests were found to be most useful in diagnosing cough in children:

1. birth to 18 months—endoscopy and therapeutic trial of bronchodilators;
2. 18 months to six years—radiographs of the paranasal sinuses and therapeutic trial of bronchodilators and endoscopy;
3. six to 16 years—pulmonary function testing with methacholine inhalation challenge test and endoscopy.

Table 1. Etiologies of Chronic Cough in Adults and Children

Adults	Children
Most common	**Most common**
Angiotensin-converting enzyme inhibitor use	Asthma
Asthma	Protracted bacterial bronchitis
Environmental triggers	Upper airway cough syndrome (in children older than six years)
Gastroesophageal/laryngopharyngeal reflux disease	**Less common**
Nonasthmatic eosinophilic bronchitis	Environmental triggers
Tobacco use	Foreign body (in younger children)
Upper airway cough syndrome	Gastroesophageal reflux disease
Less common	Pertussis
Bronchiectasis	Postinfectious bronchospasm
Chronic obstructive pulmonary disease	**Least common**
Obstructive sleep apnea	Chronic aspiration
Pertussis	Congenital abnormality
Postinfectious bronchospasm	Cystic fibrosis
Least common	Immunodeficiency
Arteriovenous malformation	Primary ciliary dyskinesia
Bronchiolitis	Psychogenic cough
Bronchogenic carcinoma	Tourette syndrome/tic
Chronic aspiration	
Chronic interstitial lung disease	
Irritation of external auditory canal	
Persistent pneumonia	
Psychogenic cough	
Sarcoidosis	
Tuberculosis	

COMMON CAUSES OF CHRONIC COUGH

1. UPPER AIRWAY COUGH SYNDROME

- The term UACS, previously referred to as postnasal drip syndrome, because the fact that multiple etiologies, including chronic rhinosinusitis, allergic rhinitis, and nonallergic rhinitis, were difficult to differentiate solely by clinical presentation.

- UACS is the most common cause of chronic cough.

- Rhinorrhea, nasal stuffiness, sneezing, itching, and postnasal drainage suggest the diagnosis, but their absence does not rule out UACS.

- Physical findings may include swollen turbinates and direct visualization of postnasal drainage and cobble-stoning of the posterior pharynx.

- If a specific cause is identified, therapy should be started; otherwise, initial treatment includes a decongestant combined with a first-generation antihistamine.

- Intranasal corticosteroids, saline nasal rinses, nasal anticholinergics, and antihistamines are also reasonable options.
- Clinical improvement should occur within days to weeks, and up to two months.

- If chronic rhinosinusitis is suspected, sinus computed tomography or flexible nasolaryngoscopy should be performed. Sinus radiography is not recommended because of limited sensitivity.

2. ASTHMA AND COPD

- Asthma should be suspected in patients with shortness of breath, wheezing, and chest tightness, but cough can be the only manifestation in cough variant asthma.

- If the physical examination and spirometry findings are nondiagnostic, bronchial challenge testing (methacholine inhalation test) should be considered. Resolution of the cough after asthma treatment is also diagnostic.

- After counseling the patient about potential triggers, treatment usually includes an inhaled bronchodilator and high-dose inhaled corticosteroid. A leukotriene receptor antagonist (e.g., montelukast) can also be useful.
- Symptoms should resolve within one to two weeks after starting treatment.

- For severe or refractory cough, a five- to 10-day course of prednisone, or equivalent oral corticosteroid can be considered if asthma is strongly suspected.

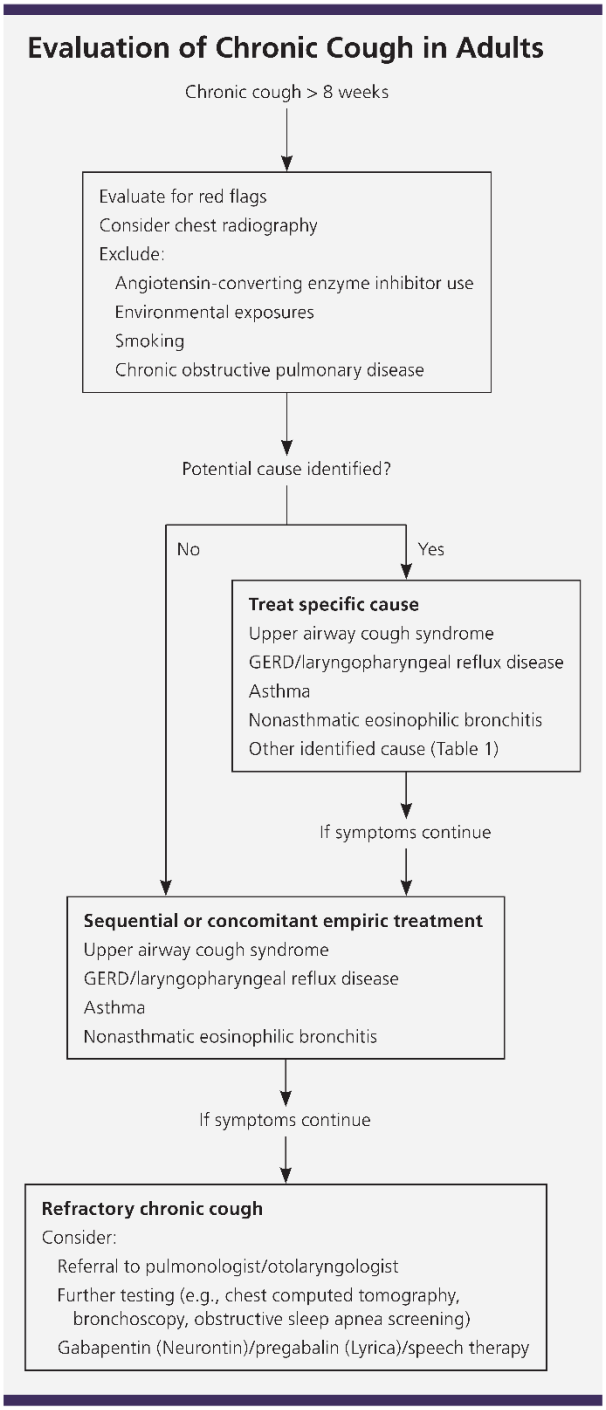


Figure 1. Algorithm for assessment of chronic cough in immunocompetent adults. (GERD = gastroesophageal reflux disease.)

- COPD commonly causes chronic cough, but most patients presenting with chronic cough do not have undiagnosed COPD. Signs and symptoms suggestive of asthma also occur in persons with COPD. Spirometry is diagnostic, and purulent sputum production may also be present. Treatment includes an inhaled bronchodilator, inhaled anticholinergic, inhaled corticosteroid, and a one- to two-week course of oral corticosteroids (with or without antibiotics).

3. Non-asthmatic Eosinophilic Bronchitis

- Nonasthmatic eosinophilic bronchitis is characterized by chronic cough in patients with no symptoms or objective evidence of variable airflow obstruction, normal airway responsiveness on a methacholine inhalation test, and sputum eosinophilia.
- Sputum evaluation is not typically performed in the primary care setting, but it can be induced by saline nebulization or obtained by bronchoalveolar lavage in a subspecialist's office.
- It does not respond to inhaled bronchodilators, but should **respond to inhaled corticosteroids**. Avoidance strategies should be recommended when the inflammation is due to occupational exposure or inhaled allergens.
- Oral corticosteroids are rarely needed but can be considered if high-dose inhaled corticosteroids are ineffective.

4. Gastroesophageal/Laryngopharyngeal Reflux Disease

- Studies have shown an association between GERD and chronic cough, but the pathophysiology is complex and treatment is controversial.
- Associated manifestations such as heartburn, regurgitation, sour taste, hoarseness, and globus sensation are clinical clues.
- Although there is poor evidence that proton pump inhibitors are universally beneficial for GERD-induced chronic cough, consensus guide-lines recommend empiric therapy (proton pump inhibitors) for at least eight weeks in conjunction with lifestyle changes such as dietary changes and weight loss.
- The addition of a histamine H2 receptor antagonist and/or baclofen (Lioresal, 20 mg per day) may be helpful.
- Treatment with continuous positive airway pressure may improve chronic cough by decreasing GERD; therefore, evaluation for obstructive sleep apnea should be considered.
- Surgery can be considered in patients with GERD-associated chronic cough who have abnormal esophageal acid exposure (as proven by pH testing) if normal peristalsis is confirmed on manometry.

5. ACE INHIBITOR–RELATED COUGH

- more common in women.
- It may start within hours to months of the first dose.
- When the medication is discontinued, resolution of the cough should occur within one week to three months; this is the only way to determine if the ACE inhibitor is causing the cough.
- Angiotensin receptor blockers are a good alternative to ACE inhibitors. However, if the patient has a strong indication, restarting the ACE inhibitor may be attempted; in some patients, the cough will not recur.
- When considering ACE inhibitors as the cause of chronic cough, other common causes should also be investigated.

TABLE 2: Clues to Common Causes of Cough That May Be Apparent by History

FINDING	*POSSIBLE DIAGNOSIS*
Infant with cough	Congenital malformations
Patient is a smoker	Tobacco-induced bronchitis
Purulent sputum	Pneumonia, bronchitis
Patient is taking an ACE inhibitor	ACE inhibitor–induced cough
High risk for tuberculosis exposure	Tuberculosis
Wheezing	Asthma
Nocturnal wheezing	Asthma, congestive heart failure
Cough is worse at work	Occupational environment cause
Cough following upper respiratory infection or exposure to allergen	Postnasal drip
Sensation of postnasal drip	Postnasal drip, asthma
Facial pain, tooth pain	Sinusitis
Heartburn or sour taste in mouth	Gastroesophageal reflux disease
History of weight loss	Cancer, tuberculosis

APP

ROACH TO CHEST PAIN

INTRODUCTION

- Approximately 1 % of primary care office visits are for chest pain, and 1.5 % of these patients will have unstable angina or acute myocardial infarction.
- The initial goal in patients presenting with chest pain is to determine if the patient needs to be referred for further testing to rule in or out acute coronary syndrome and myocardial infarction.
- For persons in whom the suspicion for ischemia is lower, other diagnoses to consider include:

- **chest wall pain/costochondritis** (localized pain reproducible by palpation)
- **gastroesophageal reflux disease** (burning retrosternal pain, acid regurgitation, and a sour or bitter taste in the mouth)
- **panic disorder/anxiety state**.

Other less common but important diagnostic considerations include pneumonia (fever, egophony, and dullness to percussion), heart failure, pulmonary embolism (consider using the Wells criteria), acute pericarditis, and acute thoracic aortic dissection (acute chest or back pain with a pulse differential in the upper extremities).

Table 1. Differential Diagnosis of Chest Pain

Diagnosis	*Clinical findings*
Acute myocardial infarction[3]	Chest pain radiates to both arms Third heart sound on auscultation Hypotension
Chest wall pain[4]	At least two of the following findings: localized muscle tension; stinging pain; pain reproducible by palpation; absence of cough
Gastroesophageal reflux disease[5,6]	Burning retrosternal pain, acid regurgitation, sour or bitter taste in the mouth; one-week trial of high-dose proton pump inhibitor relieves symptoms
Panic disorder/anxiety state[7]	Single question: In the past four weeks, have you had an anxiety attack (suddenly feeling fear or panic)?
Pericarditis[8,9]	Clinical triad of pleuritic chest pain (increases with inspiration or when reclining, and is lessened by leaning forward), pericardial friction rub, and electrocardiographic changes (diffuse ST segment elevation and PR interval depression without T wave inversion)
Pneumonia[10,11]	Egophony Dullness to percussion Fever Clinical impression
Heart failure[12]	Pulmonary edema on chest radiography Clinical impression/judgment History of heart failure History of acute myocardial infarction
Pulmonary embolism[13,14]	High pretest probability based on Wells criteria Moderate pretest probability based on Wells criteria Low pretest probability based on Wells criteria
Acute thoracic aortic dissection[15]	Acute chest or back pain and a pulse differential in the upper extremities

- Persons with a higher likelihood of acute coronary syndrome should be referred to the emergency department or hospital.

- Cardiac disease is the leading cause of death in the United States.

- The most common causes of chest pain in the primary care population include chest wall pain (20 %); reflux esophagitis (13 %); and costochondritis (13 %), although in practice, costochondritis is often included in the chest wall pain category. Other considerations include pulmonary (e.g., pneumonia, pulmonary embolism), gastrointestinal (e.g., gastroesophageal reflux disease [GERD]), and psychological (e.g., anxiety, panic disorder) etiologies, and cardiovascular disorders (e.g., acute congestive heart failure, acute thoracic aortic dissection). ***Table 1*** lists the differential diagnosis of chest pain.

INITIAL EVALUATION

- Differentiating ischemic from nonischemic causes often is difficult, and patients with chest pain with an ischemic etiology often appear well. As such, the initial diagnostic approach should always consider a cardiac etiology for the chest pain, unless other causes are apparent.

- The first decision point for most physicians is whether or not the chest pain is caused by coronary ischemia. Acute coronary syndrome (ACS) is a constellation of clinical findings that suggests acute myocardial ischemia encompassing unstable angina and acute MI.

- Angina has been described as deep, poorly localized chest or arm discomfort (pain or pressure) that is reproducibly associated with physical exertion or emotional stress and is relieved promptly with rest or sublingual nitroglycerin.

- Unstable angina is defined as angina at rest, new-onset angina, or angina that has become more severe or longer in duration.

- Acute MI is defined as ST segment changes (elevation or depression) on (ECG) and positive laboratory markers of myocardial necrosis (e.g., troponin I).

- The clinical impression is shaped by the presenting symptoms, physical examination, and initial ECG, combined with the patient's risk of ACS.

- The initial goal is to determine if the patient needs to be referred for further testing (e.g., troponin I or stress testing, coronary angiography) to rule in or out a potentially catastrophic ACS and acute MI.

- Although individual characteristics* may not rule in or out a diagnosis, a combination of signs and symptoms may increase diagnostic accuracy.
  * individual Characteristics traditionally associated with increased likelihood of acute MI include:
  - male sex plus age older than 60 years
  - diaphoresis
  - pain that radiates to the shoulder, neck, arm, or jaw
  - a history of angina or acute MI

- Predictability may be influenced by patient description of their symptoms. Patients often do not use the term pain to describe their symptoms, but frequently use other terms like discomfort, tightness, squeezing, or indigestion.

OTHER CLINICAL FEATURES THAT INCREASE THE LIKELIHOOD OF MI IN PATIENTS WITH ACUTE CHEST PAIN INCLUDE:

- pain that radiates to both arms
- a third heart sound on auscultation
- hypotension.

CLINICAL FEATURES THAT DECREASE THE LIKELIHOOD OF ACUTE MI INCLUDE:

- pleuritic chest pain
- sharp or stabbing chest pain
- chest pain reproduced by palpation.

- The presence or absence of comorbidities, such as diabetes mellitus, tobacco use, hyperlipidemia, or hypertension, as cardiac risk factors weakly predict ACS in patients older than 40 years. however, evaluating for presence or absence of comorbidities is still an important component of the initial assessment.

One recently developed and validated clinical decision rule outlines five items that best predict coronary artery disease as the cause of chest pain:

1) age/sex (55 years or older in men or 65 years or older in women)
2) known coronary artery disease, occlusive vascular disease, or cerebrovascular disease.
3) pain that is worse during exercise.
4) pain not reproducible by palpation.
5) patient assumption that the pain is of cardiac origin.

- patients with chest pain and four or five of these factors require urgent workup.
- Physicians should consider applying a validated clinical decision rule to predict heart disease as a cause of chest pain.

❖ Twelve-lead ECG is typically the test of choice in the initial evaluation of patients with chest pain. ST segment changes (elevation or depression), new-onset left bundle branch block, presence of Q waves, and new-onset T wave inversion increase the likelihood of ACS or acute MI.

❖ Concern based on the clinical impression (history, physical examination, risk factors, and 12-lead ECG) often will influence the physician's decision regarding whether to refer the patient to a higher level of care (emergency department or hospital) for further workup and treatment, or to look for other possible diagnoses for the chest pain.

OTHER DIAGNOSTIC CONSIDERATIONS

❖ If the initial evaluation indicates that a cardiac cause of ACS is less likely, other noncardiac causes of chest pain should be considered. Understanding that there are common conditions that often occur, with the clinical impression, will help lead to a correct diagnosis.

Chest wall pain, reflux esophagitis, and costo-chondritis are the most common causes of chest pain in the primary care population.

CHEST WALL PAIN

four clinical factors that predict a final diagnosis of chest wall pain in patients presenting to the primary care office with chest pain:

1) localized muscle tension
2) stinging pain
3) pain reproducible by palpation
4) the absence of a cough.

Having at least two of these findings had a 77 % positive predictive value for chest wall pain, and having none or one had an 82% negative predictive value.

COSTOCHONDRITIS

❖ Often considered a subset of chest wall pain, costochondritis is a self-limited condition characterized by **pain reproducible by palpation** in the parasternal/costochondral joints.

❖ It is sometimes called Tietze syndrome, which is distinguished from costochondritis by the presence of swelling over the affected joints.

❖ Costochondritis is a clinical diagnosis and does not require specific diagnostic testing in the absence of concomitant cardiopulmonary symptoms or risk factors.

GERD

❖ Classic symptoms of GERD include a burning retrosternal pain, acid regurgitation, and a sour or bitter taste in the mouth.

❖ No useful physical examination maneuvers exist to assist in establishing the diagnosis and there is no standard test to rule it in or out.

❖ However, a one-week trial of a high-dose proton pump inhibitor is modestly sensitive and specific for GERD.

PANIC DISORDER AND ANXIETY STATE

❖ Panic disorder and anxiety state are common. One in four persons with a panic attack will have chest pain and shortness of breath.

- Yet, concomitant panic disorder and chest pain are often not recognized, leading to more testing, follow-up, and higher costs of care.
- Panic may cause chest pain and vice versa.
- Several validated brief questionnaires are used to diagnose panic disorder and anxiety state. One question ***(In the past four weeks, have you had an anxiety attack [suddenly feeling fear or panic]?)*** is sensitive (93 %) and modestly specific (78 %) in detecting panic disorder.

LESS COMMON, BUT IMPORTANT, DIAGNOSTIC CONSIDERATIONS

PERICARDITIS

- Pericarditis is the clinical triad of:

1) pleuritic chest pain
2) pericardial friction rub
3) diffuse (ECG) ST-T wave changes.

- ECG usually demonstrates diffuse ST segment elevation and PR interval depression without T wave inversion.
- Acute pericarditis should be considered in patients presenting with new-onset chest pain that increases with inspiration or when reclining, and is lessened by leaning forward.

PNEUMONIA

- Community-acquired pneumonia is a cause of chest pain and respiratory symptoms in the outpatient setting.
- Common symptoms include fever, chills, productive cough, and pleuritic chest pain.
- Fever, egophony heard during auscultation of the lungs, and dullness to percussion of the posterior thorax are suggestive of pneumonia.
- **Clinical impression is modestly useful for ruling in or out pneumonia.**
- **The test of choice for diagnosing pneumonia is chest radiography**, although it has been more recently recommended that it be performed only if other diagnoses are being considered in the uncomplicated outpatient setting.

HEART FAILURE

- Most patients with heart failure present with dyspnea on exertion, although some will have chest pain.
- A history of heart failure or acute MI best predicts the presence of heart failure.
- Clinical impression/judgment is predictive of heart failure.

PULMONARY EMBOLISM

- Diagnosing pulmonary embolism in the office based on signs and symptoms is difficult because of its highly variable presentation.
- Although dyspnea, tachycardia, and/or chest pain are present in 97 % of those diagnosed with pulmonary embolism, there is no single clinical feature that effectively rules it in or out.

Table 3. Wells Clinical Prediction Rule for PE

Component	*Points*
Clinical signs of DVT (asymmetric leg swelling, palpable calf pain)	3
Diagnosis of PE is more likely than an alternative diagnosis	3
Heart rate greater than 100 beats per minute	1.5
Previous diagnosis of DVT or PE	1.5
Bed rest immobilization or surgery within the past four weeks	1.5
Hemoptysis	1
Malignancy within the past six months	1

Probability of PE

Points	*Risk of PE*	*Probability of PE (%)*
0 to 1 point	Low	1.3
2 to 6 points	Moderate	16
Greater than 6 points	High	41

DVT = deep venous thrombosis; PE = pulmonary embolism.

- The physician can estimate the patient's likelihood of pulmonary embolism by using a validated clinical decision rule, such as the Wells criteria (Table 3), to determine if further testing should be performed (e.g., D-dimer assay, ventilation-perfusion scan, helical computed tomography of the pulmonary arteries).

ACUTE THORACIC AORTIC DISSECTION

Patients with acute thoracic aortic dissection may present with chest or back pain.

History and physical examination are only modestly useful for ruling in or out the condition; acute chest or back pain and a pulse differential in the upper extremities modestly increase the likelihood of an acute thoracic aortic dissection.

DYSLIPIDEMIA

INTRODUCTION

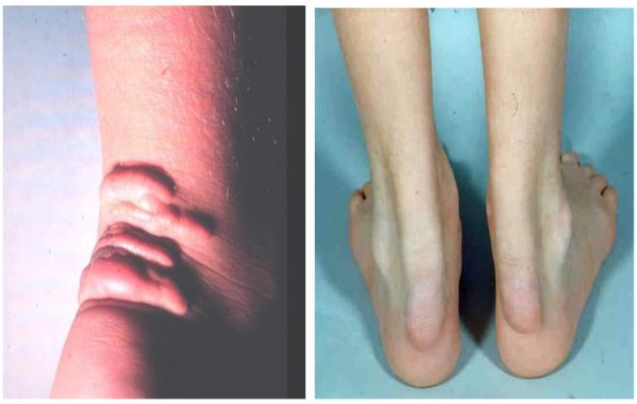
- Dyslipidemia: A disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency.

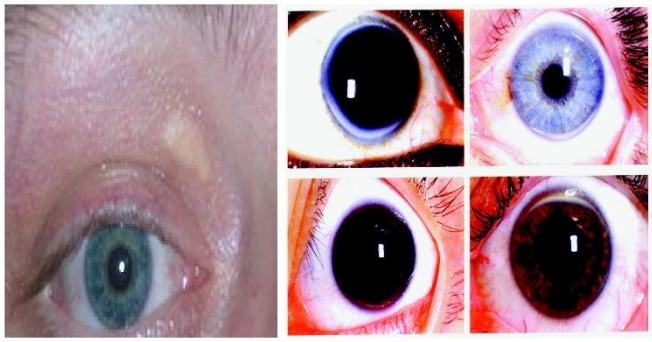
- Dyslipidemias may be manifested by elevation of the total cholesterol, the "bad" low density lipoprotein (LDL) cholesterol and triglyceride concentrations, and a decrease in the "good" high-density lipoprotein (HDL) cholesterol concentration in the blood.

Values of Lipids		
LDL	< 100	Optimal
	100-129	Near optimal
	130-159	Borderline
	160-189	High
	≥ 190	Very High
Total Cholesterol	< 200	Desirable
	200-239	Borderline
	≥240	High
HDL	< 40	Low
	≥ 60	High
Serum Triglycerides	< 150	normal
	150-199	Borderline
	200-499	High
	≥ 500	Very High

SCREEN FOR Dyslipidemia

1) Men over 35 and woman 0ver 45.
2) Anyone with atherosclerotic symptoms regardless of age
3) Anyone with diabetes regardless of age
4) Family history of premature CVD
5) Inflammatory diseases (lupus, rheumatoid arthritis, psoroasis)
6) Children of patients with severe dyslipidemia
7) HIV infection with HAART therapy
8) Clinical signs of hyperlipidemia; Xanthelasma, tendon xanthoma, corneal arcus.
9) Erectile dysfunction
10) Chronic renal disease
11) Metabolic syndrome?





THE GOAL FOR DYSLIPIDEMIA TREATMENT (PRIMARY PREVENTION VS. SECONDARY PREVENTION) :

PRIMARY PREVENTION FOR:

1- LDL-C >=190 mg/dl

2- Diabetes and aged 40-75 years with LDL-C between 70-189 mg/dl

3- No diabetes and estimated 10 year ASCVD risk of >= 7.5 % who are between 40 to 75 years of age with LDL-C between 70-189 mg/dl

SECONDARY PREVENTION FOR:

1- Patients with known coronary heart disease (CHD; including myocardial infarction, angina, and prior coronary revascularization)

2- other cardiovascular disease (CVD; including stroke, transient ischemic attack, and peripheral arterial disease)

3- combinations of risk factors that result in a 10-year risk of ASCVD events of more than 20 %

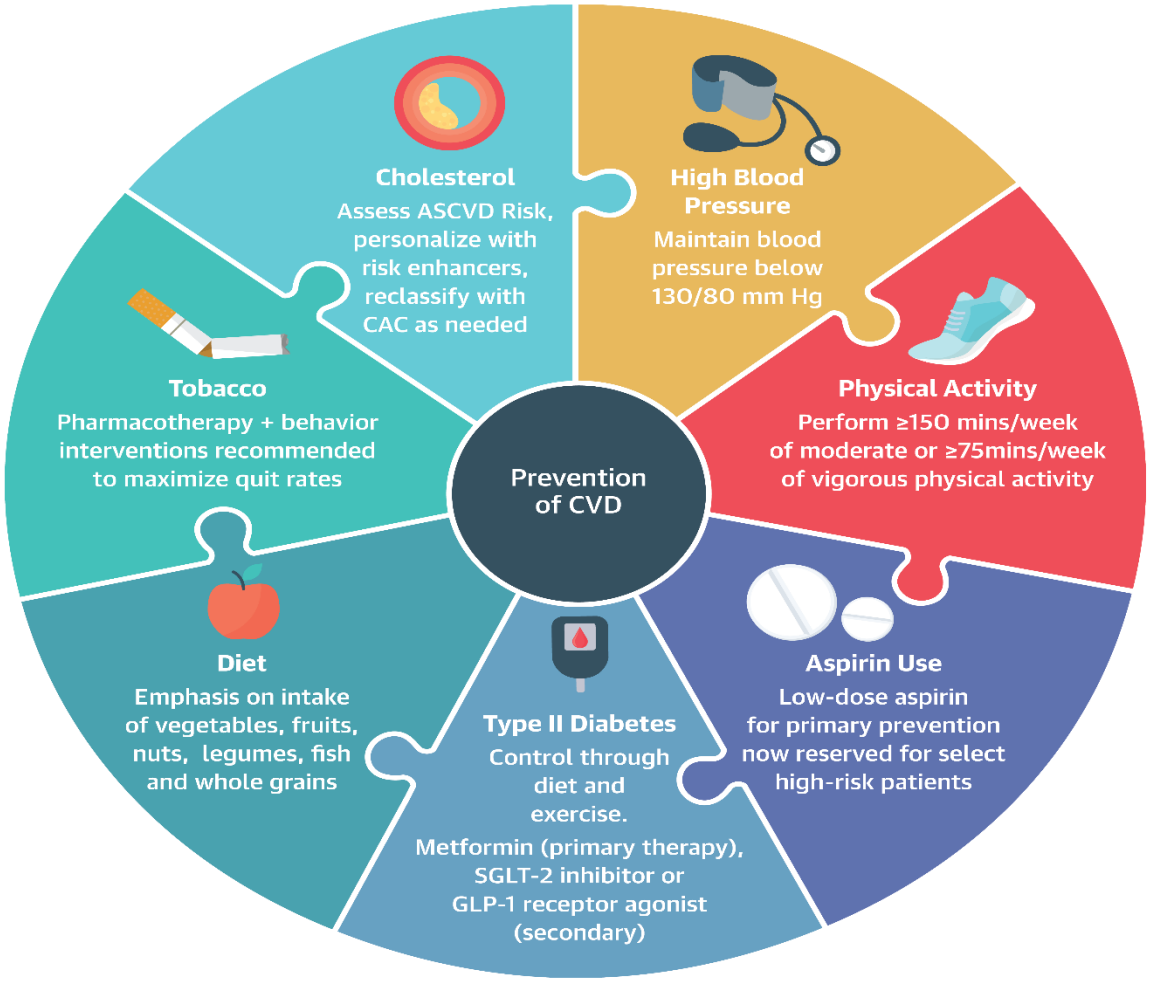
4- Chronic kidney disease with estimated GFR <45ml/min/1.73m^2

5- Risk equivalent for CV in diabetic patients:

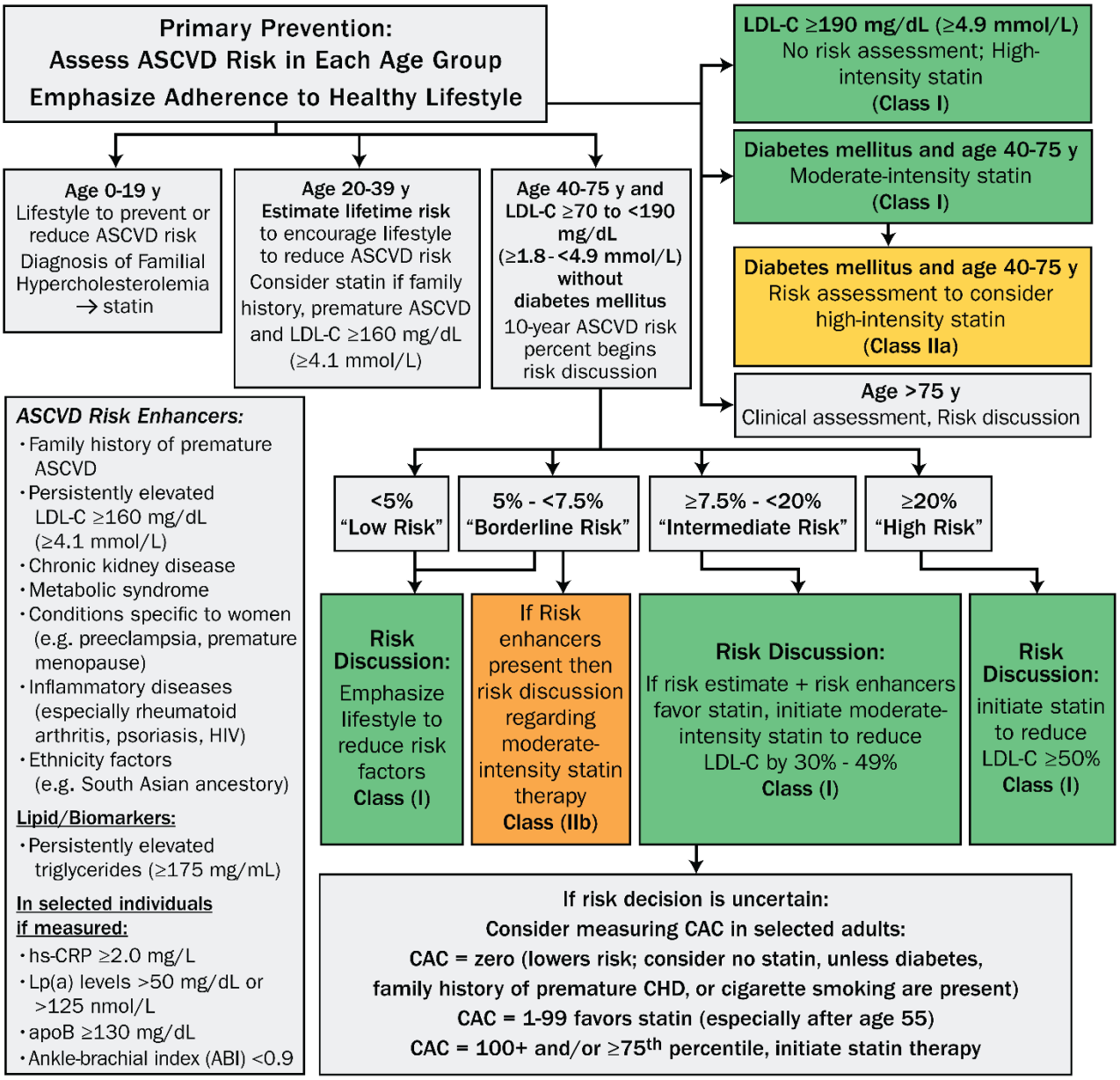
- Although some guidelines have considered all patients with diabetes mellitus (DM) to have a risk of CV events similar to patients with known CVD, this actually averages events across patients with widely differing risks of CHD. Issues that may affect risk with DM include patient age, sex, other CV risk factors, duration of DM, and whether the patient has type 1 or type 2 DM.

- Given this, it is preferable to calculate patient-specific risks rather than to simply consider all patients with DM to require treatment for secondary prevention, particularly in patients who are under age 60 without multiple cardiovascular risk factors. A downloadable calculator for this purpose is available for patients with type 2 DM from the UK Prospective Diabetes Study (www.dtu.ox.ac.uk/riskengine)

Primary Prevention: Lifestyle Changes and Team-Based Care

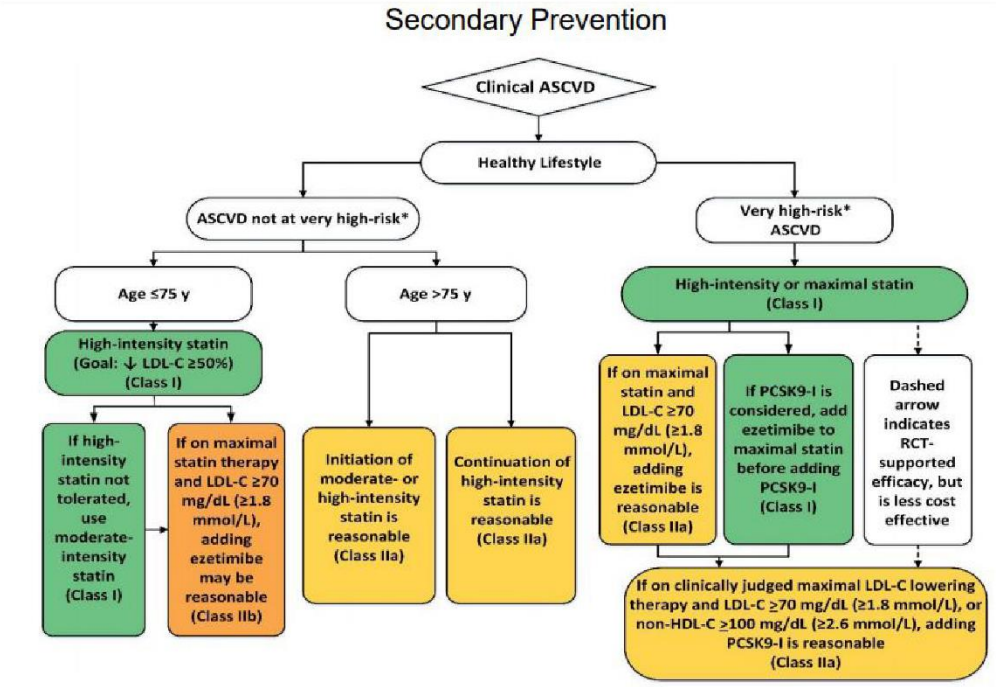


Primary Prevention



Risk-Enhancing Factors for Clinician-Patient Risk Discussion

- **Family history of premature ASCVD** (males, age <55 y; females, age <65 y)
- **Primary hypercholesterolemia** (LDL-C, 160–189 mg/dL [4.1–4.8 mmol/L]; non–HDL-C 190–219 mg/dL [4.9–5.6 mmol/L])*
- **Metabolic syndrome** (increased waist circumference [by ethnically appropriate cutpoints], elevated triglycerides [>150 mg/dL, nonfasting], elevated blood pressure, elevated glucose, and low HDL-C [<40 mg/dL in men; <50 mg/dL in women] are factors; a tally of 3 makes the diagnosis)
- **Chronic kidney disease** (eGFR 15–59 mL/min/1.73 m2 with or without albuminuria; not treated with dialysis or kidney transplantation)
- **Chronic inflammatory conditions**, such as psoriasis, RA, lupus, or HIV/AIDS
- **History of premature menopause (before age 40 y) and history of pregnancy-associated conditions that increase later ASCVD risk, such as preeclampsia**
- **High-risk race/ethnicity** (e.g., South Asian ancestry)
- **Lipids/biomarkers**: associated with increased ASCVD risk
  - Persistently elevated,* primary hypertriglyceridemia (≥175 mg/dL, nonfasting)
  - If measured:
    - **Elevated high-sensitivity C-reactive protein** (≥2.0 mg/L)
    - **Elevated Lp(a)**: A relative indication for its measurement is family history of premature ASCVD. An Lp(a) ≥50 mg/dL or ≥125 nmol/L constitutes a risk-enhancing factor, especially at higher levels of Lp(a).
    - **Elevated apoB** (≥130 mg/dL): A relative indication for its measurement would be triglyceride ≥200 mg/dL. A level ≥130 mg/dL corresponds to an LDL-C >160 mg/dL and constitutes a risk-enhancing factor
    - **ABI** (<0.9)



PHARMACOLOGICAL TREATMENT:

- When a pharmacologic agent is required for treatment in primary prevention, a statin is the preferred medication.
- If a statin is not tolerated or a particular LDL-C goal is not achieved on a statin alone, we suggest administering or adding a non-statin lipid-lowering medication; Ezetimibe.
- Cardiovascular risk should be calculated by Pooled Cohort Equations CV risk calculator (ACC/AHA Calculator) to determine 10 year risk factor for CV events.
- 10 year ASCVD risk: a quantitative estimation of absolute risk based upon data from representative population samples. Example: if 10 year ASCVD risk estimates is 10%, this indicates that among 100 patients with the entered risk factor profile, 10 would be expected to have a heart attack or stroke in the next 10 years.

STATIN THERAPY:

1) High-Intensity Statin Daily dose lowers LDL-C, on average by approximately ≥50%:
- Atorvastatin 40-80 mg
- Rosuvastatin 20-(40) mg

2) Moderate-Intensity Statin Daily dose lowers LDL-C, on average by approximately 30% to <50%:
- Atorvastatin 10-(20) mg
- Rosuvastatin (5)-10 mg
- Simvastatin 20-40 mg

3) Low-Intensity Statin Daily dose lowers LDL-C, on average by approximately <30%:
- Fluvastatin 20-40 mg
- Simvastatin 10 mg

STATIN SAFETY RECOMMENDATIONS

- Characteristics predisposing individuals to statin adverse effects include, but are not limited to:

1. Multiple or serious comorbidities, including impaired renal or hepatic function.

2. History of previous statin intolerance or muscle disorders. It is reasonable to evaluate and treat muscle symptoms, including pain, tenderness, stiffness, cramping, weakness, or fatigue, in statin-treated patients according to the following management algorithm:

1. If unexplained severe muscle symptoms or fatigue develop during statin therapy, promptly discontinue the statin and address the possibility of rhabdomyolysis by evaluating CK, creatinine, and urinalysis for myoglobinuria.

2. If mild to moderate muscle symptoms develop during statin therapy:

A- Discontinue the statin until the symptoms can be evaluated.

B- Evaluate the patient for other conditions that might increase the risk for muscle symptoms (e.g., hypothyroidism, reduced renal or hepatic function, rheumatologic disorders such as polymyalgia rheumatica, steroid myopathy, vitamin D deficiency, or primary muscle diseases).

C- If muscle symptoms resolve, and if no contraindication exists, give the patient the original or a lower dose of the same statin to establish a causal relationship between the muscle symptoms and statin therapy.

D- Once a low dose of a statin is tolerated, gradually increase the dose as tolerated.

E- If, after 2 months without statin treatment, muscle symptoms or elevated CK levels do not resolve completely, consider other causes of muscle symptoms listed above.

F- If persistent muscle symptoms are determined to arise from a condition unrelated to statin therapy, or if the predisposing condition has been treated, resume statin therapy at the original dose.

3. Unexplained ALT elevations >3 times ULN.

A. Baseline measurement of hepatic transaminase levels (ALT) should be performed before initiating statin therapy.

B. During statin therapy, it is reasonable to measure hepatic function if symptoms suggesting hepatotoxicity arise (e.g., unusual fatigue or weakness, loss of appetite, abdominal pain, dark colored urine or yellowing of the skin or sclera).

4. Patient characteristics or concomitant use of drugs affecting statin metabolism.

5. >75 years of age - For individuals taking any dose of statins, it is reasonable to use caution in individuals >75 years of age, as well as in individuals that are taking concomitant medications that alter drug metabolism; cyclosporine, macrolides, various antifungals, cytochrome p-450 inhibitors, can cause drug interaction leads to high incidence of myositis., taking multiple drugs, or taking drugs for conditions that require complex medication regimens (e.g., those who have undergone solid organ transplantation or are receiving treatment for HIV). A review of the manufacturer's prescribing information may be useful before initiating any cholesterol-lowering drug. (IIa C)

NON-STATIN THERAPY:

EZETIMIBE

modestly lowers the LDL-C when used alone and may be helpful for avoiding high doses of statins (and potentially increased susceptibility to muscle injury) in patients who do not meet cholesterol goals on statin therapy alone. However, the clinical benefits of either ezetimibe monotherapy or combining ezetimibe with statin therapy remain to be proven.

FIBRATES

the major effects of the fibrates are to lower plasma triglyceride and raise HDL-C levels. They are effective for the treatment of hypertriglyceridemia and combined hyperlipidemia with or without hypoalphalipoproteinemia. There is an increased risk of muscle toxicity in patients taking some fibrates and a statin.

PCSK9 INHIBITORS

are a class of injectable monoclonal antibodies approved in 2015 that have been shown to dramatically lower LDL cholesterol levels -- by up to 60% in some cases when combined with a statin.

NICOTINIC ACID

Nicotinic acid (niacin) is effective in improving lipid parameters in patients who have hypercholesterolemia or combined hyperlipidemia associated with normal and low levels of HDL-C (hypoalphalipoproteinemia). The HDL raising properties of nicotinic acid occur with dosages as low as 1 to 1.5 g/day.The use of nicotinic acid is often limited by poor tolerability, and there are concerns about the safety of nicotinic acid as well as its efficacy for clinical endpoints.

BILE ACID SEQUESTRANTS

Bile acid sequestrants are effective in patients with mild to moderate elevations of LDL-C. Bile acid sequestrants are also effective when used in combination with a statin or nicotinic acid in patients with markedly elevated plasma levels of LDL-C . The use of a bile acid sequestrant is often limited by side effects.

TAKE-HOME MESSAGES FOR THE PRIMARY PREVENTION OF CARDIOVASCULAR DISEASE

1) The most important way to prevent atherosclerotic vascular disease, heart failure, and atrial fibrillation is to promote a healthy lifestyle throughout life.

2) A team-based care approach is an effective strategy for the prevention of cardiovascular disease. Clinicians should evaluate the social determinants of health that affect individuals to inform treatment decisions.

3) Adults who are 40 to 75 years of age and are being evaluated for cardiovascular disease prevention should undergo 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimation and have a clinician–patient risk discussion before starting on pharmacological therapy, such as antihypertensive therapy, a statin, or aspirin. In addition, assessing for other risk-enhancing factors can help guide decisions about preventive interventions in select individuals, as can coronary artery calcium scanning.

4) All adults should consume a healthy diet that emphasizes the intake of vegetables, fruits, nuts, whole grains, lean vegetable or animal protein, and fish and minimizes the intake of trans fats, red meat and processed meats, refined carbohydrates, and sugar-sweetened beverages.

For adults with overweight/obesity, counseling and caloric restriction are recommended for achieving and maintaining weight loss.

5) Adults should engage in at least 150 minutes per week of accumulated moderate-intensity physical activity or 75 minutes per week of vigorous-intensity physical activity.

6) For adults with type 2 diabetes mellitus, lifestyle changes, such as improving dietary habits and achieving exercise recommendations, are crucial. If medication is indicated, metformin is first-line therapy, followed by consideration of a sodium-glucose cotransporter 2 inhibitor or a glucagon-like peptide-1 receptor agonist.

7) All adults should be assessed at every healthcare visit for tobacco use, and those who use tobacco should be assisted and strongly advised to quit.

8) Aspirin should be used infrequently in the routine primary prevention of ASCVD because of lack of net benefit.

9) Statin therapy is first-line treatment for primary prevention of ASCVD in patients with elevated low-density lipoprotein cholesterol levels (≥190 mg/dL), those with diabetes mellitus, who are 40 to 75 years of age, and those determined to be at sufficient ASCVD risk after a clinician–patient risk discussion.

OSTEOPOROSIS

DEFINITION

A skeletal disease characterized by low bone mass, deterioration of bone tissue, and disruption of bone architecture that leads to compromised bone strength and an increased risk of fracture.

RISK FACTORS

❖ sun exposure

NONMODIFIABLE

1) Advanced age (>65 years)
2) Female gender and menopause
3) Caucasian or Asian
4) Family history of osteoporosis
5) History of atraumatic fracture

MODIFIABLE

1) Low body weight (58 kg or body mass index [BMI] <21)
2) Calcium/vitamin D deficiency
3) Inadequate physical activity
4) Cigarette smoking
5) Excessive alcohol intake (>3 drinks/day)
6) Medications

GENERAL PREVENTION

The aim in the prevention and treatment of osteoporosis is to prevent fractures:

❖ Regularly perform weight-bearing exercise.

❖ Consume a diet that includes adequate calcium (1,000 mg/day for men aged 50 to 70 years; 1,200 mg/day for women aged 51+ years and men 70+ years) and vitamin D (800 to 1,000 IU/day).

❖ The USPSTF has concluded that vitamin D supplementation is effective in preventing falls in community-dwelling adults aged 65 years or older who are at increased risk for falls .

❖ Avoid smoking.

❖ Limit alcohol consumption (<3 drinks/day).

❖ Fall prevention (vitamin D supplementation, home safety assessment, correction of visual impairment)

SCREENING (USPSTF RECOMMENDATIONS):

– All women ≥65 years of age (

– Women >50 years of age with 10-year fracture risk; Fracture Risk Assessment [FRAX] Tool >9.3%

– The current evidence is insufficient to recommend screening for osteoporosis in men; however, the National Osteoporosis Foundation recommends screening men age >70 years, especially if at increased risk.

COMMONLY ASSOCIATED CONDITIONS

❖ Malabsorption syndromes: gastrectomy, inflammatory bowel disease, celiac disease

❖ Hypoestrogenism: menopause, hypogonadism, eating disorders, female athlete triad

❖ Endocrinopathies: hyperparathyroidism, hyperthyroidism, hypercortisolism, diabetes mellitus

❖ Hematologic disorders: hemophilia, sickle cell disease, multiple myeloma, thalassemia, hemochromatosis

❖ Other disorders: multiple sclerosis, end-stage renal disease, rheumatoid arthritis, lupus, chronic obstructive pulmonary disease (COPD), HIV/AIDS

❖ Medications: antiepileptics, aromatase inhibitors (raloxifene), chronic corticosteroids (>5-mg prednisone or equivalent for >3 months), medroxyprogesterone acetate, heparin, SSRI, thyroid hormone (in supraphysiologic doses), PPI

DIFFERENTIAL DIAGNOSIS

1) Multiple myeloma/other neoplasms
2) Osteomalacia
3) Type I collagen mutations
4) Osteogenesis imperfecta

DIAGNOSIS

- DEXA of the lumbar spine/hip is the gold standard for measuring bone mineral density (BMD).
- A BMD at the hip or lumbar spine that is ≤2.5 standard deviations (SDs) below the mean BMD of a young-adult reference population is diagnostic of osteoporosis.
- A minimum of 2 years may be needed to reliably measure a change in BMD.
- BMD is expressed in terms of T-scores and Z-scores:

1) T-score is the number of SDs a patient's BMD deviates from the mean for young, normal (age 25 to 40 years) control individuals of the same sex.

2) WHO defines normal BMD as a T-score ≥−1, osteopenia as a T-score between −1 and −2.5, and osteoporosis as a T-score ≤−2.5.

3)WHO thresholds can be used for ostmenopausal women and men >50 years of age.

4) The Z-score is a comparison of the patient's BMD with an age-matched Population.

TREATMENT

Criteria for patients who benefit from treatment for their osteoporosis includes:

– All patients with a T-score ≤−2.5 with no risk factors
– All postmenopausal women and men >50 years old who have had an osteoporotic vertebral/hip fracture

– All postmenopausal women who have BMD values consistent with osteoporosis (T-score ≤2.5) at the lumbar spine, femoral neck, or total hip Region

– Postmenopausal women and men >50 years old with T-scores from −1.0 to−2.5 and a 10-year risk, based on FRAX calculator of an osteoporotic fracture (spine, hip, shoulder, and wrist) of at least 20% or hip of at least 3%.

MEDICATION

FIRST LINE

1) **Bisphosphonates:**
- **Alendronate 10 mg PO daily or 70 mg PO weekly**
- **Risedronate**

2) **Zoledronic acid IV**

SIDE EFFECTS :

1.gastrointestinal problems such as difficulty swallowing and inflammation of the esophagus and stomach.

2.Osteonecrosis of the jaw has been associated with bisphosphonates, particularly in patients with cancer who receive high doses and those who receive IV treatment .

3.There is a possible risk of midfemur fractures in patients receiving bisphosphonates for >5 years.

Avoid oral bisphosphonates in patients with:

- Delayed esophageal emptying
- Inability to stand/sit upright for at least 30 to 60 minutes after taking the bisphosphonates
- Hypocalcemia (correct prior to initiating therapy)

- Severe renal impairment

SECOND LINE

- Denosumab: SQ every 6 months; Human monoclonal antibody
- Teriparatide 20 mg SC daily; Recombinant formulation of PTH
- **Estrogen 0.625 mg PO daily (with progesterone if patient has a uterus):** effective in prevention and treatment of osteoporosis; the risks (e.g., increased rates of myocardial infarction, stroke, breast cancer, pulmonary embolus, and deep vein thrombosis) must be weighed against the benefits (Women's Health Initiative)
- Raloxifene; a selective estrogen receptor modulator
- Calcitonin

PATIENT MONITORING

- ❖ BMD testing by DEXA scanning retesting 2 years after starting bisphosphonate therapy.
- ❖ For many women, 3 to 5 years of treatment with a bisphosphonate is as good as 10 years of treatment.
- ❖ A comprehensive risk assessment should be performed after 3 to 5 years of treatment. Those at high risk for vertebral fracture or with very low BMD may benefit by continuing treatment beyond 5 years.
- ❖ Physicians prescribing bisphosphonates should advise patients of the small risk of osteonecrosis and encourage dental examinations

CLINICAL PEARLS

- ❖ Regular weight-bearing exercise from adolescence onward is recommended for prevention.
- ❖ Screen all women ≥65 years of age with DEXA scans.
- ❖ Premenopausal women with osteoporosis should be screened for secondary causes such as malabsorption syndromes, hyperparathyroidism, hyperthyroidism, and medication sensitivity.
- ❖ Evaluate and treat all patients presenting with fractures from minimal trauma. -Bisphosphonates are first line for treatment of osteoporosis in most patients. -If the patient is not responding to treatment, consider screening for a secondary, treatable cause of osteoporosis.

HYPERTENSION

1. HYPERTENSION, ESSENTIAL

INTRODUCTION

- ❖ Essential hypertension (HTN) is HTN without an identifiable cause; it is also known as primary HTN and benign HTN.

- Although its importance as a risk factor for cardiovascular and other morbidity and mortality is well-established, there is significant and increasing controversy regarding recommended thresholds for diagnosis and treatment.

- HTN is defined (Joint National Committee [JNC] 8) as ≥2 elevated BPs:
  – Age <60 years: systolic BP (SBP) ≥140 mm Hg and/or diastolic BP (DBP) ≥90 mm Hg at ≥2 visits

  – Age 60 years or older: SBP ≥150 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits

  – With diabetes or chronic kidney disease: SBP ≥140 and/or DBP ≥90 mm Hg

- Synonym(s): benign, chronic, idiopathic, familial, or genetic HTN; high BP

GERIATRIC CONSIDERATIONS

- Isolated systolic HTN is common.

- Therapy has been shown to be effective and beneficial at preventing stroke, although target SBP is higher than in younger patients (~150 mm Hg systolic), and adverse reactions to medications are more frequent.

- The benefit of therapy has been conclusively demonstrated in older patients for SBP ≥160 mm Hg. More aggressive targets may be appropriate for higher risk individuals.

PEDIATRIC CONSIDERATIONS

- Measure BP during routine exams for >3 years of age.

- Defined as SBP or DBP ≥95th percentile on repeated measurements

- Pre-HTN: SBP or DBP between 90th and 95th percentile

PREGNANCY CONSIDERATIONS

- Elevated BP during pregnancy may be either chronic HTN or pregnancy- induced HTN or preeclampsia.
- ACE inhibitors and angiotensin II receptor blockers (ARBs) are contraindicated.
- Maternal and fetal mortality benefit from treatment of severe HTN. Evidence is not clear for mild HTN.
- Preferred agents: methyldopa, labetalol, hydralazine, or nifedipine

EPIDEMIOLOGY

Incidence

Incidence and prevalence is higher among men. Depending on the definition used, 32–46% of adults in the United States have HTN.

ETIOLOGY AND PATHOPHYSIOLOGY

- >90% of cases of HTN have no identified cause.
- For differential diagnosis and causes of secondary HTN, see "Hypertension, Secondary and Resistant."

GENETICS

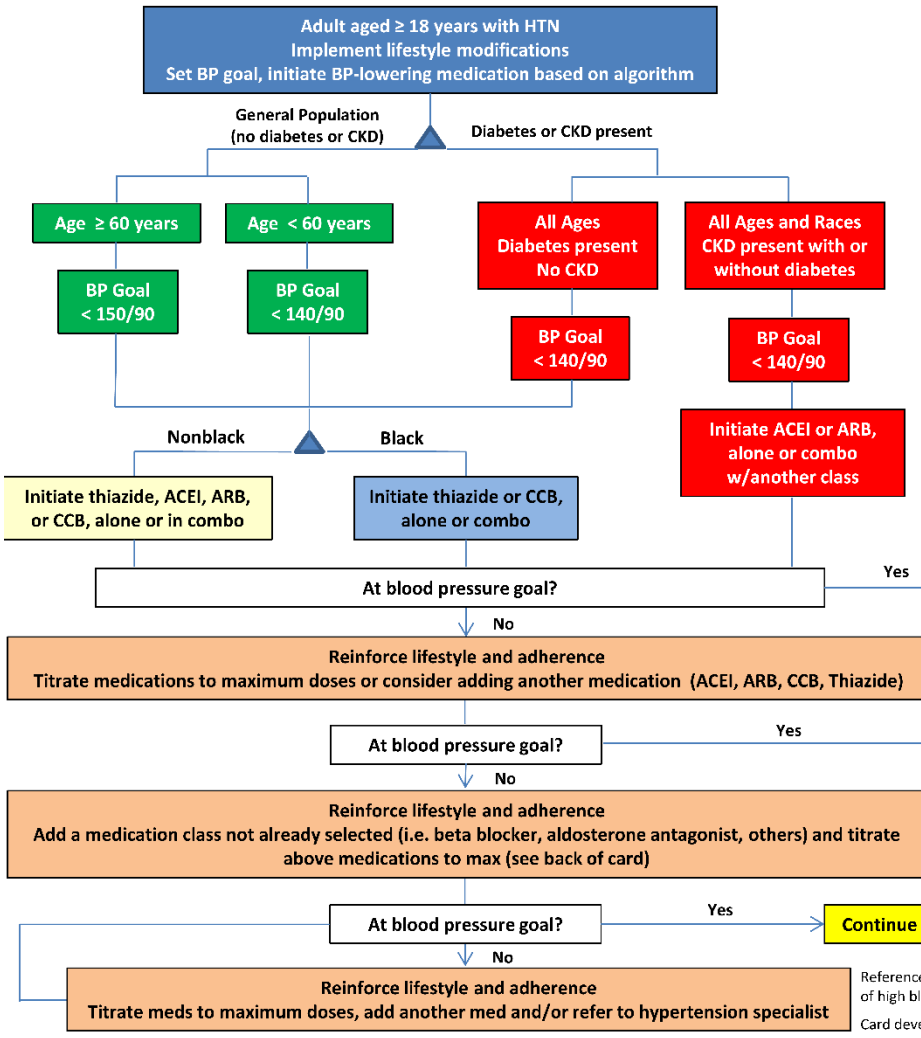
BP levels are strongly familial, but no clear genetic pattern exists. Familial risk for cardiovascular diseases should be considered.

RISK FACTORS

1) Family history
2) Obesity
3) alcohol use
4) excess dietary sodium
5) stress
6) physical inactivity
7) tobacco use
8) insulin resistance

DIAGNOSIS

JNC 8 Hypertension Guideline Algorithm



Initial Drugs of Choice for Hypertension
- ACE inhibitor (ACEI)
- Angiotensin receptor blocker (ARB)
- Thiazide diuretic
- Calcium channel blocker (CCB)

Strategy	Description
A	Start one drug, titrate to maximum dose, and then add a second drug.
B	Start one drug, then add a second drug before achieving max dose of first
C	Begin 2 drugs at same time, as separate pills or combination pill. **Initial combination therapy is recommended if BP is greater than 20/10mm Hg above goal**

Lifestyle changes:
- Smoking Cessation
- Control blood glucose and lipids
- Diet
  - ✓Eat healthy (i.e., DASH diet)
  - ✓Moderate alcohol consumption
  - ✓Reduce sodium intake to no more than 2,400 mg/day
- Physical activity
  - ✓Moderate-to-vigorous activity 3-4 days a week averaging 40 min per session.

Reference: James PA, Ortiz E, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: (JNC8). JAMA. 2014 Feb 5;311(5):507-20

Card developed by Cole Glenn, Pharm.D. & James L Taylor, Pharm.D.

HISTORY

- ❑ HTN is asymptomatic except in extreme cases or after related cardiovascular complications develop.
- ❑ Headache can be seen with higher BP, often present on awakening and occipital in nature.

PHYSICAL EXAM

1) Body mass index (BMI), waist circumference
2) BP in both arms
3) Complete cardiac and peripheral pulse exam: Compare radial and femoral pulse for differences in volume and timing, auscultation for carotid and femoral bruits.
4) Evaluate for signs of end-organ damage.
5) Funduscopic exam for arteriolar narrowing, AV compression, hemorrhages, exudates, and papilledema.

DIFFERENTIAL DIAGNOSIS

1) **Secondary HTN**: Because of the low incidence of reversible secondary HTN, special tests should be considered only if the history, physical exam, or basic laboratory evaluation indicate the possibility or for patients who prove nonresponsive to treatment. (See "Hypertension, Secondary and Resistant.")
2) **White coat HTN**: elevation of BP in office setting and normal BP outside office
3) **Masked HTN**: elevated BP at home and normal BP in office

DIAGNOSTIC TESTS & INTERPRETATION

❖ Measuring BP:
– Caffeine, exercise, and smoking avoided >30 minutes before measurement
– Patient seated quietly for 5 minutes with feet on floor
– Patient's arm supported at heart level
– Correct cuff size
– Average of two or more measurements
– Avoid "rounding" results.

❖ A diagnosis of HTN should be made under the following circumstances:

– **Age <60 years**: SBP ≥140 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits

– **Age 60 years or older**: SBP ≥150 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits

– **Age 60 years or older with CKD or diabetes**: SBP ≥140 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits

INITIAL TESTS (LAB, IMAGING)

1) Hemoglobin or hematocrit or CBC
2) Complete urinalysis (may reveal proteinuria)
3) Potassium, calcium, creatinine, and uric acid
4) Lipid panel (total, HDL, LDL, triglyceride [TG])
5) Fasting blood glucose or hemoglobin A1c
6) ECG to evaluate possible presence of left ventricular hypertrophy (LVH) or rhythm abnormalities

FOLLOW-UP TESTS & SPECIAL CONSIDERATIONS

❑ Special tests only if suggested by history, physical, or labs. In particular, consider possibility of sleep apnea.

❑ Ambulatory (24-hour) BP monitoring if "white coat" HTN is suspected, episodic HTN, or autonomic dysfunction

❑ Home BP monitoring is effective, especially when white coat HTN is a consideration; elevated home BPs correlate with adverse outcomes, possibly more so than office BPs, and normal readings are reassuring.

TREATMENT

GENERAL MEASURES

❖ The treatment discussed follows JNC 8 guidelines.

❖ Some recent studies have concluded that more intensive therapy may be warranted for certain high-risk individuals.

❖ Recommend lifestyle improvements, including diet, exercise, and reducing or eliminating tobacco/alcohol.

❖ Benefit of pharmacologic treatment of low-risk patients with class I HTN (SBP 140 to 150 mm Hg, DBP 90 to 99 mm Hg) remains uncertain.

❖ Treating patients age <60 years or with CKD or diabetes to lower than standard BP targets, <140/90 mm Hg, does not further reduce mortality or morbidity.

❖ Individualize goal BP based on risk factors.

❖ Target SBP at or just below 150 mm Hg in patients >60 years of age is acceptable in the general population.

❖ Majority of treatment benefit is attained with initial 2 to 3 medications.

❖ Striving for small additional drops in BP to achieve a "target" is less clinically beneficial and more likely to cause adverse effects.

❖ Lower than standard DBP targets are not associated with decreased morbidity/mortality.

- Assess overall risk and individualize treatment decisions.
- The decision to treat more aggressively should be considered in patients meeting enrollment criteria for SPRINT, as aggressive treatment does show improvement in outcomes, but 61 nondiabetic patients would need to be treated to a goal of SBP <120 to prevent a major cardiovascular outcome and 90 to prevent one death

MEDICATION

- Multiple drugs at submaximal dose may achieve target BP with fewer side effects. In patients on >1 medication, divide between morning and nighttime for better 24-hour antihypertensive effect.
- Sequential monotherapy attempts should be tried with different classes because individual responses vary.
- Many patients will require multiple medications.
- For initial monotherapy, choose from one of four classes of medications: ACE inhibitors, ARBs, calcium channel blockers (CCBs), or diuretics.
- Thiazide diuretics or CCB preferred as first line in the general black population Chlorthalidone has a longer half-life than HCTZ and is the preferred diuretic based on established trial evidence of benefit.
- If concomitant conditions, choose first-line agent based on comorbidity.
- β-Blockers had been strongly recommended until recent meta-analyses.
- Atenolol may be particularly ineffective in reducing adverse outcomes of HTN (except in patients with left ventricle hypertrophy undergoing dialysis).
- β-Blockers might benefit patients with ischemic heart disease, CHF, migraine, and patients with history of ST-segment elevation myocardial infarction (STEMI).
- ACE inhibitors should be used in patients with diabetes, proteinuria, atrial fibrillation, or heart failure with reduced ejection fraction (HFrEF) but not in pregnancy.
- α-Adrenergic blockers are not the first choice for monotherapy but remain as second line after combination therapy of first-line agents; might benefit males with benign prostatic hypertrophy (BPH)
- CCB could be considered in patients with isolated systolic HTN, atherosclerosis, angina, migraine, or asthma; well documented to reduce risk of stroke

FIRST LINE

1) **Thiazide diuretics** may not be effective with Cr clearance <30.
   a. **Chlorthalidone**: 12.5 to 25.0 mg/day (more potent than hydrochlorothiazide but causes more hyponatremia and hypokalemia)
   b. **Hydrochlorothiazide**: 12.5 to 50.0 mg/day
   c. **Indapamide**: 1.25 to 2.50 mg/day
2) **ACE inhibitors**
   a) **Lisinopril**: 10 to 40 mg/day
   b) **Enalapril**: 5 to 40 mg/day
   c) **Ramipril**: 2.5 to 20.0 mg/day
   d) **Benazepril**: 10 to 40 mg/day
3) **CCB**
   a) **Diltiazem** CD: 180 to 360 mg/day
   b) **Nifedipine** (sustained release): 30 to 90 mg/day
   c) **Verapamil** (sustained release): 120 to 480 mg/day
   d) **Amlodipine**: 2.5 to 10.0 mg/day
4) **ARBs**
   a) **Losartan**: 25 to 100 mg in 1 or 2 doses; has unique but modest uricosuric effect
   b) **Valsartan**: 80 to 320 mg daily
   c) **Irbesartan**: 75 to 300 mg daily
   d) **Candesartan**: 4 to 32 mg daily
   e) **Renin inhibitor**: aliskiren 150 to 300 mg daily

CONTRAINDICATIONS

- ❑ Diuretics may worsen gout.
- ❑ β-Blockers (relative) in reactive airway disease, heart block, diabetes, and peripheral vascular disease; probably

should be avoided in patients with metabolic syndrome or insulin-requiring diabetes

- ❑ Diltiazem or verapamil: Do not use with systolic dysfunction or heart block. Amlodipine may cause peripheral edema.

SECOND LINE

- ❖ Before escalating therapy, ensure that patient is adherent to prescribed regimen.
- ❖ Many may be combined. Choose additional medications with complementary effects (i.e., ACE inhibitors/ARBs with diuretic or a vasodilator with a diuretic or β-blocker). Don't combine ACE-I and ARB.
- ❖ Medication-refractory HTN: **spironolactone**: 25 to 100 mg/day especially effective
- ❖ Centrally acting α-2 agonists: **clonidine** 0.1 to 1.2 mg BID or weekly patch 0.1 to 0.3 mg/day, guanfacine 1 to 3 mg daily, or **methyldopa** 250 to 2,000 mg BID
- ❖ α-Adrenergic antagonists: **prazosin** 1 to 10 mg BID, **terazosin** 1 to 20 mg/day, or **doxazosin** 1 to 16 mg/day
- ❖ Vasodilators:
  - **Hydralazine**: 10 to 25 mg QID; risk of tachycardia, so generally combined with β-blocker; also drug-induced systemic lupus erythematosus (SLE)
  - **Minoxidil**: rarely used due to adverse effects; may be more effective than other medications in renal failure and refractory HTN
- ❖ **Metolazone** and **loop diuretics** may be used with more severe renal impairment, but outcomes data are absent; loop diuretics (for volume overload): **furosemide** 20 to 320 mg/day or bumetanide 0.5 to 2.0 mg/day
- ❖ K+-sparing diuretics in patients with hypokalemia while taking thiazides: **amiloride** 5 to 10 mg/day or **triamterene** 50 to 150 mg/day

COMPLEMENTARY & ALTERNATIVE MEDICINE

- Biofeedback and relaxation exercise
- Dietary supplements such as garlic have been suggested for lowering BP, but evidence is lacking.

FOLLOW-UP RECOMMENDATIONS

PATIENT MONITORING

- ❖ Reevaluate patients 3–6mo until stable and then 6–12mo. Consider use of home self-BP monitoring; quality-of-life issues including sexual function should be considered.
- ❖ Poor medication adherence is a leading cause of apparent medication failure.
- ❖ At least annual creatinine, and potassium for patients on diuretics, ACE inhibitors, and ARBs

DIET

- ~20% of patients will respond to reduced-salt diet (<100 mmol/day; <6 g NaCl or <2.4 g Na).
- Limit alcohol consumption to <1 oz/day.

COMPLICATIONS

1) Heart failure
2) renal failure
3) LVH
4) myocardial infarction
5) retinal hemorrhage
6) stroke
7) hypertensive heart disease
8) drug side effects
9) erectile dysfunction

Compelling Indications	
Indication	**Treatment Choice**
Heart Failure	ACEI/ARB + BB + diuretic + spironolactone
Post –MI/Clinical CAD	ACEI/ARB AND BB
CAD	ACEI, BB, diuretic, CCB
Diabetes	ACEI/ARB, CCB, diuretic
CKD	ACEI/ARB
Recurrent stroke prevention	ACEI, diuretic
Pregnancy	labetolol (first line), nifedipine, methyldopa

Beta-1 Selective Beta-blockers – possibly safer in patients with COPD, asthma, diabetes, and peripheral vascular disease:
- metoprolol
- bisoprolol
- betaxolol
- acebutolol

Drug Class	Agents of Choice	Comments
Diuretics	HCTZ 12.5-50mg, chlorthalidone 12.5-25mg, indapamide 1.25-2.5mg triamterene 100mg *K+ sparing* – spironolactone 25-50mg, amiloride 5-10mg, triamterene 100mg furosemide 20-80mg twice daily, torsemide 10-40mg	Monitor for hypokalemia Most SE are metabolic in nature Most effective when combined w/ ACEI Stronger clinical evidence w/chlorthalidone Spironolactone - gynecomastia and hyperkalemia Loop diuretics may be needed when GFR <40mL/min
ACEI/ARB	*ACEI:* lisinopril, benazapril, fosinopril and quinapril 10-40mg, ramipril 5-10mg, trandolapril 2-8mg *ARB*: candesartan 8-32mg, valsartan 80-320mg, losartan 50-100mg, olmesartan 20-40mg, telmisartan 20-80mg	SE: Cough (ACEI only), angioedema (more with ACEI), hyperkalemia Losartan lowers uric acid levels; candesartan may prevent migraine headaches
Beta-Blockers	metoprolol succinate 50-100mg and tartrate 50-100mg twice daily, nebivolol 5-10mg, propranolol 40-120mg twice daily, carvedilol 6.25-25mg twice daily, bisoprolol 5-10mg, labetalol 100-300mg twice daily,	Not first line agents – reserve for post-MI/CHF Cause fatigue and decreased heart rate Adversely affect glucose; mask hypoglycemic awareness
Calcium channel blockers	*Dihydropyridines*: amlodipine 5-10mg, nifedipine ER 30-90mg, *Non-dihydropyridines*: diltiazem ER 180-360 mg, verapamil 80-120mg 3 times daily or ER 240-480mg	Cause edema; dihydropyridines may be safely combined w/ B-blocker Non-dihydropyridines reduce heart rate and proteinuria
Vasodilators	hydralazine 25-100mg twice daily, minoxidil 5-10mg terazosin 1-5mg, doxazosin 1-4mg given at bedtime	Hydralazine and minoxidil may cause reflex tachycardia and fluid retention – usually require diuretic + B-blocker Alpha-blockers may cause orthostatic hypotension
Centrally-acting Agents	clonidine 0.1-0.2mg twice daily, methyldopa 250-500mg twice daily guanfacine 1-3mg	Clonidine available in weekly patch formulation for resistant hypertension

CLINICAL PEARLS

- Treatment of HTN reduces risk of many serious medical conditions with numbers needed to treat to prevent one serious event (e.g., stroke or myocardial infarction) ranging from ~20 patients per year for severe HTN to more than several hundred per year for milder HTN.
- Multiple submaximal doses are likely to have fewer side effects and more effectiveness than fewer maximum-dosed drugs.
- Treatment decisions should always be informed by BP measured by proper technique; measures obtained outside the clinical office may be more predictive of CV risk.
- Appropriate lifestyle changes should be recommended before and during pharmacologic treatment.
- Overly-aggressive treatment may cause significant harms, including syncope and electrolyte abnormalities; such effects are more likely and often more significant in the elderly.
- Treatment goals should be periodically revisited and jointly established through shared decision making informed by anticipated potential benefits and harms.

2. HYPERTENSION,

SECONDARY AND RESISTANT

INTRODUCTION

- **Resistant HTN:** defined as blood pressure that remains above goal in spite of the concurrent use of three antihypertensive agents of different classes. Ideally, one of the three agents should be a diuretic, and all agents should be prescribed at optimal dose amounts .
- **Secondary HTN:** elevated BP that results from an identifiable underlying mechanism Both the Eighth Joint National Committee (JNC 8) and AHA/ACC/CDC guidelines recommend a goal BP of <140/90 mm Hg, although JNC 8 allows for a goal of <150/90 mm Hg for patients age >60 years.
- **Uncontrolled hypertension (HTN)** comprises the following entities (see "Alert" below)

GERIATRIC CONSIDERATIONS

- Onset of HTN in adults >60 years of age is a strong indicator of secondary HTN.
- Secondary causes more common in the elderly include sleep apnea, renal disease, renal artery stenosis, and primary aldosteronism (PA).
- In patients >80 years of age, consider a higher target systolic blood pressure (SBP) of ≥150 mm Hg. Be cautious to avoid excessive diastolic lowering.
- Elderly may be particularly responsive to diuretics and dihydropyridine calcium channel blockers.
- Systolic HTN is particularly problematic in the elderly.

ALERT

Pseudo-resistance

- Inaccurate measurement of BP
  - Cuff too small
  - Patient not at rest; sitting quietly for 5 minutes
- Poor adherence: In primary care settings, this has been estimated to occur in 40–60% of patients with HTN. See USPSTF recommendations."below"
- White coat effect: prevalence 20–40%. Do not make clinical decisions about HTN based solely on measurement in the clinic setting. Home BP monitoring and/or ambulatory BP monitoring is more reliable.
- Inadequate treatment

EPIDEMIOLOGY

- Predominant age: In general, HTN has its onset between ages 30 and 50 years.
- Patients with resistant HTN are more likely to experience the combined outcomes of death, myocardial infarction, congestive heart failure (CHF), stroke, or chronic kidney disease.
- Depending on etiology, age of onset can vary. Age of onset <20 or >50 years increases likelihood of a secondary cause for HTN.
- The strongest predictors for resistant HTN are age (>75 years), presence of left ventricular hypertrophy (LVH), obesity (body mass index [BMI] >30), and high baseline systolic BP.
- Other predictors include chronic kidney disease, diabetes, living in the southeastern United States, African American race (especially women), and excessive salt intake.

PREVALENCE

- Prevalence of resistant HTN is unknown.
- NHANES analysis indicates only 53% of adults are controlled to a BP of <140/90 mm Hg.

- Secondary HTN occurs in about 5–10% of adults with chronic HTN.

ETIOLOGY AND PATHOPHYSIOLOGY

1) **Obstructive sleep apnea (OSA):** One study diagnosed OSA in 83% of treatment-resistant hypertensives.
2) **Primary hyperaldosteronism** (17–22% of resistant HTN cases)
3) **Chronic renal disease**
4) **Renovascular disease** (0.2–0.7%, up to 35% of elderly, 20% of patients undergoing cardiac catheterization)
5) **Cushing syndrome**
6) **Pheochromocytoma**
7) Other rare causes: **hyperthyroidism**, **hyperparathyroidism**, **aortic coarctation**, **intracranial tumor**
8) **Drug-related causes:**

- Medications, especially NSAIDs (may also blunt effectiveness of ACE inhibitors), decongestants, stimulants (e.g., amphetamines, attention deficient hyperactivity disorder [ADHD] medications), anorectic agents, erythropoietin, natural licorice (in some chewing tobacco), yohimbine, glucocorticoids

- Oral contraceptives: unclear association; mainly epidemiologic and with higher estrogen pills

- Cocaine, amphetamines, other illicit drugs; drug and alcohol withdrawal syndromes

9) **Lifestyle factors:** Obesity and dietary salt may negate the beneficial effect of diuretics. Excessive alcohol may cause or exacerbate HTN. Physical inactivity also contributes.

RISK FACTORS

resistant HTN (16.2%) were more likely to be:

1) male
2) Caucasian
3) Older
4) diabetic.
5) They were also more likely to be taking β-blockers, calcium channel blockers, and α-adrenergic blockers compared with other drug classes.

Factors predictive of resistant or secondary HTN:

1) female sex
2) African American race
3) Obesity
4) diabetes
5) worsening of control in previously stable hypertensive patient
6) onset in patients age <20 years or >50 years
7) lack of family history of HTN
8) significant target end-organ damage
9) stage 2 HTN (systolic BP >160 mm Hg or diastolic BP >100 mm Hg)
10) renal disease
11) alcohol or drug use

GENERAL PREVENTION

The prevention of resistant and secondary HTN is thought to be the same as for primary or essential HTN:

- Adopting a Dietary Approaches to Stop Hypertension (DASH) diet
- a low-sodium diet
- weight loss in obese patients
- exercise,
- limitation of alcohol intake, and smoking cessation
- Relaxation techniques may be of help, but data are limited.

DIAGNOSIS

1.HISTORY

- Ask or review at every visit: "The Big Four" **SANS** mnemonic:
  (i) Salt intake
  (ii) Alcohol intake
  (iii) NSAID use
  (iv) Sleep
- Review home BP readings; consider ambulatory BP monitoring.
- History will vary with etiology of HTN.
- Pheochromocytoma: episodes of headache, palpitations, sweating
- **Cushing syndrome:** weight gain, fatigue, weakness, easy bruising, amenorrhea
- Obstructive sleep apnoea (OSA): loud snoring while asleep, daytime somnolence
- Increased intravascular volume: swelling

2.PHYSICAL EXAM

- Ensure that the BP is measured correctly.
- The patient should be sitting quietly with back supported for 5 minutes before measurement.
- Proper cuff size: bladder encircling at least 80% of the arm. Support arm at heart level.
- Minimum of two readings at least 1 minute apart.
- Check BP in both arms.
- Also check standing BP for orthostasis.

- The USPSTF recommends "obtaining measurements outside of the clinical setting for diagnostic confirmation."
- Attention to findings related to possible etiologies:
  1) **renovascular HTN:** systolic/diastolic abdominal bruit
  2) **pheochromocytoma:** diaphoresis, tachycardia
  3) **Cushing syndrome:** hirsutism, moon facies, dorsal hump, purple striae, truncal obesity
  4) **thyroid disease:** enlarged thyroid, tremor, exophthalmos, tachycardia
  5) **coarctation of the aorta:** upper limb HTN with decreased or delayed femoral pulses Funduscopic exam

3.diagnostic tests & interpretation

- ECG performed as part of the initial workup; LVH is an important marker of resistant HTN.
- Sleep study if history and physical indicate. The Epworth Sleepiness Scale is recommended.
- Home-based polysomnography has been shown to be accurate in screening for OSA. Overnight oximetry is not helpful.

4.INITIAL TESTS (LAB, IMAGING)

- Initial limited diagnostic testing should include:
  - urinalysis
  - CBC
  - potassium, sodium, calcium, glucose, creatinine
  - lipids
  - thyroid-stimulating hormone (TSH)
- 50% of patients with hyperaldosteronism may have normal potassium levels.
- Imaging tests listed are necessary only if history, physical, or lab data indicate:
  - Abdominal US: if renal disease is suspected
  - Duplex ultrasonography may be the preferred test for renovascular disease. MR angiography (MRA) of renal vasculature is sensitive but has low specificity and potentially more harmful. Conventional catheter angiography or CT angiography may be required to confirm the diagnosis.
  - Adrenal "incidentaloma" frequently arises in this era of multiple CT studies. If present in the setting of

resistant HTN, consider hyperaldosteronism or hyperadrenal corticoid states.

FOLLOW-UP TESTS & SPECIAL CONSIDERATIONS

- Further testing for PA may be considered.
- Empiric treatment with an aldosterone inhibitor may be preferable and more clinically relevant: **spironolactone** or **eplerenone**. **Amiloride** may be more effective in African Americans.
- Plasma aldosterone-to-renin ratio (ARR) is the preferred lab test, but the test is difficult to perform and interpret properly.
- Further testing for pheochromocytoma: plasma

Other tests to consider for resistant or secondary HTN:

- 24-hour urine for free cortisol, calcium, parathyroid hormone (PTH), overnight 1-mg dexamethasone suppression test, urine toxicology screen

DIAGNOSTIC PROCEDURES/OTHER

- ❑ Consider 24-hour ambulatory BP monitoring, especially if white coat effect is suspected. Home BP monitor results predict mortality, stroke, and other target organ damage better than office BP.
- ❑ Optimal protocol involves two paired measurements: morning and evening (four measurements) over 4 to 7 days.
- ❑ Oscillometric, electronic, upper arm, fully automatic device with memory: average multiple readings over several days

TREATMENT

- Treatment modality depends on etiology of HTN. Please see each etiology listed for information on proper treatment.
- Emphasize adherence to JNC 8 and/or AHA/ACC guidelines, with emphasis on lifestyle modification.
- Obese patients, African Americans, and elderly may be particularly responsive to diuretics.
- Tolerance to diuretics may occur: long-term adaptation to thiazides or the "braking effect." Consider increasing the dose of thiazide or adding an aldosterone inhibitor.
- **Treatment specific to certain secondary etiologies:**
  1) **PA:** aldosterone receptor antagonist: spironolactone or eplerenone
  2) **Cushing syndrome:** aldosterone receptor antagonist
  3) **OSA:** continuous positive airway pressure (CPAP) ± oxygen, surgery, weight loss
     Mandibular advancement devices may be equally effective in some patients.
  4) **Nocturnal hypoxia:** oxygen supplementation
  5) **Renal sympathetic denervation** has been disproven as an effective strategy.

MEDICATION

- ❑ Adding a medication to the regimen may have greater efficacy than increasing the dose of medications
- ❑ Aldosterone antagonists may offer significant benefit.
- ❑ Central-acting agents (e.g., clonidine) are effective at reducing BP, but outcome data are lacking.

ALERT

- Agents specific for treatment of HTN emergencies should be initiated under a situation in which immediate BP reduction will prevent or limit end-organ damage (see "Hypertensive Emergencies").
- Renovascular HTN: Angioplasty is the treatment of choice for fibromuscular dysplasia of a renal artery.
- The recent CORAL study concluded that in patients with atherosclerotic renovascular disease and HTN, renal artery stenting did not improve outcomes over medical therapy alone.

- Referral to an HTN specialist or clinic: Retrospective studies indicate improved control rates for patients with resistant HTN referred to special HTN clinics.

FIRST LINE

- For non-black patients: thiazide diuretics, ACEi or ARB (not both), CCB
- For black patients: thiazide diuretics, CCBs

SECOND LINE

Combine thiazide diuretic with ACEi, ARB, or CCB or add a K+-sparing diuretic

THIRD LINE

Add agent not used in second line; if this does not adequately lower BP, initiate work up for secondary causes (chronic NSAID use, alcohol abuse, RAS, etc.).

CLINICAL PEARLS

- Hospitalization may be necessary for hypertensive urgency or emergency general measures.
- Onset of HTN in adults >60 years of age is a strong indicator of secondary HTN.
- Common causes of resistant HTN: OSA, excessive salt intake, medication nonadherence, alcohol, NSAIDs
- Common secondary causes include sleep apnea, renal disease, renal artery stenosis, and PA.
- Aldosterone inhibitors should be considered in all cases of resistant HTN. Home BP monitoring predicts outcomes better than office monitoring of BP.

EMERGENCIES IN PRIMARY HEALTH CARE

DEFINITION OF EMERGENCY

- An event demanding immediate medical attention.
- An obvious yet important concept is that of 'time criticality', which implies that certain patients are at high risk of a critical outcome of deterioration if there is significant delay in appropriate management (ex.:acute coronary syndrome).
- The immediate approach to a specific emergency differs from normal, less urgent medical practice. The usual method of history and examination is replaced with a

technique of rapid assessment and immediate management.

PRINCIPLES OF MANAGEMENT

The important principles of management of the emergency call can be

summarized as follows:

1. The practitioner must be aware of life-threatening conditions.
2. The practitioner should be prepared mentally and physically.
3. PLAN, EQUIP and PRACTICE.
4. Chest pain/collapse/myocardial infarction (collectively) represents the premium emergency call.
5. Beware of children with respiratory distress and traumatic injuries.
6. The most savable patients are those with blood loss. Hence IV fluids for intravascular volume expansion are essential.
7. The necessary basic skills to cope with most emergencies involve DRSABCD—danger, response, send for help, airway, breathing, circulation, defibrillator.
8. Have the equipment and the skills to handle body fluids potentially contaminated with blood-borne viruses.
9. 70% of cardiac arrests occur in the home, so the availability of a portable defibrillator is important.

VITAL BASIC SKILLS

1) Rapid intravenous access
2) Cardiopulmonary resuscitation, including upper airway relief, intubation and ventilation, treatment of cardiac arrhythmias and defibrillation
3) Cricothyroidotomy
4) Arrest of hemorrhage
5) Knowledge of usage of common emergency drugs

ALARMING SYMPTOMS AND SIGNS

1. Unconsciousness
2. Convulsions
3. Chest pain in an adult, especially associated with pallor and sweating
4. Pallor and sweating in any patient with pain, collapse or injury
5. Collapse, especially at toilet
6. Significant hemorrhage
7. Breathlessness, including asthma
8. The agitated patient threatening homicide or suicide (take a police officer for company)

DON'T FORGET THE VALUE OF OXYGEN

- Ideally, the doctor who attends emergency calls should carry an oxygen delivery unit or at least rely on the simultaneous arrival of an ambulance with resuscitation equipment.
- Remember, oxygen is a treatment for hypoxia, not for breathlessness;
- a pulse oximeter is very useful.
- Most cases require a high flow rate of 8–10 L/minute.

MEDICAL EMERGENCIES TYPICALLY REQUIRING HIGH-FLOW OXYGEN:

1. Acute pulmonary oedema
2. Acute anaphylaxis
3. Cardiopulmonary arrest
4. Collapse
5. Status epilepticus
6. Shock, Sepsis

7. Major haemorrhage, Major trauma

MEDICAL EMERGENCIES WHERE OXYGEN SHOULD BE USED WITH CAUTION, UNLESS OXIMETRY CONFIRMS HYPOXIA:

1. Underlying COPD.
2. Myocardial infarction—high-flow oxygen may increase infarct size.
3. Stroke.
4. Obstetric emergencies.

TWELVE GOLDEN RULES

for the diagnostic approach to the emergency call:

1. Always consider the possibilities of hypoglycaemia **and opioid overdosage** in the unconscious patient.
2. Consider **intra-abdominal bleeding first** and foremost in a patient with abdominal pain who collapses at the toilet.
3. Acute chest pain represents **myocardial infarction** until proven otherwise.
4. Exclude meningitis **and septicaemia** in a child with a rapid onset of Drowsiness and pallor.
5. Consider a ruptured **intra-abdominal viscus** in any person, especially a child, with persistent post-traumatic abdominal pain.
6. Consider **acute anaphylaxis** in patients with a past history of allergies.
7. Consider **substance use and organic causes** for patients with acute psychosis or bizarre behaviour.
8. Consider **ectopic pregnancy** in any woman of child-bearing age presenting with acute abdominal pain
9. If a patient is found cyanosed, always consider **upper-airway obstruction** first.
10. Beware of the **asthmatic** who is cyanosed with a 'silent chest' and tachycardia.
11. Consider **ventricular fibrillation or other arrhythmia** in an adult with sudden collapse or dizziness.
12. Consider **subarachnoid haemorrhage** in anyone with a sudden onset of headache.

ACUTE ANAPHYLAXIS AND ANAPHYLACTIC REACTIONS

- Anaphylaxis is a severe allergic reaction that is rapid in onset and may cause death.
- Common causes: stings, bites (e.g. imported red fire ants, jack jumper ants), parenteral antibiotics (especially penicillin), food reactions (e.g. peanuts, fish).
- Other causes: blood products, antivenom, radiological contact materials, anaesthetic agents, NSAIDs
- The onset of anaphylaxis from exposure is usually rapid—typically 10–20 minutes. The early administration of adrenaline saves lives. The diagnosis is basically a clinical one.

SYMPTOMS

1) Skin: pruritus—generalised, palate, hands, feet, urticaria, angio-oedema.
2) Respiratory: wheeze, stridor.
3) Nausea and vomiting, abdominal pain, diarrhoea
4) Hypotension: syncope, collapse.
5) Palpitations

- Note: The early danger symptom is itching of the palms of hands and soles Of the feet.

FIRST-LINE TREATMENT (ADULTS)

1) Call for **help**.
2) Remove **cause** (e.g. bee sting) if possible. Lay the patient down.
3) **Oxygen** 8 L/min (by face mask).

4) **Adrenaline** 0.5 mg IM best given in mid antero-lateral thigh

5) Set up IV access.

6) If no rapid improvement:

   ✓ repeat IM adrenaline every 3–5 minutes.

   ✓ set up adrenaline infusion: *1 mg* adrenaline to *1000 mL* N saline (best with ECG monitor).

7) Set up additional IV line (preferably two 'wide bore' lines) and infuse crystalloid solution (e.g. N saline 1-2 L) with 20 mL/kg) over 1–2 min.

8) Salbutamol aerosol inhalation (or nebulisation if severe), especially if wheeze/stridor.

9) Consider promethazine (25 mg) IV and hydrocortisone (250 mg) IV.

10) Admit to hospital (observe at least 4 hours)

11) Discharge on promethazine 25 mg tds + prednisolone 50 mg/day for 3 days.

12) Provide an adrenaline autoinjector.

ACUTE CARDIOGENIC PULMONARY OEDEMA

❖ Keep in mind underlying cause e.g.:

➢ Myocardial infarction

➢ Arrhythmia

➢ Cardiomyopathy

➢ Anaemia

KEEP IN MIND THE MNEMONIC LMNOP (LASIX, MORPHINE, NITRATES, O2, CPAP).

1. Keep the patient propped up in bed with legs over side.
2. Oxygen by mask or intranasally (8 L/min).
3. Glyceryl trinitrate (nitroglycerin) 300–600 mcg sublingual (tablet or spray); can use IV nitrates (if BP >100 mmHg).
4. Insert IV line (large bore cannula).
5. Furosemide 40 mg IV , increasing to 80 mg IV as necessary (or twice the patient' s normal oral dose).
6. Morphine 1–2.5 mg IV—but caution: reserve for chest pain and anxiety.
7. CPAP (continuous positive airway pressure)—for unresponsive cases or BiPAP (if available)—in ambulance, ED or CCU.
8. Give amiodarone 3–5 mg/kg loading dose IV infusion if rapid atrial fibrillation.

SEVERE HYPOGLYCEMIA

❖ Confirm by blood sugar levels (under 70 mg/dL)

O 50% dextrose 20 mL IV through securely positioned cannula, large vein (if IV line difficult, administer rectally by pressing the nozzle of a large syringe into the anus and injecting slowly)
OR
O Glucagon 1 mL IM or SC (most practical option) then oral glucose if able to swallow—can repeat glucagon

MYOCARDIAL INFARCTION / UNSTABLE ANGINA

FIRST-LINE MANAGEMENT

1) Insert IV line.

2) Glyceryl trinitrate (nitroglycerin) 300 mcg (½ tab) SL or spray, unless BP <90 mmHg or pulse <50/min.

3) Aspirin 300 mg (½ or 1 tab).

4) Morphine 1 mg per minute IV until pain relief (up to 15 mg)—usually 2.5–10 mg (lower dose if elderly/frail). ECG (set up by an assistant).

5) CPAP or BiPAP (if available).

6) Arrange ambulance and hospitalisation.

HYPERVENTILATION

Calm the patient with a firm but reassuring voice. Remove them from anxiety-provoking stimuli such as panicking bystanders Consider the possibility of an alternative diagnosis causing hypoxia.

STATUS EPILEPTICUS AND SERIAL SEIZURES

- ❖ Status epilepticus = repeated convulsions (usually >5 minutes) without regaining consciousness after initial tonic–clonic seizure. Significant mortality at >30 minutes.
- ❖ Serial seizures = repeated convulsions after regaining consciousness.

MANAGEMENT

- Lie patient on side.
- Ensure adequate oxygenation: attend to airway (e.g. Guedel tube); give oxygen 8 L/min (check blood sugar).
- ❖ If persisting >5 minutes: (summarised) midazolam 5–10 mg IM or IV over 2–5 minutes (lorazepam, if available, has stronger evidence).

OR

- ❖ midazolam 5–10 mg buccally (tear top off plastic ampoule,5 slowly drip into patient's mouth between gums and cheek—with syringe or from ampoule)

OR

- ❖ midazolam 5–10 mg intranasally—1–3 drops at a time into alternate nostrils repeat once in 15 minutes (if necessary)

OR

- ❖ **Diazepam 0.05 mg/kg/minute IV** until the seizures cease or respiratory depression begins (beware of respiratory depression and other vital parameters): usually 10–20 mg bolus in adult
- ❖ When seizures cease, follow immediately with: phenytoin 15–20 mg/kg IV over 30 minutes

OR

- ❖ **Sodium valproate** 10 mg/kg by slow IV infusion.

➢ Other drugs to consider:
Phenobarbitone, Thiopentone.

DIABETIC KETOACIDOSIS

- ❖ It is an acute, major, life-threatening complication of diabetes characterized by **hyperglycemia**, **ketoacidosis** and **ketonuria**.
- ❖ It is typically characterized by:

1. hyperglycemia > 250 mg/dL
2. bicarbonate level < 18 mEq/L
3. pH<7.3 with ketonemia or ketonuria.

- ❖ DKA occurs mainly in patients with type 1 diabetes, but it is not uncommon in some patients with type 2 diabetes.

LABORATORY STUDIES:

- ❖ Serum glucose levels
- ❖ Serum electrolytes (potassium, sodium, chloride, magnesium, calcium, phosphorus)
- ❖ Blood urea nitrogen (BUN) and creatinine levels
- ❖ Bicarbonate levels
- ❖ Arterial blood gas (ABG) measurements
- ❖ Amylase and lipase levels
- ❖ Urine dipstick
- ❖ Ketone levels
- ❖ Serum or capillary beta-hydroxybutyrate levels

- ❖ CBC
- ❖ Urine and blood cultures if intercurrent infection is suspected
- ❖ ECG (or telemetry in patients with comorbidity)

MANAGEMENT (DURING THE FIRST 24-48 HOURS):

1) Fluid resuscitation (IV fluids: isotinic sodium chloride solution or Ringer lactate)

-administer 1-3 L during the **first hour**

-administer 1 L during the **second hour**

-administer 1 L during the **following 2 hours**

-administer 1 L **every 4 hours** depending on the degree of dehydration and CVP readings

2) Reversal of the acidosis and ketosis

- **sodium bicarbonate 100-150 mL** of 1.4% concentration is infused initially, this may be repeated **every half an hour** if necessary. Rapid and early correction may worsen hypokalemia and cause paradoxical cellular acidosis.

3) insulin Reduction in the plasma glucose concentration to normal

Initial insulin dose is a continuous IV infusion using an infusion pump at a rate of 0.1 U/Kg/h.

A mix of 24 units of regular insulin in 60 mL of isotonic sodium chloride solution usually is infusd at a rate of 15 mL/h (6U/h) **until blood glucose level drops to less than 180 mg/dL** then the rate of infusion is decreased to 5-7.5 mL/h (2-3U/h) until the ketoacidotic state abates. Blood glucose level should not fall lower than 200mg/dL during the first 4-5 hours.

- Replenishment of electrolyte

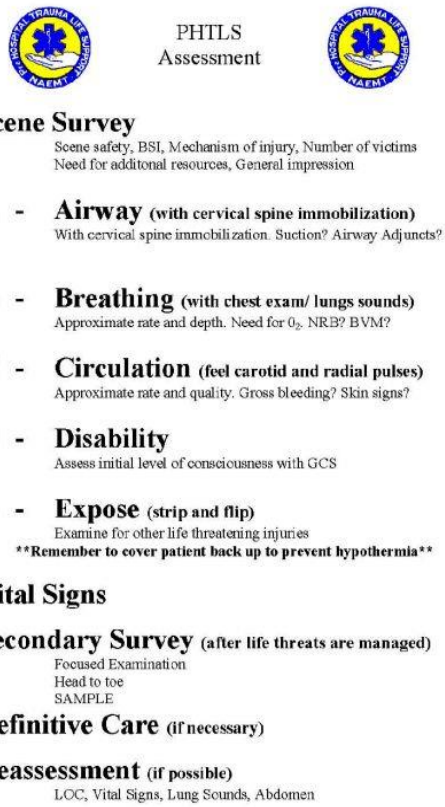
-if K+ > 6 mEq/L do not administer potassium supplement

-if K+ 4.5-6 mEq/L administer 10 mEq/h of potassium chloride

If K+ < 3-4.5 mEq/L administer 20 mEq/h of potassium chloride

- Identification of the underlying cause and any complication.

EMERGENCY FIRST AID



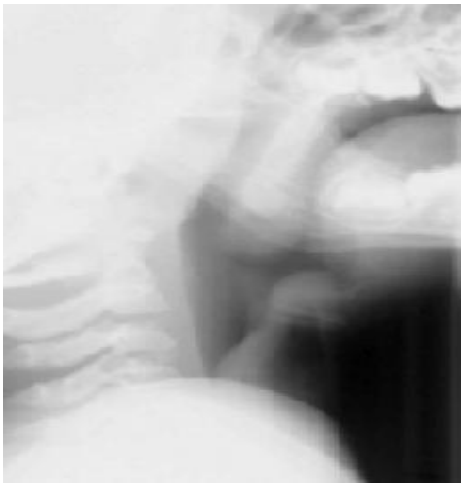
UPPER RESPIRATORY TRACT INFECTIONS

INTRODUCTION

- ❖ upper respiratory tract infection (URTI) is an illness caused by an acute infection, which involves the upper respiratory tract, including the nose, sinuses, pharynx, or larynx. This commonly includes nasal obstruction, sore throat, tonsillitis, pharyngitis, laryngitis, sinusitis, otitis media, and the common cold.
- ❖ Upper respiratory tract infection (URI) represents the most common acute illness evaluated in the outpatient setting. URIs range from the common cold—typically a mild, self- limited—to life-threatening illnesses such as

epiglottitis

- The upper respiratory tract includes the sinuses, nasal passages, pharynx, and larynx, which serve as gateways to the trachea, bronchi, and pulmonary alveolar spaces. Rhinitis, pharyngitis, sinusitis, epiglottitis, laryngitis, and tracheitis are specific manifestations of URIs.



Lateral neck radiograph demonstrates

COMMON URI TERMS ARE DEFINED AS FOLLOWS:

- **Rhinitis**: Inflammation of the nasal mucosa
- **Rhinosinusitis or sinusitis**: Inflammation of the nares and paranasal sinuses, including frontal, ethmoid, maxillary, and sphenoid
- **Nasopharyngitis** (rhinopharyngitis or the common cold): Inflammation of the nares, pharynx, hypopharynx, uvula, and tonsils
- **Pharyngitis:** Inflammation of the pharynx, hypopharynx, uvula, and tonsils
- **Epiglottitis (supraglottitis):** Inflammation of the superior portion of the larynx and supraglottic area
- **Laryngitis**: Inflammation of the larynx
- **Laryngotracheitis**: Inflammation of the larynx, trachea, and subglottic area
- **Tracheitis:** Inflammation of the trachea and subglottic area

ETIOLOGY

Most URIs are viral in origin. Typical viral agents that cause URIs include the following:

- Rhinoviruses
- Coronaviruses
- Adenoviruses
- Coxsackieviruses

SEASONALITY

- Although URIs may occur year round, in the United States most colds occur during fall and winter. Beginning in late August or early September, rates of colds increase over several weeks and remain elevated until March or April. Epidemics and mini-epidemics are most common during cold months, with a peak incidence from late winter to early spring.

- Cold weather results in more time spent indoors (eg, at work, home, school) and close exposure to others who may be infected. Humidity may also affect the prevalence of colds, because most viral URI agents thrive in the low humidity that is characteristic of winter months. Low indoor air moisture may increase friability of the nasal mucosa, increasing a person's susceptibility to infection.

HISTORY

- Itchy, watery eyes
- Nasal discharge
- Nasal congestion
- Sneezing
- Sore throat
- Cough
- Headache
- Fever
- Duration
- Malaise, Myalgias
- Fatigue, weakness

EXAMINATION

Patients with the common cold may have a paucity of clinical findings despite notable subjective discomfort. Findings may include the following:

- Nasal mucosal erythema and edema are common

- Nasal discharge: Profuse discharge is more characteristic of viral infections than bacterial infections; initially clear secretions typically become cloudy white, yellow, or green over several days, even in viral infections

- Foul breath

- Fever: Less common in adults but may be present in children with rhinoviral infections
- Erythema, swelling, or exudates of the tonsils or pharynx
- Temperature of 38.3°C (100.9°F) or higher
- Tender anterior cervical nodes (≥1 cm)
- Absence of conjunctivitis, cough, and rhinorrhea, which are symptoms that may suggest viral illness

DIAGNOSIS

- Group A streptococcal infection: Clinical findings or a history of exposure to a case, supported by results of rapid-detection assays and cultures (positive rapid antigen detection tests do not necessitate a backup culture)
- Acute bacterial rhinosinusitis: Laboratory studies are generally not indicated; Computed tomography (CT) scanning or other sinus imaging may be appropriate if symptoms persist despite therapy or if complications (eg, extension of disease into surrounding tissue) are suspected
- Influenza: Rapid tests have over 70% sensitivity and more than 90% specificity
- Mononucleosis: Heterophile antibody testing (eg, Monospot)
- Herpes simplex virus infection: Cell culture or polymerase chain reaction (PCR) assay
- Pertussis: Rapid tests; culture of a nasopharyngeal aspirate (criterion standard)
- Epiglottitis: Direct visualization by laryngoscopy, performed by an otorhinolaryngologist
- Gonococcal pharyngitis: Throat culture for*Neisseria gonorrhoeae*

RISK FACTORS FOR URIS

- Contact
- Inflammation
- Travel
- Smoking and exposure to second-hand smoke
- Immunocompromise that affects cellular or humoral immunity
- Anatomic changes due to facial dysmorphisms, previous upper airway trauma, and nasal polyposis
- Carrier state: Although some people are chronic carriers of group A streptococci, repeated URIs in such patients may be viral in origin

MEDICATIONS

- Therapy addressing specific symptoms is the mainstay for most upper respiratory infections (URIs). Most URIs are self-limited viral infections that resolve without prescription drugs.
- Recognizing viral and bacterial diseases for which specific therapy is available is important. Awareness of local trends in prevalent organisms and local resistance patterns is key. Antibacterial therapy is appropriate for patients with any of the following:
- Group A streptococcal pharyngitis
- Bacterial sinusitis
- Epiglottitis
- Pertussis
- Diphtheria

ANTIBIOTICS USED IN GROUP A STREPTOCOCCAL INFECTION ARE AS FOLLOWS:

- Penicillin VK (Penicillin V)
- Amoxicillin (Amoxil, Moxatag, Trimox)
- Penicillin G benzathine (Bicillin LA, Permapen)
- Cefadroxil (Duricef)
- Erythromycin (E.E.S., Erythrocin, E-Mycin, Eryc)
- Amoxicillin and clavulanate (Augmentin, Augmentin XR)
- Cefaclor (Ceclor)
- Cefuroxime (Ceftin)
- Ceftriaxone (Rocephin)
- Azithromycin (Zithromax)

ANTIBIOTICS USED IN EPIGLOTTITIS ARE AS FOLLOWS:

- Cefuroxime (Ceftin)
- Ceftriaxone (Rocephin)
- Cefotaxime (Claforan)
- Antibiotics used in pertussis are as follows:
- Clarithromycin (Biaxin)
- Erythromycin (E-Mycin, Erythrocin, Eryc, Ery-Tab, E.E.S.)
- Azithromycin (Zithromax)

ANTIBIOTICS USED IN ACUTE BACTERIAL RHINOSINUSITIS ARE AS FOLLOWS:

- Amoxicillin/clavulanate
- Doxycycline

PHARYNGITIS

This is most often viral in origin. Recognition of group A streptococcal pharyngitis is vital because serious complications may follow untreated disease.

VIRAL CAUSES OF PHARYNGITIS INCLUDE THE FOLLOWING:

- Adenovirus: May also cause laryngitis and conjunctivitis
- Influenza viruses
- Coxsackievirus
- Herpes simplex virus (HSV)
- EBV (infectious mononucleosis)
- Cytomegalovirus (CMV)

BACTERIAL CAUSES OF PHARYNGITIS INCLUDE THE FOLLOWING:

- Group A streptococci (approximately 5-15% of all cases of pharyngitis in adults; 20-30% in children)
- Group C and G streptococci
- Neisseria gonorrhoeae
- Arcanobacterium (Corynebacterium) hemolyticum
- Corynebacterium diphtheriae
- Atypical bacteria (eg, Mycoplasma pneumoniae and Chlamydia pneumoniae)
- Anaerobic bacteria

➢ Viral pharyngitis typically resolves in 1-2 weeks, but immunocompromised persons may have a more severe course.

➢ Untreated group A streptococcal pharyngitis can result in the following:

1) Acute rheumatic fever
2) Acute glomerulonephritis
3) Peritonsillar abscess
4) Toxic shock syndrome
5) Impetigo
6) Cellulitis or abscess
7) Otitis
8) Sinusitis
9) Conjunctivitis
10) Bronchitis

➢ Mortality from group A streptococcal pharyngitis is rare, but serious morbidity or death may result from one of its complications.

➢ Streptococcal pharyngitis without complications rarely poses significant risk for morbidity. The risk of mortality is significant in patients who progress to streptococcal toxic shock syndrome, which is characterized by multiorgan failure and hypotension.

➢ In patients with penicillin-sensitive streptococcal pharyngitis, symptomatic improvement is expected within 24-72 hours if antibiotic treatment is started in the first 24 hours after onset. Treatment failures are common and are mainly attributed to poor adherence, antibiotic resistance, and untreated close contacts, usually within the household or family.

➢ A chronic carrier state may develop with group A streptococcal infection. Eradicating the pathogen is difficult in these cases; however, carriers without active symptoms are unlikely to spread group A streptococci, and they are at low risk for developing rheumatic fever.

➢ Mononucleosis:

With infectious mononucleosis from EBV, complete resolution of symptoms may take up to 2 months. Acute symptoms rarely last more than 4 months. EBV typically remains dormant throughout the patient's life. Reactivation of the virus is not usually symptomatic.

HISTORY

The history alone is rarely a reliable differentiator between viral and bacterial pharyngitis. However, persistence of

symptoms beyond 10 days or progressive worsening after the first 5-7 days suggests a bacterial illness. Assessment for group A streptococci warrants special attention.

The health status of contacts and local epidemiologic trends are important factors to consider. A personal history of rheumatic fever (especially carditis or valvular disease) or a household contact with a history of rheumatic fever increases a person's risk. Other factors include occurrence from November through May and patient age of 5-15 years.

Pharyngeal symptoms of sore or scratchy throat, odynophagia, or dysphagia are common. If the uvula or posterior pharynx is inflamed, the patient may have an uncomfortable feeling of a lump when swallowing. Nasal obstruction may cause mouth breathing, which may result in dry mouth, especially in the morning. Group A streptococcal infections often produce a sudden sore throat.

Fever increases the suspicion that infection with group A streptococci is present, as does the absence of cough, rhinorrhea, and conjunctivitis, because these are common in viral syndrome; however, symptoms overlap between streptococcal and viral illness.

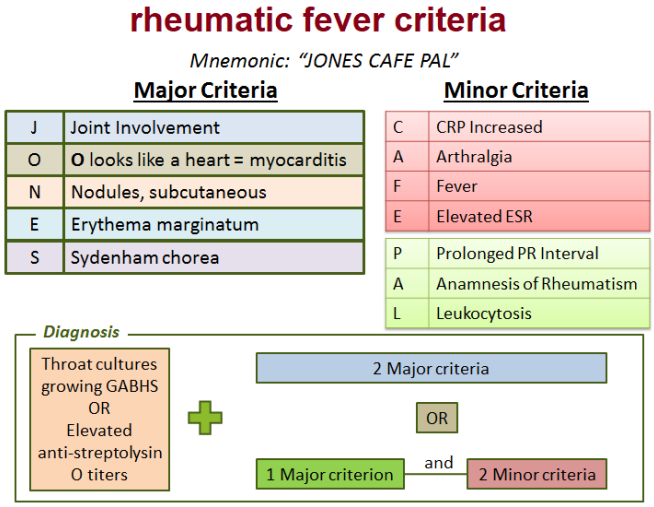
OTHER MANIFESTATIONS ARE AS FOLLOWS:

- **Secretions:** May be thick or yellow; however, these features do not differentiate a bacterial infection from a viral one
- **Cough:** May be due to laryngeal involvement or upper airway cough syndrome related to nasal secretions (postnasal drip)
- **Foul breath**: May occur because resident flora processes the products of the inflammatory process; foul breath may also occur with allergic rhinitis and viral infections
- **Headache:** While common with group A streptococci and mycoplasma infections, it also may reflect URI from other causes
- **Fatigue or malaise**: These may occur with any URI; extreme exhaustion is typical of influenza
- **Fever**: While usually slight or absent in adults, temperatures may reach 102°F in infants and young children
- **Rash**: A rash may be seen with group A streptococcal infections, particularly in children and in adolescents younger than 18 years
- **Abdominal pain**: This symptom may occur in streptococcal disease, most commonly in young children, but also in influenza and other viral conditions

- A history of recent orogenital contact suggests possible gonococcal rather than streptococcal pharyngitis. However, most gonococcal infections of the pharynx are asymptomatic.

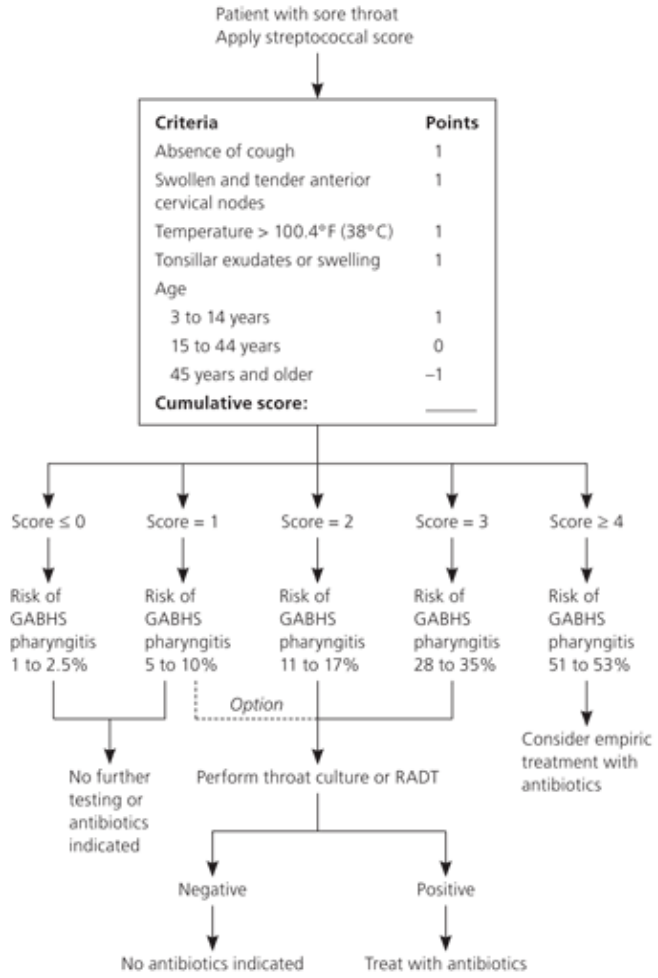
CLINICAL DECISION RULE FOR MANAGEMENT OF SORE THROAT

- The original Centor score uses four signs and symptoms to estimate the probability of acute streptococcal pharyngitis in adults with a sore throat. The score was later modified by adding.
- The cumulative score determines the likelihood of streptococcal pharyngitis and the need for antibiotics (Figure 1).
- Patients with a score of zero or 1 are at very low risk for streptococcal pharyngitis and do not require testing (i.e., throat culture or rapid antigen detection testing [RADT]) or antibiotic therapy.



- Patients with a score of 2 or 3 should be tested using RADT or throat culture; positive results warrant antibiotic therapy.

- Patients with a score of 4 or higher are at high risk of streptococcal pharyngitis, and empiric treatment may be considered



ACUTE BACTERIAL RHINOSINUSITIS IN CHILDREN

- Persistent nasal discharge (any type) or cough lasting 10 days or more without improvement
- Worsening course (new or worse nasal discharge, cough, fever) after initial improvement
- Severe onset (fever with nasal discharge) for at least 3 consecutive days

In older children and adults, symptoms (eg, pain, pressure) tend to localize to the affected sinus.

EPIGLOTTITIS

This condition is more often found in children aged 1-5 years, who present with a sudden onset of the following symptoms:

- Sore throat
- difficulty or pain during swallowing, globus sensation of a lump in the throat
- Muffled dysphonia or loss of voice
- Dry cough or no cough, dyspnea
- Fever, fatigue or malaise (may be seen with any URI)

Laryngotracheitis and laryngotracheobronchitis

- Nasopharyngitis often precedes laryngitis and tracheitis by several days
- Swallowing may be difficult or painful
- Patients may experience a globus sensation of a lump in the throat
- Hoarseness or loss of voice is a key manifestation of laryngeal involvement

DIABETES MELLITUS

DM: is an endocrine disorder characterized by hyperglycemia resulting from variable degrees of insulin resistance and deficiency. It is a leading cause of morbidity and mortality.

- prevalence = 17.1% in Jordan.

- 50% of T2DM are not diagnosed

DIABETES CAN BE CLASSIFIED INTO THE FOLLOWING GENERAL CATEGORIES:

1) Type 1 diabetes (due to auto-immune β-cell destruction, usually leading to absolute insulin deficiency)

2) Type 2 diabetes (85% of all diabetics) (due to a progressive loss of β-cell insulin secretion frequently on the background of insulin resistance)

3) Gestational diabetes mellitus (GDM) (diabetes diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation)

4) Specific types of diabetes due to other causes, e.g., monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young), diseases of the exocrine pancreas (such as cystic fibrosis and pancreatitis),

TABLE 1. Criteria for Testing for Diabetes or Prediabetes in Asymptomatic Adults

1.	Testing should be considered in overweight or obese (BMI ≥25 kg/m^2 or ≥23 kg/m^2 in Asian Americans) adults who have one or more of the following risk factors: • First-degree relative with diabetes • High-risk race/ethnicity (e.g., African American, Latino, Native American, Asian American, Pacific Islander) • History of CVD • Hypertension (≥140/90 mmHg or on therapy for hypertension) • HDL cholesterol level <35 mg/dL (0.90 mmol/L) and/or a triglyceride level >250 mg/dL (2.82 mmol/L) • Women with polycystic ovary syndrome • Physical inactivity • Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans)
2.	Patients with prediabetes (A1C ≥5.7% [39 mmol/mol], IGT, or IFG) should be tested yearly.
3.	Women who were diagnosed with GDM should have lifelong testing at least every 3 years.
4.	For all other patients, testing should begin at age 45 years.
5.	If results are normal, testing should be repeated at a minimum of 3-year intervals, with consideration of more frequent testing depending on initial results and risk status.

IFG, impaired fasting glucose; IGT, impaired glucose tolerance.

and drug- or chemical-induced diabetes (such as with glucocorticoid use, in the treatment of HIV/AIDS, or after organ transplantation)

IN EQUIVOCAL SETTING (DM 1 OR 2?):

a) C-peptide or insulin level (low in type 1 DM).

b) Glutamic acid decarboxylase antibodies.

C) Pancreatic islet cell antibodies (positive in 90% of new onset type 1 DM

SCREENING

Table 1 & Table 2

TABLE 2. Risk-Based Screening for Type 2 Diabetes or Prediabetes in Asymptomatic Children and Adolescents in a Clinical Setting

Testing should be considered in youth* who are overweight (≥85% percentile) or obese (≥95 percentile) **A** and who have one or more additional risk factors based on the strength of their association with diabetes:

- Maternal history of diabetes or GDM during the child's gestation **A**
- Family history of type 2 diabetes in first- or second-degree relative **A**
- Race/ethnicity (Native American, African American, Latino, Asian American, Pacific Islander) **A**
- Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome, or small-for-gestational-age birth weight) **B**

**After the onset of puberty or after 10 years of age, whichever occurs earlier. If tests are normal, repeat testing at a minimum of 3-year intervals, or more frequently if BMI is increasing, is recommended.*

DIAGNOSIS

Table 3

TABLE 3. Criteria for the Screening and Diagnosis of Diabetes

	Prediabetes	Diabetes
A1C	5.7–6.4%*	≥6.5%†
FPG	100–125 mg/dL (5.6–6.9 mmol/L)*	≥126 mg/dL (7.0 mmol/L)†
OGTT	140–199 mg/dL (7.8–11.0 mmol/L)*	≥200 mg/dL (11.1 mmol/L)†
RPG		≥200 mg/dL (11.1 mmol/L)‡

**For all three tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at the higher end of the range. †In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate samples. ‡Only diagnostic in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis. RPG, random plasma glucose.*

—Criteria for the diagnosis of diabetes

FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.*

OR

2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.*

OR

A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.*

OR

In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

*In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples.

PREVENTION OR DELAY OF TYPE 2 DIABETES

- Both IFG and IGT are risk factors for future diabetes and for cardiovascular disease and associated with insulin resistance and metabolic syndrome.

-Unless lifestyle modifications are made most people with pre-diabetes develop type 2 diabetes within 10 years.

-At least annual monitoring for the development of type 2 diabetes in those with prediabetes is suggested.

A. Life style modification:

-intensive behavioral lifestyle intervention to achieve and maintain 7% loss of initial body weight

-increase moderate-intensity physical activity (such as brisk walking) to at least 150 min/week. -improve eating patterns; Mediterranean eating plan, low-calorie and low-fat eating plan. emphasis on whole grains, legumes, nuts, fruits, and vegetables and minimal refined and processed foods.Higher intakes of nuts, berries, yogurt, coffee, and tea are associated with reduced diabetes risk. Red meats and sugar-sweetened beverages are associated with an increased risk of type 2 diabetes.(observational studies)

-limit alcohol intake; for women, no more than one drink per day, and two for men.

B. Pharmacological interventions:

-Metformin therapy for prevention of type 2 diabetes should be considered in those with prediabetes, especially for those with BMI ≥35 kg/m2, those aged <60 years, and women with prior GDM.

C. Prevention of Cardiovascular Disease:

-Prediabetes is associated with heightened cardiovascular risk; therefore, screening for and treatment of modifiable risk factors for cardiovascular disease is suggested (dyslipidemia, hypertension).

-Advise all patients not to use cigarettes and other tobacco products or e-cigarettes.

EVALUATION FOR NEWLY DIAGNOSED DIABETES

-Fasting glucose , Lipid profile, HbA1C, Urine analysis, Kidney function test.

-Microalbuminuria (spot albumin:creatinine ratio) and eGFR should be measured annually.

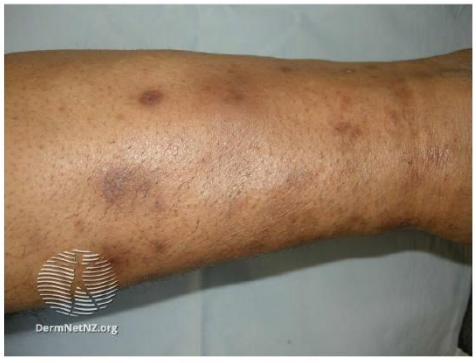
-Physical Exam. must include height, weight, blood pressure.

-Vision measurement and exam for retinopathy should be done annually.

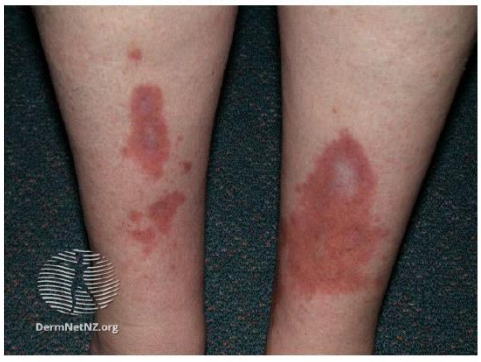
-Baseline neurological and cardiovascular exam should be obtained.

-Foot exam. should include peripheral pulses, sensation at every visit.

-Skin exam. for diabetic skin changes (dermopathy, acanthosis nigricans, necrobiosis lipoidica..)



Diabetic dermopathy



Necrobiosis lipoidica

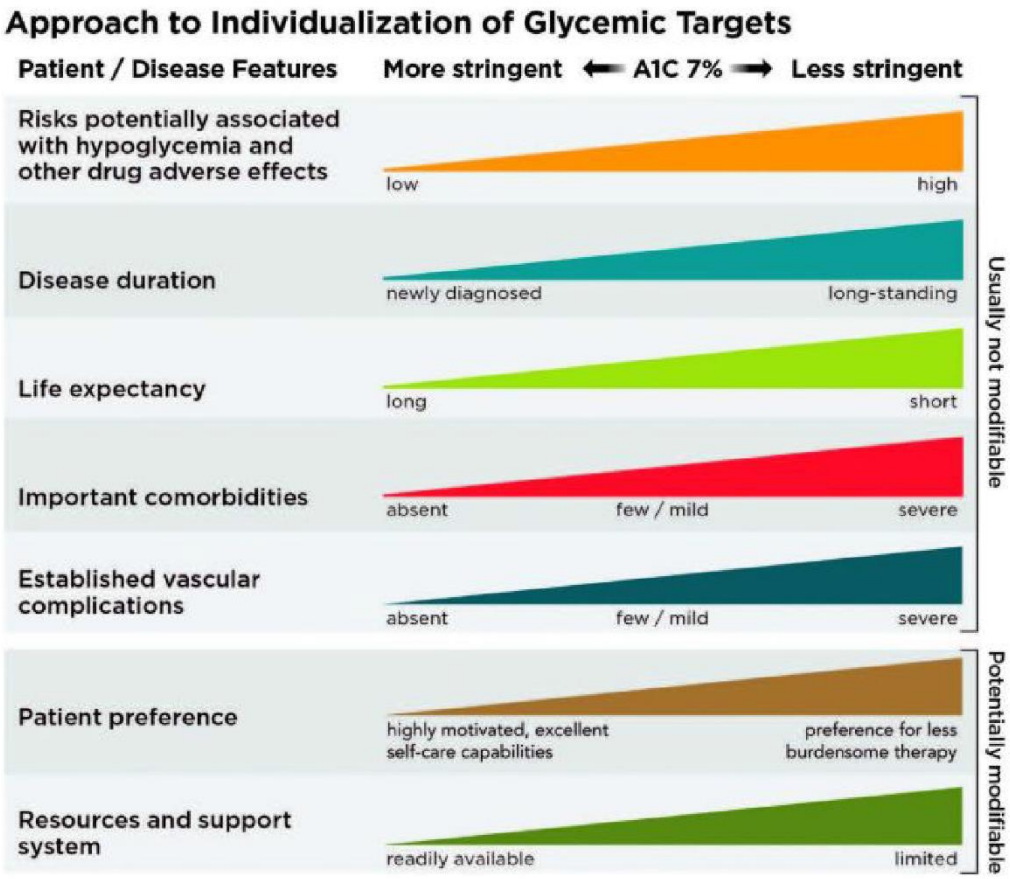
Acanthosis nigricans

GLYCEMIC TARGETS

-Glycemic management is primarily assessed with the A1C test, the primary measure studied in clinical trials demonstrating the benefits of improved glycemic control. Self-monitoring of blood glucose (SMBG) may help with self-management and medication adjustment, particularly in individuals taking insulin.

-Perform the A1C test at least two times a year in patients who are meeting treatment goals (and who have stable glycemic control).

-Perform the A1C test quarterly in patients whose therapy has changed or who are not meeting glycemic goals



-GENERALLY:

HbA1C < 7%

Pre-prandial glucose 70-130 mg/dl (3.9-7.2 mmol/L) Peak postprandial < 180 mg/dl (<10 mmol/L)

-PATIENT-CENTERED APPROACH/ INDIVIDUALIZED MANAGEMENT:

- A patient-centered approach should be used to guide the choice of pharmacologic agents. Considerations include comorbidities (ASCVD, heart failure, CKD), hypoglycemia risk, impact on weight, cost, risk for side effects, and patient preferences.

- Providers might reasonably suggest more stringent A1C goals (such as <6.5% [48 mmol/mol]) for selected individual patients if this can be achieved without significant hypoglycemia or other adverse effects of treatment (i.e., polypharmacy). Appropriate patients might include those with short duration of diabetes, type 2 diabetes treated with lifestyle or metformin only, long life expectancy, or no significant cardiovascular disease.

- Less stringent A1C goals (such as <8% [64 mmol/mol]) may be appropriate for patients with a history of level 3 hypoglycemia (altered mental and/or physical state requiring assistance), limited life expectancy, advanced microvascular or macrovascular complications, extensive comorbid conditions, or long-standing diabetes in whom the goal is difficult to achieve despite diabetes self-management education, appropriate glucose monitoring, and effective doses of multiple glucose-lowering agents including insulin.

MANAGEMENT

-Life style modification (as mentioned above)

-Medications:

METFORMIN (↓ HBA1C 1-2%)

-(insulin sensitizers) is the drug of choice for treatment of type 2 diabetic patients.

-Metformin reduces the incidence of myocardial infarction and increases survival (UKPDS). -It does not cause hypoglycemia and causes less weight gain and improves lipid profile.

-Metformin decreases blood glucose by:

a. Decreasing hepatic gluconeogenesis
b. Increasing glucose uptake in the muscles
c. Decreasing glucose absorption ?

-Daily dose 1.5 – 2.5 gm

-Once initiated, metformin should be continued as long as it is tolerated and not contraindicated; other agents, including insulin, should be added to metformin.

-Side effects = diarrhoea, nausea,B12 deficiency, lactic acidosis (avoid in patient with renal, hepatic and unstable heart failure, stop 48 hrs before surgery or contrast media).

SULPHONYLUREAS: (↓ HBA1C 1-2%)

-Act by stimulation of insulin release from B-cells of the pancreas (secretagogues).

-e.g. Glibenclamide, glipizide, gliclazide, glimepiride.

-Side effects: weight gain, hypoglycemia, skin reaction and hematological complications.

THIAZOLIDINEDIONES

-(Insulin Sensitizers)

-e.g. Pioglitazone and Troglitazone can be used as monotherapy or in combination with metformin, sulfonylurea or insulin.
-Can be used in end stage renal failure.

-Contraindications: Hepatic Impairment, Heart failure.

DPP-4 INHIBITORS: GLIPTINS

-Increase incretin levels which inhibit glucagon release, which in turn increases insulin secretion

-Have synergistic effect when combined with metformin.

GLP 1 (GLUCAGON-LIKE PEPTIDE-1 RECEPTOR AGONISTS)

-Injectable

-Favorable in patients with established ASCVD because of CVD benefit, ASCVD with HF, obesity and CKD.

SGLT2 (SODIUM-GLUCOSE TRANSPORT PROTEIN 2) INHIBITORS

- Inhibit reabsorption of glucose in the kidney .

-Favorable in patients with established ASCVD, ASCVD with HF, obesity and CKD.

INSULIN

-The early introduction of insulin should be considered if there is evidence of ongoing catabolism (weight loss), if symptoms of hyperglycemia are present, or when A1C levels (>10% [86 mmol/mol]) or blood glucose levels (≥300 mg/dL [16.7 mmol/L]) are very high.

-Basal Insulin accounts for approximately 50% of total insulin secreted each day where as the remaining 50% of the insulin is secreted in response to meals.

-Types of Insulin:

- Rapid-acting analogue e.g Aspart, Lispro
- Short-acting e.g Regular
- Intermediate-acting e.g NPH
- Long-acting e.g Glargine, Detemir
- Premixed Insulin

COMPLICATIONS

MACROVASCULAR COMPLICATIONS

- Atherosclerotic vascular disease (a) Coronary artery disease (b) MI with sudden death (c) C.V.A (d) Peripheral vascular dis. (e) Intestinal ischemia (f) Renal artery stenosis

-A patient with diabetes has 2-3 fold ↑ risk of developing CVD

MICROVASCULAR COMPLICATIONS

1) Diabetic nephropathy :

-Diabetic nephropathy occurs in 20-40% of pts. with diabetes & is the single leading cause of end-stage renal disease (ESRD)

-Microalbuminuria (persistent albuminuria in the range of 30-299 mg/24 hr) is the earliest stage of diabetic nephropathy and a marker of CVD risk

-The most Useful screening test is the albumin: creatinine ratio (AC Ratio)

-Control of diabetes and BP will reduce the risk or slow the progression of nephropathy

-ACE inhibitors usage is associated with significant reduction in progression to overt proteinuria & ↑ regression to normoalbuminuria

2) Diabetic neuropathy

-Distal symmetrical polyneuropathy, mononeuropathy, autonomic neuropathy are the main types

- Numbness, paresthesia, pain, decreased sensation especially vibration and proprioception, neuropathic ulcer may occur.

- Pregabalin, duloxetine, or gabapentin are recommended as initial pharmacologic treatments for neuropathic pain in diabetes , Tricyclic antidepressants, capsaicin also may be used.

3) Diabetic retinopathy

-DR is the leading cause of blindness and diabetic pts have 10% chance of acquiring blindness from retinopathy.

-Glaucoma, cataracts and other disorders of the eye occur earlier & more frequently in people with diabetes

-Screening by yearly fundoscopic exam or fundus photography

-Laser photocoagulation is indicated in pts with proliferative diabetic retinopathy (PDR), macular edema and some cases of NPDR.

PREVENTION & MANAGEMENT OF DIABETIC COMPLICATIONS

I) Hypertension

- A major risk factor for both CVD & microvascular complications in diabetics

● Lowering BP will reduce the incidence of coronary heart disease, stroke and nephropathy

● Target BP (JNC 8) : 140/90

● Multiple drug therapy is generally required

● An ACE inhibitor or ARB, at the maximum tolerated dose indicated for blood pressure treatment, is the recommended first-line treatment for hypertension in patients with diabetes and urinary albumin-to-creatinine ratio ≥300 mg/g creatinine or 30–299 mg/g creatinine.

2) Dyslipidemia

● For patients of all ages with diabetes and ASCVD or 10-year ASCVD risk >20%, high-intensity statin therapy should be added to lifestyle therapy.

● For patients with diabetes aged 40–75 years and >75 years without ASCVD, use moderate-intensity statin in addition to lifestyle therapy.

● In patients with diabetes who have multiple ASCVD risk factors, it is reasonable to consider high-intensity statin therapy.

● For patients with diabetes and ASCVD, if LDL cholesterol is ≥70 mg/dL on maximally tolerated statin dose, consider adding additional LDL-lowering therapy (such as ezetimibe or PCSK9 inhibitor). Ezetimibe may be preferred due to lower cost

3) Antiplatelet agents

● Use aspirin therapy (75–162 mg/day) as a secondary prevention strategy in those with diabetes and a history of ASCVD.

● For patients with ASCVD and documented aspirin allergy, clopidogrel (75 mg/day) should be used.

● Aspirin therapy (75–162 mg/day) may be considered as a primary prevention strategy in those with diabetes who are at increased cardiovascular risk (ASCVD risk after a discussion with the patient on the benefits versus increased risk of bleeding.

4) Smoking Cessation

-Advice all pts not to smoke cigarettes or e-cigarettes.

PATIENT SELF-CARE AND PROVIDER PRACTICES TO IMPROVE OUTCOMES

Patient counseling, education and motivation are vital for short term and long term goal achievements.

1. Patient Self-Care Practices

1. Regular medical appointments to assess control and for complication surveillance / prevention.

2. Healthful meal planning

3. Regular exercise

4. Regular medication use as prescribed

5. Glucose self-monitoring (take results to appointment)

6. Adjustment of medication based on glucose results

7. Daily foot check

8. Annual eye check

9. Annual dental check

2. Provider Office Practices

1. Patient education (team approach)

Nutrition education for patient

Exercise prescription

Glucose self-monitoring

Medication adjustment

Foot Care

2. Ask about:

-Hyper / Hypoglycemic symptoms

-Impotence and autonomic dysfunction symptoms

-Cardiac and vascular disease symptoms

3. Each visit:

Check weight, Blood Pressure, glucose, condition of feet

Check glucose self-monitoring results / make recommendations

4.Referrals

* Annual dilated eye exam

* Family planning for women of reproductive age

* Registered dietitian.

* Diabetes self-management education

* Dental exam

* Mental health professional if needed

5.Laboratory: HbA1C every 3-6 months

KFT, lipids, urine microalbuminuria and eGFR yearly or as needed

6.Vaccination according to indications and age; Influenza, pneumococcal vaccines.

n

Foot care

-Inspect your feet daily .

-Bathe feet in lukewarm, never hot, water and keep your feet clean by washing them daily.

-Be gentle when bathing your feet .

-Moisturize your feet but not between your toes .

-Cut nails carefully.

-Never treat corns or calluses yourself .

-Wear clean, dry socks & Change them daily.

-Wear socks to bed. If your feet get cold at night, wear socks & Never use a heating pad or a hot water bottle.

-Shake out your shoes and feel the inside before wearing .

-Keep your feet warm and dry .

-Consider using an antiperspirant on the soles of your feet .

-Never walk barefoot & Get periodic foot exams .

DYSURIA

- The most common cause of acute dysuria is infection, especially **cystitis**. Other infectious causes include urethritis, sexually transmitted infections, and vaginitis.
- Noninfectious inflammatory causes include a foreign body in the urinary tract and dermatologic conditions.
- Noninflammatory causes of dysuria include medication use, urethral anatomic abnormalities, local trauma, and interstitial cystitis/bladder pain syndrome.

An initial targeted history includes features of a local cause (e.g., vaginal or urethral irritation), risk factors for a complicated urinary tract infection (e.g., male sex, pregnancy, presence of urologic obstruction, recent procedure), and symptoms of pyelonephritis.

- Women with dysuria who have no complicating features can be treated for cystitis without further diagnostic evaluation. Women with vulvovaginal symptoms should be evaluated for vaginitis.

Any complicating features or recurrent symptoms warrant a history, physical examination, urinalysis, and urine culture.

Findings from the secondary evaluation, selected laboratory tests, and directed imaging studies enable physicians to progress through a logical evaluation and determine the cause of dysuria or make an appropriate referral.

DIFINITION

Dysuria is burning, tingling, or stinging of the urethra and meatus associated with voiding.

- ❖ It should be distinguished from other forms of **bladder discomfort,** such as suprapubic or retropubic pain, pressure, or discomfort that usually increases with bladder volume.

Dysuria is present at least occasionally in approximately 3% of adults older than 40 years, according to a survey of roughly 30,000 men and women.

Acute cystitis is the most common cause in women, accounting for 8.6 million outpatient visits in 2007 and 2.3 million emergency department visits in 2011.

This article describes an evidence-based approach to the evaluation of adult outpatients with dysuria, focusing on the history, physical examination, and selected tests.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*	*Comments*
In low-risk women with dysuria and no vaginal symptoms or other typical UTI symptoms, physicians should obtain a dipstick urinalysis for nitrites and leukocyte esterase.	C	24, 25	Nitrites have higher predictive value for UTI but also higher false-negative rates than leukocyte esterase.
Patients with dysuria who are at risk of complications or whose symptoms do not respond to initial treatment should undergo a detailed history, directed physical examination, and urinalysis and culture.	C	8, 10	Clinical evaluation is useful to direct additional workup.
Further investigation and urology referral should be considered in patients with recurrent UTI, urolithiasis, known or suspected urinary tract abnormality or cancer, history of urologic surgery, hematuria, persistent symptoms, or in men with abnormal postvoid residual urine level (greater than 100 mL).	C	8, 10, 11, 29, 33	Some evaluations, such as postvoid residual urine, computed tomography urography, and symptom questionnaires, can be initiated by the family physician.
Women with an uncomplicated history who present with acute dysuria, urinary urgency or frequency, and no vaginal discharge can be treated for acute cystitis without other evaluation.	B	9, 23-25, 31, 35	Uncomplicated history includes 16 to 55 years of age, not pregnant, no history of recurrent or childhood UTI, not immunocompromised, no diabetes mellitus, and no anatomic urologic abnormality or recent urologic instrumentation.

UTI = urinary tract infection.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.

PATHOPHYSIOLOGY AND DIFFERENTIAL DIAGNOS

Sensory nerves are located just beneath the urothelium. Chemical irritation and inflammatory conditions (e.g., acute bacterial infection) can alter the mucosal barrier and stimulate these nerves, causing pain.

Chronic inflammation and other unknown factors can lead to altered nerve sensitivity and persistent pain. Inflammation from adjacent abdominal structures, such as the colon, can also affect function and sensation in the bladder.

Inflammatory disorders of the bladder and urethra are the most common causes of dysuria. Among these, infections of the bladder, urethra, kidneys, and genital organs are the most prevalent, including uncomplicated cystitis, pyelonephritis, and urethritis.

- ❖ Distinguishing a complicated urinary tract infection (UTI) from cystitis is important, because misdiagnosis increases the risk of treatment failure. Risk factors for a complicated infection may include patient characteristics, medical conditions, and urologic conditions (***Table 1***). In women, dysuria is also a common presentation of **vaginitis**. In men, **prostatitis** can present with dysuria. Sexually transmitted infections (STIs) can also cause dysuria.

Table 1. Risk Factors for Complicated Urinary Tract Infections*

Patient characteristics	**Urologic conditions**
Male sex	History of childhood or recurrent urinary tract infections†
Postmenopausal†	Indwelling catheter
Pregnant	Neurogenic bladder
Presence of hospital-acquired urinary tract infection	Polycystic kidney disease
Symptoms present for seven or more days before presentation	Recent urologic instrumentation
Medical conditions	Renal transplant
Diabetes mellitus†	Urolithiasis
Immunocompromised status	Urologic obstruction
	Urologic stents

*—Increased chance of treatment failure.
†—Some experts consider the following groups to be uncomplicated: healthy postmenopausal women; patients with well-controlled diabetes; and patients with recurrent cystitis that responds to treatment.[11]

Information from references 8 through 11.

Inflammatory, noninfectious conditions that can lead to dysuria include the presence of a foreign body (e.g., stent, bladder stone), noninfectious urethritis (e.g., reactive arthritis, formerly Reiter syndrome), and dermatologic conditions.

Noninflammatory conditions can be divided into the following categories: anatomic; endocrine; neoplastic; medication-, food-, or recreational drug–related; iatrogenic; and idiopathic.

Any condition that causes hematuria with clots can cause dysuria, including renal neoplasms and nephrolithiasis. Interstitial cystitis (also known as bladder pain syndrome) refers to chronic bladder pain, often with voiding symptoms, lasting six weeks or more without an identifiable cause. The differential diagnosis of dysuria is summarized in (***Table 2)***.

Table 2. Differential Diagnosis of Dysuria in Adults

Category	*Sex*	*Causes**
Inflammatory		
Dermatologic	Both	Irritant or contact dermatitis, lichen sclerosus, lichen planus, psoriasis, Stevens-Johnson syndrome, Behçet syndrome
Infectious	Both	Cystitis, urethritis, pyelonephritis, other sexually transmitted infections
	Women	Vulvovaginitis, cervicitis
	Men	Prostatitis, epididymo-orchitis
Noninfectious	Both	Foreign body (e.g., stent, stone), urethritis (e.g., reactive arthritis)
Noninflammatory		
Anatomic	Both	Urethral stricture or diverticulum
	Men	Benign prostatic hyperplasia
Drug- or food-related	Both	Spermacides, topical deodorants, cyclophosphamide, opioids, ketamine (Ketalar), nifedipine, and others; bladder-irritating foods
Endocrine	Women	Atrophic vaginitis, endometriosis
Idiopathic	Both	Interstitial cystitis/bladder pain syndrome
Neoplastic	Both	Bladder or renal cancer, lymphoma, metastatic cancer†
	Women	Vaginal or vulvar cancer, paraurethral leiomyoma
	Men	Prostate or penile cancer
Trauma/iatrogenic	Both	Genitourinary instrumentation or surgery, pelvic irradiation, foreign body presence, horseback or bicycle riding

*—Infectious causes, particularly acute cystitis, are the most common. There are few data to rank other diagnoses by prevalence; specific causes are listed by estimated prevalence.
†—Some cancers (e.g., renal cell) present with dysuria primarily by causing hematuria, and others by bladder-wall irritation, which may be difficult to distinguish from true dysuria.

Information from references 2, 4, 8, and 11 through 19.

HISTORY AND PHYSICAL EXAMINATION

HISTORY

The medical history should characterize the:

1. Timing
2. Persistence
3. Severity
4. Duration
5. exact location of the dysuria.

Pain occurring at :

1. the start of urination may indicate urethral pathology
2. the end of urination is usually of bladder origin.

-Physicians should ask about other bladder symptoms: frequency , Urgency , Incontinence , hematuria , malodorous urine and nocturia.

The history should include the presence of flank pain, nausea, fever, and other systemic symptoms.

A history of dysuria, UTIs, STIs, and recent sexual activity are crucial. Additionally, medication use, family history, and procedural history can help identify the cause of dysuria.

In women, the history should also include the presence of vaginal discharge or irritation, most recent menstrual period, and type of contraception used.

Specific localization of the discomfort varies between men and women. **Women with vaginitis** often describe external dysuria, as well as vaginal irritation or discharge. With cystitis, the dysuria is characteristically felt in the bladder or urethra.

In addition to dysuria, **men with prostatitis** may have deep perineal pain and obstructive urinary symptoms, whereas those with epididymo-orchitis may have localized testicular pain.

Lesions from herpes simplex virus of the vulvar or penile area may cause dysuria. Patients with interstitial cystitis may have suprapubic or abdominal pain related to bladder filling. These patients nearly always report urinary frequency and urgency, whereas dysuria is variable.

A meta-analysis in which approximately 50% of patients had a UTI found that the highest positive predictive value (PV+) of cystitis in women was selfdiagnosis of cystitis (86%), followed by the absence of vaginal discharge (82%), presence of hematuria (75%), and urinary frequency (73%). This review found that the combination of dysuria and urinary frequency without vaginal discharge or irritation yielded a very high likelihood of UTI (positive likelihood ratio [LR+] = 24.6).

A woman with dysuria and frequency, no risk factors for complicated infection, and no vaginal discharge had a 90% probability of UTI; thus, treatment based on symptoms alone was advocated.

A study of 196 symptomatic women found that 79% of patients with "considerable" dysuria, suspicion of a UTI, and absence of vaginal symptoms had a UTI. In a prospective study of 490 men with symptoms of a UTI, symptoms of dysuria and urgency were significantly associated with a positive urine culture. A suggested history for patients with dysuria is provided in (***eTable A)***.

eTable A. Elements of a Detailed History for Select Patients with Dysuria

Category	*Question or finding*	*Possible diagnoses*
Dermatologic	Perineal rash or irritation; generalized dermatitis; mucosal symptoms or lesions	Sexually transmitted infection; atrophy; lichen sclerosus; dermatitis (psoriasis, lichen planus)
Exposures	Smoking/tobacco; occupational or environmental (benzene, aromatic amines); past pelvic irradiation	Genitourinary cancer
Family history	Autosomal dominant polycystic kidney disease; other renal disease	Autosomal dominant polycystic kidney disease; other causes or hematuria, pyuria, or abnormal urinalysis result
Gastrointestinal	Nausea; vomiting; diarrhea; constipation; rectal pain; relationship of symptoms to defecation	Pyelonephritis; adjacent abdominal inflammatory condition (inflammatory bowel disease, diverticulitis); interstitial cystitis/bladder pain syndrome
Genitourinary	Bladder outlet obstructive symptoms (hesitancy, decreased stream, dribbling); irritative symptoms (urgency, frequency); incontinence; pain with intercourse; urethral and testicular symptoms; pain with ejaculation; cyclic symptoms/relation to menses; postmenopausal; vaginal symptoms; rash	Vaginitis; sexually transmitted infection; prostatitis; urethritis; epididymitis; stricture; interstitial cystitis/bladder pain syndrome; endometriosis; urethral diverticulum
Medical history	Neurologic disease or injury; systemic inflammatory disease; active cancer	Neurogenic bladder; interstitial nephritis or glomerulitis; metastatic disease
Medications	Current medication and supplement use; recreational drug use; prior treatment with cyclophosphamide	Can cause irritative bladder symptoms directly; interstitial nephritis or cystitis; bladder cancer
Surgical/trauma history	Remote or recent genitourinary or abdominal surgery or irradiation; history of stones; recent bladder catheterization or procedure	Nephrolithiasis; stricture; radiation cystitis; hematuria from instrumentation
Systemic symptoms	Fever; arthralgias; ocular symptoms	Pyelonephritis; transient glomerular injury due to febrile illness; spondyloarthropathy or autoimmune disease

Information from:

Bent S, Nallamothu BK, Simel DL, Fihn SD, Saint S. Does this woman have an acute uncomplicated urinary tract infection? JAMA. 2002;287(20):2701-2710.

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Hooton TM. Clinical practice. Uncomplicated urinary tract infection. N Engl J Med. 2012;366(11):1028-1037.

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PHYSICAL EXAMINATION

especially when complicated UTI is a consideration

1. should include vital signs
2. evaluation for costovertebral angle pain
3. palpation for abdominal mass or tenderness,
4. inspection for dermatologic conditions and acute joint effusions.

Often the most relevant findings on physical examination are sexspecific, including inspecting for infectious or atrophic vaginitis and STIs in women, and prostatitis and STIs in men.

The presence of costovertebral angle tenderness on examination modestly increases the likelihood of having a UTI in women (LR+ = 1.7). Key physical examination findings are discussed in (**eTable B**).

Risk factors for complicated UTI and failure to respond to initial treatment are indications for a more detailed history and physical examination.

eTable B. Physical Examination for Select Patients with Dysuria

Area of examination	*Physical findings*	*Possible implications*
Abdomen	Abdominal tenderness or mass	Suprapubic tenderness consistent with cystitis; other tenderness or mass suggests adjacent process, such as diverticulitis or malignancy; bladder distention suggests obstruction or retention
	Costovertebral angle tenderness	Pyelonephritis, stone, or obstruction with hydronephrosis
Dermatologic	Rash, generalized or local to genital area	Psoriasis; lichen planus; Stevens-Johnson syndrome; other systemic dermatoses with genital manifestations causing local irritation; candidiasis
	Isolated pustules of extremities	Systemic gonococcal infection
General/vital signs	Hypertension	Glomerulonephritis
	Fever	Pyelonephritis; systemic illness
Genitals (women)	Vulvar vesicles, ulcers, pustules; inguinal lymphadenopathy; cervical discharge	Herpes simplex virus infection; chancroid; other sexually transmitted infections
	Vaginal discharge and/or mucosal inflammation	Vaginitis
	Cervical motion tenderness; mass or tenderness on bimanual examination	Endometriosis; pelvic inflammatory disease; gynecologic mass; urethral diverticulum
	Vulvovaginal atrophy	Atrophic vaginitis; lichen sclerosus
	Locally tender areas with otherwise normal findings	Vulvodynia; tension myalgia; interstitial cystitis/bladder pain syndrome
	Urethral mass or tenderness	Urethral diverticulum or endometriosis
Genitals (men)	Penile discharge; urethral meatal inflammation; penile vesicles, ulcers, pustules; inguinal lymphadenopathy	Urethritis; herpes simplex virus infection; chancroid; other sexually transmitted infections
	Swelling, tenderness of epididymis or testicle	Epididymo-orchitis
	Prostate boggy swelling with tenderness	Prostatitis
	Enlarged symmetric prostate	Benign prostatic hyperplasia
	Prostate with focal abnormality or hardness, asymmetry, nodule	Prostate cancer
	Locally tender areas with otherwise normal findings	Tension myalgia; interstitial cystitis/bladder pain syndrome
Other	Joint effusions; conjunctivitis	Reactive arthritis (conjunctivitis, arthritis, and urethritis/cervicitis); other systemic disease with local genital manifestations
	Polyarticular tenosynovitis	Systemic gonococcal infection
	Neurologic disease	Neurogenic bladder

Information from:
Bent S, Nallamothu BK, Simel DL, Fihn SD, Saint S. Does this woman have an acute uncomplicated urinary tract infection? JAMA. 2002;287(20):2701-2710.
Grady D. Clinical practice. Management of menopausal symptoms. N Engl J Med. 2006;355(22):2338-2347.
Gupta K, Trautner B. In the clinic. Urinary tract infection. Ann Intern Med. 2012;156(5):ITC3-1-ITC3-15.
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LABORATORY TESTS

URINALYSIS

Urinalysis is the most useful test in a patient with dysuria; most studies have used dipstick urinalysis. Multiple studies of women with symptoms suggestive of a UTI have demonstrated that the presence of **nitrites** is highly predictive of a positive culture (PV+ = 75% to 95%); dipstick showing more than trace **leukocytes** is nearly as predictive (PV+ = 65% to 85%); and the presence of **both** is almost conclusive (PV+ = 95%).

Urinary nitrites may be falsely negative in women with a UTI. Few studies specifically address the value of urinalysis in men with dysuria, but evidence suggests similar value to the combination of leukocyte esterase, nitrites, and possibly blood. Leukocyte esterase or pyuria alone with isolated dysuria suggests urethritis .

CULTURES AND CYTOLOGY

Any patient with risk factors for a complicated UTI (Table 1) or whose symptoms do not respond to initial treatment should have a urine culture and sensitivity analysis.

Patients with suspected pyelonephritis should have renal function assessed with serum creatinine measurement, and electrolyte levels should be measured if there is substantial nausea and vomiting.

Blood cultures are usually not necessary, but can be obtained in patients with high fever or risk of infectious complications.

- In **women** with vaginal symptoms, secretions should be evaluated with wet mount and potassium hydroxide microscopy or a vaginal pathogens DNA probe. Urethritis should be suspected in younger, sexually active patients with dysuria and pyuria without bacteriuria; in men, urethral inflammation and discharge is typically present. In patients with suspected urethritis, a urethral, vaginal, endocervical, or urine nucleic acid amplification test for Neisseria gonorrhoeae and Chlamydia trachomatis is indicated. Genital ulcerations can be sampled for herpes simplex virus culture or polymerase chain reaction testing, as well as testing for other STIs.
- In **men** with suspected chronic prostatitis, urine culture after gentle prostatic massage can yield the causative bacterial agent. Prostate-specific antigen level is transiently elevated during acute prostatitis and should not be measured in patients with acute inflammatory symptoms. Urine cytology is helpful if bladder cancer is suspected, such as in older patients with hematuria and a negative culture result .

IMAGING AND OTHER ADVANCED STUDIES

Imaging is not necessary in most patients with dysuria, although it may be indicated in patients with a complicated UTI, a suspected anatomic anomaly (e.g., abnormal voiding, positive family history of genitourinary anomalies), obstruction or abscess, relapsing infection, or hematuria.

1. Ultrasonography : is the preferred initial test for patients with obstruction, abscess, recurrent infection, or suspected kidney stones, because it avoids radiation exposure.
2. Helical computed tomography urography : is used to view the kidneys and adjacent structures, and may be considered to further evaluate patients with possible abscess, obstruction, or suspected anomalies when ultrasonography is not diagnostic.

3. If urinalysis is unrevealing, cystoscopy can be performed to evaluate for bladder cancer, hematuria, and chronic bladder symptoms.
4. Urodynamic studies can be performed for persistent voiding symptoms with otherwise unrevealing workup, although a recent Cochrane review found no evidence that these tests led to reduction in symptoms in men with such indications.

Further investigation and urology referral should be considered in patients with recurrent UTI, urolithiasis, known urinary tract abnormality or cancer, history of urologic surgery, hematuria, persistent symptoms, or in men with abnormal postvoid residual urine level (greater than 100 mL) (***Table 3***)

Table 3. Diagnostic Tests for Select Patients with Dysuria

*Test**	*Indications*
Ultrasonography	Initial imaging study for most patients when imaging is indicated; useful in patients who have iodinated contrast media allergy or pregnancy; measurement of the bladder residual volume helps evaluate benign prostatic hyperplasia; secondary study in recurrent UTIs, complicated pyelonephritis, or hematuria
Plain abdominal radiography (kidneys, ureters, bladder)	Most useful in known urolithiasis
CT of abdomen and pelvis with and without contrast media (CT urography)	Evaluation of hematuria, recurrent UTI (with risk factors or relapses), and complicated pyelonephritis
CT of abdomen and pelvis without contrast media	Suspected urolithiasis (ultrasonography is best initial study)
Cystoscopy	Voiding symptoms, hematuria, recurrent UTI, concern for urethral diverticula, bladder cancer, or interstitial cystitis/bladder pain syndrome
Intravenous urography	Useful for hematuria evaluation if CT urography is unavailable
Magnetic resonance imaging of abdomen and pelvis with and without contrast media (MR urography)	Most useful for complicated pyelonephritis; helpful, not preferred, for stones and hematuria

CT = computed tomography; UTI = urinary tract infection.

**—Depending on complicating features, tests are listed in order of preferred use.*

Information from references 4, 8, 9, 11, 21, 23 through 26, 28 through 30, 33, and 34.

CLINICAL DECISION RULES

More than 400 patients were included in an English study to develop and validate a clinical decision rule for women presenting with dysuria and urinary frequency. The scoring system they developed can be used in a two-stage process, with some patients treated empirically based on symptoms alone (Table 4).

Negative predictive values are poor; many women with UTI will not have all three clinical symptoms that were found to be predictive of UTI (dysuria, nocturia, and offensive urine odor), and about one-fourth of patients with urinary symptoms and a normal dipstick result have UTI, so appropriate follow-up is important.

Table 4. Clinical Decision Rule for UTI in Women Without Signs of Complicated Disease

*Clinical score**		*Dipstick score**	
Symptom	*Points*	*Dipstick result*	*Points*
Urine cloudiness	1	Nitrites	2
Any burning dysuria	1	Leukocyte esterase	1.5
Nocturia of any degree	1	Blood	1
Total:	____	**Total:**	____
0 points: LR = 0.23; prevalence = 19%		*0 points: LR = 0.16; prevalence = 14%*	
1 or 2 points: LR = 0.82; prevalence = 46%		*1 to 2.5 points: LR = 1.1; prevalence = 53%*	
3 points: LR = 2.25; prevalence = 70%		*≥ 3 points: LR = 5.4; prevalence = 85%*	

NOTE: *Stratum-specific likelihood ratios and prevalences are calculated from data in Tables 2 and 5 in reference 23.*

LR = likelihood ratio; UTI = urinary tract infection.

**—Suggested use: First, calculate clinical score. If score equals 3, treat empirically. If less than 3, obtain urinalysis and treat if dipstick score is 2 or higher, or based on clinical judgment.*[23]

Information from reference 23.

A Dutch study included 490 outpatient men with dysuria, frequency, or urgency, while excluding men with symptoms suggestive of an STI or a complicated UTI. The authors found that the combination of age (60 years or older) and either a positive leukocyte esterase or nitrite test result had the best positive and negative predictive values for UTI (83% and 60%, respectively).

Another study developed a symptoms- history-urinalysis score that included symptoms of frequency, nocturia, dysuria, hematuria, and offensivesmelling urine; history of a previous UTI; and urinalysis results (protein, blood, and nitrites). The study found that a score of 0 or greater on the 13-item score sheet (range of possible scores from –19 to +31) identified 85% of women with infection; 25% of women without infection also had a score of 0 or greater.

All of the decision rules caution that in a patient with dysuria, these combinations of variables can reassure physicians that a UTI is likely present, but are not very useful in ruling out a UTI when they are absent. It is important to address the

other clinical features discussed here to narrow the differential diagnosis.

APPROACH TO THE PATIENT

Many studies advise that, in the right clinical setting, symptoms alone can identify patients with a high likelihood of UTI who are candidates for empiric therapy. Women with an uncomplicated history who present with acute dysuria, urinary urgency or frequency, and no vaginal discharge can be treated for acute cystitis without other evaluation.

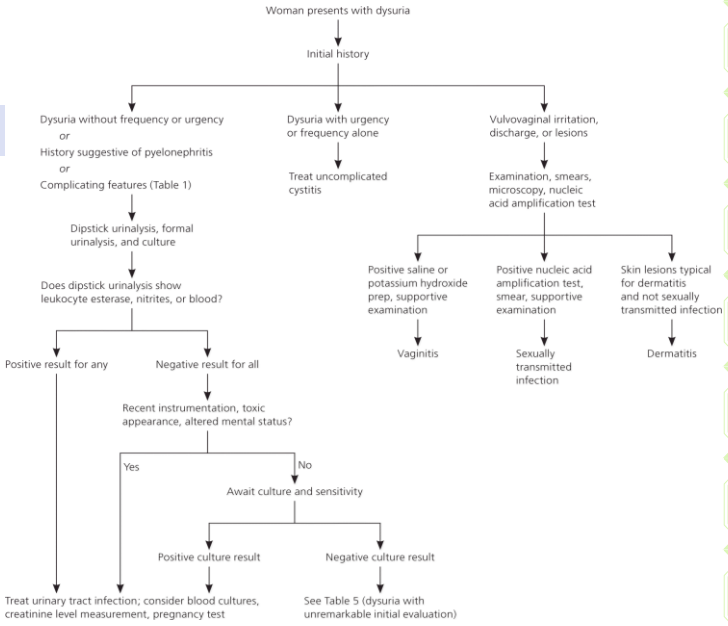
Several studies suggest that this approach is effective in reducing costs while improving patient satisfaction, with no increase in adverse outcomes.

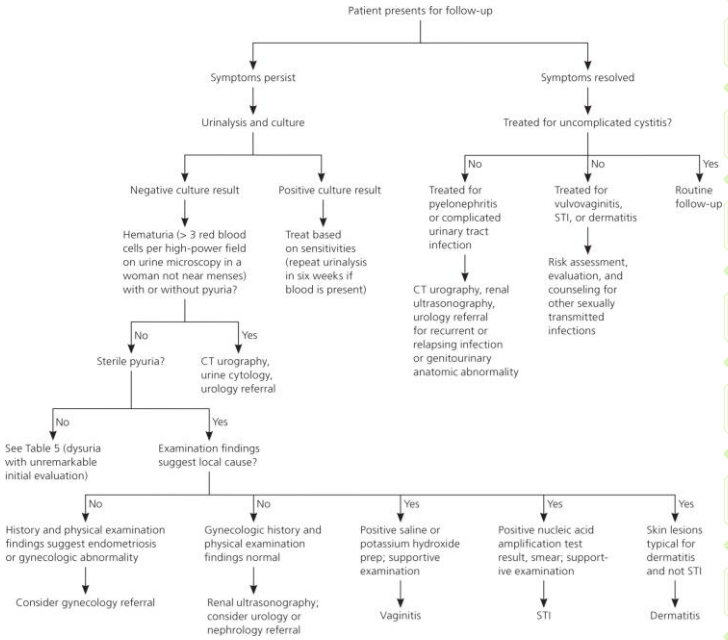
This approach is reflected in the algorithms presented here for the evaluation and follow-up of dysuria. These algorithms are based on evaluation of the existing evidence.

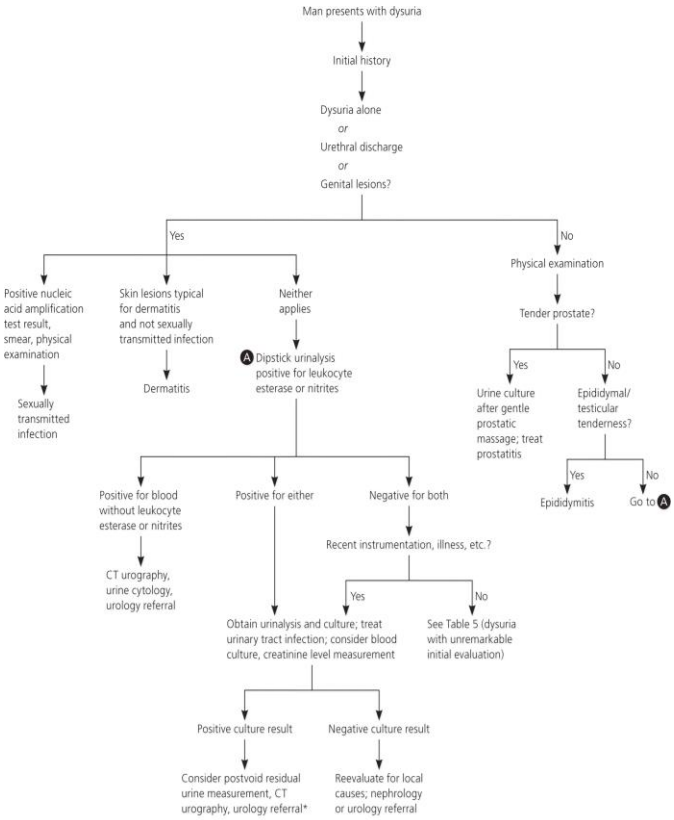
For patients with initially normal dipstick urinalysis and culture results, ***Table 5*** lists common conditions that may be causing dysuria, the typical presentation, and management recommendations.

It is important to use clinical judgment and to be aware of the inclusion and exclusion criteria for the studies on which these algorithms are based. For example, a sexually active adolescent with dysuria is more likely to have an STI than cystitis, and urinalysis results may be negative.

Similarly, an older patient who experiences dysuria shortly after having an indwelling catheter and who has a negative urinalysis result still has a high likelihood of having a UTI.







*—Men do not routinely need imaging, cystoscopy, and urinary flow measurement; some experts suggest that postvoid residual urine levels should be measured routinely. However, there are a number of features that should prompt further urologic evaluation: presence of fever, abnormal physical examination findings, history of recurrent urinary tract infections, history of urolithiasis, concern for renal impairment or urologic cancer, or postvoid residual urine volume greater than 100 mL.[27,39]

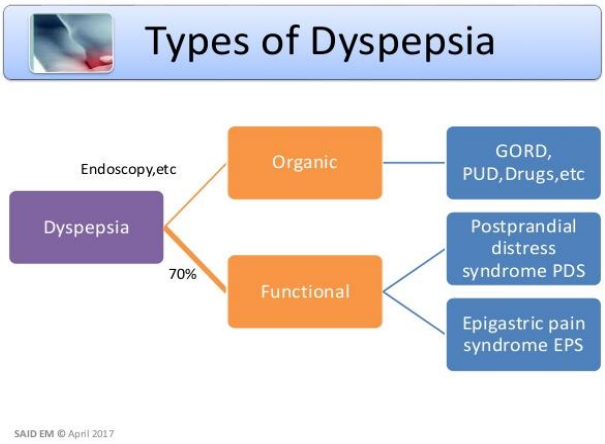
Table 5. Considerations in Patients with Dysuria and Unremarkable Initial Evaluation

Condition suspected	*Typical presentation*	*Recommendation*
Interstitial cystitis/bladder pain syndrome	Variable dysuria; frequency and urgency as primary symptoms; pain with bladder filling and relief with emptying are most specific	Initiate conservative treatment (symptom diary to modify fluid intake, diet, and physical activity; bladder training)
Overactive bladder	Prominent urgency, frequency, possible urge incontinence	Fluid restriction, bladder training, pelvic floor muscle exercises, drug therapy as needed empirically
Potentially offending topical irritant	History of topical use with or without examination findings	Discontinue use of offending agent
Suspected bladder irritants	Based on review of medications and diet*	Dietary and medication modification*
Urethral diverticulum or endometriosis (women)	Localized symptoms with or without physical examination findings	Urology or gynecology referral
Urethritis	Localized symptoms; suspect based on exposures and physical examination	Examination, smears, microscopy, and/or nucleic acid amplification testing

*—For a detailed list of bladder-irritating foods, see http://my.clevelandclinic.org/disorders/overactive_bladder/hic_bladder_irritating_foods.aspx.

Information from references 4, 12 through 19, and 28.

DYSPEPSIA



DEFINITION

DESCRIPTION

A group of epigastric symptoms classified based on presenting symptoms .

The presence of bothersome postprandial fullness, early satiety, or epigastric pain/burning in the absence of causative structural disease (to include normal upper endoscopy) for at least 1 to 3 days per week for the preceding 3 months, with initial symptom onset at least 6 months prior to diagnosis (Rome IV criteria) .

TABLE 1

Rome IV Diagnostic Criteria for Functional Dyspepsia

Presence of at least one of the following:

- Postprandial fullness (3 days per week)
- Early satiety (3 days per week)
- Epigastric pain (1 day per week)
- Epigastric burning (1 day per week)

and

No evidence of structural disease

Note: Criteria must be present for at least the past three months, with symptoms starting at least six months before diagnosis.

Information from reference 3.

- Rome IV criteria divide patients into two subtypes:
  - Postprandial distress syndrome (PDS)
  - Epigastric pain syndrome (EPS)

- Functional dyspepsia also called

  -diopathic dyspepsia

  -nonulcer dyspepsia

  -nonorganic dyspepsia

INCIDENCE

Unknown, accounts for 70% of patients with dyspepsia, and 5% of primary care visits .

PREVALENCE

- prevalence worldwide **10 -30 %**
- Predominant age: **adults** but can be seen in children
- Predominant gender: **female** > male

ETIOLOGY AND PATHOPHYSIOLOGY

Unknown, but proposed mechanisms or associations include:

1. gastric motility disorders
2. visceral pain hypersensitivity
3. Helicobacter pylori infection
4. alteration in upper GI microbiome
5. Medications
6. anxiety
7. depression
8. immune activation
9. Inflammation
10. gut-brain axis disorders

GERIATRIC CONSIDERATIONS

Patients ≥60 years with new-onset dyspepsia should undergo endoscopy .

PREGNANCY CONSIDERATIONS

Pregnancy may exacerbate symptoms .

RISK FACTORS

- Anxiety/depression
- psychosocial factors: divorce, unemployment
- Smoking
- Female gender
- :Other functional disorders
  - fibromyalgia
  - temporomandibular joint pain
  - chronic fatigue syndrome

DIAGNOSIS

HISTORY

1. Postprandial fullness
2. Early satiety
3. Epigastric pain
4. Epigastric burning
5. Symptoms for 3 months

ALARM FEATURES THAT MAY NECESSITATE ENDOSCOPY INCLUDE

1. Unintended weight loss
2. Progressive dysphagia
3. Odynophagia
4. Persistent vomiting
5. GI bleeding
6. Family history of upper GI cancer
7. Age ≥60 years

PHYSICAL EXAM

1. Document weight status and vital signs
2. Murphy sign for cholelithiasis
3. Rebound and guarding for ulcer perforation
4. Palpation during muscle contraction for abdominal wall
5. pain
6. Jaundice
7. Thyromegaly

DIFFERENTIAL DIAGNOSIS

1. Peptic ulcer disease; gastroesophageal reflux disease
2. Cholecystitis; choledocholithiasis
3. Gastric or esophageal cancer; esophageal spasm
4. Malabsorption syndromes; celiac disease
5. Pancreatic cancer; pancreatitis
6. Inflammatory bowel disease; carbohydrate malabsorption;
7. gastroparesis
8. Ischemic bowel disease
9. Intestinal parasites
10. Irritable bowel syndrome
11. Ischemic heart disease
12. Diabetes mellitus
13. thyroid disease
14. Connective tissue disorders
15. Medication effects

INITIAL TESTS (LAB, IMAGING)

- Functional dyspepsia is a diagnosis of exclusion
- Order labs based on clinical suspicion

1. Test for **H. pylori** (stool antigen or urea breath test) in areas of high H. pylori prevalence
2. **CBC**(if anemia or infection are suspected)
3. **Liver-associated enzymes**/right upper quadrant ultrasound (if hepatobiliary disease is suspected)
4. **Pancreatic enzymes** (if pancreatic disease is suspected)
5. **Upper endoscopy** for patients ≥60 to rule out malignancy

GENERAL MEASURES

- Reassurance and physician support are helpful
- Treatment is based on presumed etiologies
- Discontinue offending medications
- Routine endoscopy not recommended in dyspeptic patients <60 years evenwith alarm features

MEDICATION

FIRST LINE

- Treat H. pylori if confirmed on testing
- Trial of once daily proton pump inhibitor (PPI) medication (e.g., omeprazole 20 mg PO QD) or H2RA (e.g., ranitidine 150 mg BID) for up to 8 weeks in patients without alarm symptoms. This is most effective in EPS .

PROKINETICS

- Prokinetics have been proposed as first-line agents in PDS
- Prokinetics should be prescribed at the lowest effective dose to avoid potential side effects
- Use with caution in elderly patients due to side effects of tardive dyskinesia and parkinsonian symptoms

SECOND LINE

- Trial of tricyclic antidepressant (TCA) medication is more helpful for EPS than PDS
- There is no benefit to SSRI/SNRI

ADDITIONAL THERAPIES

- Stress reduction
- Psychotherapy effective in some patients
- Patients should be given a positive diagnosis and reassured of benign prognosis

DIET

- Consider limiting fatty foods
- Avoid foods that exacerbate symptoms:

1. wheat
2. cow milk
3. Peppers
4. spices
5. coffee
6. tea
7. alcohol

FOLLOW-UP RECOMMENDATIONS

- Patient Monitoring
- Provide ongoing support and reassurance
- Upper endoscopy if persistent symptoms
- Change medications if no difference in symptoms after 4 weeks
- Discontinue medications once symptoms resolve

CLINICAL PEARLS

- Dyspepsia without underlying organic disease is functional or idiopathic
- Consider empiric acid suppression therapy as first line for functional dyspepsia
- Extensive diagnostic testing is not recommended unless alarm symptoms are present

PROMOTING SMOKING CESSATION

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
All adults should be screened routinely for tobacco use.	A	6
All smokers should be encouraged to quit at every clinical contact.	A	4, 20
Motivational interventions should be used with patients who are not yet ready to quit smoking.	A	4, 25
Physicians should encourage appropriate patients to use effective medications for treatment of tobacco dependence to improve quit rates.	A	4, 27-29
Heavy smokers should be encouraged to use higher dosages of a nicotine replacement therapy, or more than one form ("patch plus" regimen).	B	4
Pregnant smokers should be offered person-to-person psychosocial interventions that exceed minimal advice to quit.	B	4
Sustained-release bupropion (Zyban) or a nicotine replacement therapy (particularly gum and lozenges) may be more appropriate for smokers who are concerned about weight gain after quitting.	C	4

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.xml.

SMOKING IN JORDAN

- Jordan is mentioned among the countries with high smoking prevalence and medium consumption (10-20 cigarettes per day per smoker)
- The prevalence of daily cigarette smoking in Jordan is very high: about 50% among men and 10% among women
- Health workers : The measured prevalence was between 60% and 39% .
- The prevalence of cigarette smoking is higher among the poor.
- Smoking cost the country 1 billion Jordanian dinars in 2012, including money spent on tobacco and smoking-related diseases
- cigarette consumption in the country increased from 5 billion cigarettes in 2003 to 8 billion cigarettes in 2013.
- Cigarette smoking is a major modifiable health risk factor ,significantly contributing to
- deaths from cancer and cardiovascular and pulmonary diseases.
- Smoking cessation is difficult, with the average smoker attempting to quit five times before permanent success. However, smoking cessation results in considerable health benefits.

- Primary care physicians have many opportunities to counsel patients about smoking cessation.
- The five A's framework (ask, advise, assess, assist, arrange) has been developed to allow physicians to incorporate smoking cessation counseling into busy clinical practices.

ASK

- Adding smoking status as a vital sign to all patients' charts increases the likelihood that physicians will address tobacco use as a risk behavior with smokers and provide them with cessation-related advice.

ADVISE

- Even brief physician advice may prompt an additional 1 to 3 percent of patients to attempt cessation and improve quit rates compared with patients who receive no advice .

ASSESS

- Patients' motivation to quit smoking should be assessed at every visit.
- Patients not yet willing to quit should receive a motivational intervention.
- Behavior change can be conceptualized into five progressive stages: precontemplation, contemplation, preparation, action, and maintenance (Table 2).
- Although tailoring interventions to a patient's stage of change may not be necessary, these stages emphasize that not all patients are equally motivated to quit smoking, motivation is malleable, and patients can be assisted toward behavior change through physician intervention.
- Confrontational interactions vs. Motivational interventions .

- Motivational interventions explore a patient's ambivalence to smoking cessation in an empathetic, questioning manner, which respects the patient's autonomy and builds self-efficacy.
- Motivational interventions, especially those in which physicians take a central role in the counseling, are more effective than brief advice and usual care in promoting smoking cessation.
- The Agency for Healthcare Research and Quality has identified several components of discussion to enhance patients' motivation to stop smoking. These components are **the five R's (relevance, risks, rewards, roadblocks, repeat)** .

Table 2. Stages of Behavior Change

Stage	Description	Comments
Precontemplation	No intention to take action within the foreseeable future (next six months)	Possibly unaware of the need to change; may overestimate the costs of change and underestimate the benefits; consider reluctance (does not want to consider change, inertia), rebellion (does not like being told what to do), resignation (overwhelmed and demoralized by the idea of change), rationalization (understands the consequences of the behavior, but denies that they apply to him or herself)
Contemplation	Considering change within the next six months	Ambivalent about change; perceives that costs equal benefits
Preparation	Planning to take action within the next month	May have already made steps toward change; often concerned about failure
Action	Actively changing (first six months of new behavior)	Needs vigilance to prevent relapse and encouragement to keep up the momentum
Maintenance	More than six months since behavior change	May benefit from reminders about high-risk situations

Information from reference 23.

Table 3. Five R's Strategy for Motivating Patients to Quit Smoking

Component	Description	Examples
Relevance	Encourage the patient to identify reasons to stop smoking that are personally relevant	Pregnancy, personal or family risk of disease, person in the household with asthma
Risks	Advise the patient of the harmful effects of continued smoking, both to the patient and to others, incorporating aspects of the personal and family history whenever possible	Effects on the patient and the patient's family, friends, and coworkers; measuring "lung age"* through spirometry can help personalize risk[26]
Rewards	Ask the patient to identify the benefits of smoking cessation	Improved health, financial savings from not buying cigarettes, decreased cigarette odor
Roadblocks	Explore the barriers to cessation that the patient may encounter	Presence of other smokers in the home or workplace, history of failed quit attempts or severe withdrawal symptoms, stress, psychiatric comorbidity, low motivation, weight gain, enjoyment of smoking
Repeat	Include aspects of the five R's in each clinical contact with unmotivated smokers	—

**—The age at which a healthy nonsmoker would perform similarly on spirometry.*

Information from references 4 and 26.

ASSIST (OR REFER)

- Asking patients who are willing to quit to set a quit date can prompt change, and physicians should help patients anticipate obstacles to cessation.
- Nicotine withdrawal symptoms, depression, and weight gain are specific areas in which patients may benefit from clinical guidance

ARRANGE FOR FOLLOW-UP

- Patients should be contacted around the time of their quit date to be congratulated on their (presumed) abstinence.
- Contacting patients at least four more times to support their smoking-cessation attempts increases abstinence rates.
- Patients who are unable to quit or who relapse should be reassessed.
- Pharmacologic therapies and additional behavioral counseling should be considered, and patients should be encouraged to set a new quit date.

MEDICATIONS

NICOTINE REPLACEMENT THERAPIES

- ✓ The goal of nicotine replacement therapies (NRTs) is to relieve cravings for nicotine and reduce nicotine withdrawal symptoms.
- ✓ NRTs are available as slow release skin patches and in more rapidly acting forms (i.e., chewing gum, nasal spray, inhalers, and lozenges), which deliver nicotine to the brain more quickly than skin patches but more slowly than smoking cigarettes.
- ✓ A Cochrane review of 132 trials concluded that all forms of NRTs increase the chances of quitting successfully by 50 to 70 percent.
- ✓ Heavy smokers should be encouraged to use higher dosages of an NRT or try a "patch plus" method, using the nicotine patch to provide a base level of slowly delivered nicotine and adding a more rapidly acting NRT to control breakthrough cravings. This regimen is safe because smokers typically obtain less nicotine than through smoking, and it is more effective than using a single NRT.

NICOTINIC RECEPTOR AGONIST

- ✓ Varenicline (Chantix) is a selective alpha4-beta2 nicotinic receptor partial agonist that reduces cravings and withdrawal symptoms while blocking the binding of smoked nicotine.
- ✓ Varenicline increases the chances of a successful quit attempt two- to threefold compared with no pharmacologic assistance.
- ✓ In a direct comparison, varenicline was superior to bupropion in promoting abstinence. However, new data suggest increased risk of coronary events with varenicline

BUPROPION

- ✓ Initially developed as an antidepressant, bupropion has been shown to be effective as a smoking cessation aid.
- ✓ A Cochrane review of 19 randomized trials showed that bupropion doubles the odds of smoking cessation compared with placebo.

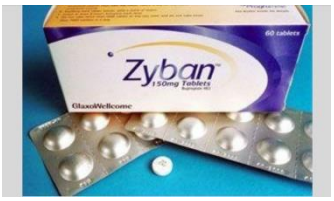


Table 5. Complementary and Alternative Therapies to Assist in Smoking Cessation

Therapy	*Comments*
Acupuncture	Acupuncture, acupressure, laser acupuncture, and electroacupuncture all have been proposed as cures for nicotine addiction or as a means to reduce withdrawal symptoms A Cochrane review did not find consistent evidence that these therapies are more beneficial for smoking cessation than no treatment or sham acupuncture[33]
Exercise	Small, heterogeneous studies provide little evidence that exercise improves quit rates[34]; however, it may be useful to promote weight control after smoking cessation[35]
Hypnotherapy	A Cochrane review found a lack of evidence that hypnotherapy is beneficial compared with other therapies or no treatment[36]
Internet-based interventions (e.g., http://www.smokefree.gov)	A Cochrane review of 20 randomized and quasi-randomized studies did not find conclusive evidence of benefit, especially at long-term follow-up[37]; Web sites providing personally tailored information and repeated automated contacts may be beneficial as an adjunct to other cessation strategies[4]
Telephone quitlines (e.g., 800-QUIT-NOW)	Physician encouragement to use quitlines has a two to three times greater effect on smoking cessation than counseling alone in the primary care setting[38]; the use of quitlines results in a relative risk of cessation of 1.37[39]

Information from references 4, and 33 through 39.

SPECIAL POPULATIONS

PREGNANT WOMEN

- Tobacco is a known teratogen that can harm a developing fetus.
- Smoking cessation therapies also carry risks in pregnant women.
- Pregnant smokers should be offered intensive person-to-person interventions that exceed minimal advice to quit, such as behavioral support and problem solving, counseling, and referral to support organizations

Women who quit smoking before pregnancy or in early pregnancy significantly reduce the risk of preterm birth, low birth weight, and infant mortality.

PERSONS WITH CORONARY HEART DISEASE

- Smoking cessation reduces the risk of coronary heart disease.
- The success rate of bupropion in patients with established cardiovascular disease is similar to that in healthy smokers and the drug is recommended for smoking cessation in patients with cardiovascular disease. However, bupropion is cardiotoxic in overdose and should be considered as a cause of unexplained widening of the QRS complex on electrocardiography.
- Although varenicline improves one-year abstinence rates over placebo in smokers with cardiovascular disease, the U.S. Food and Drug Administration recently released a warning about a possible increase in cardiovascular risk with the drug.
- Uncommon: increased rates of angina pectoris, nonfatal myocardial infarction, need for coronary revascularization, and peripheral vascular disease have been reported in patients taking varenicline.
- Data indicate that the benefits of NRTs for smoking cessation outweigh the risks of the therapies.
- Physicians may consider a lower initial dosage, more frequent monitoring of side effects, and alternative therapies, such as behavioral interventions.

Table 4. First-Line Therapies for Smoking Cessation in Adults

Therapy	*Dosage*	*Comments*	*Cost**
Nicotine gum† (Nicorette)	Available in 2-mg and 4-mg (per piece) doses Patients smoking less than 25 cigarettes per day: 2 mg Patients smoking 25 or more cigarettes per day: 4 mg Maximum dosage: 24 pieces per day	Over the counter Intermittently chew, then "park" between gum and cheek for maximum benefit; eating or drinking acidic foods or beverages within 30 minutes of use decreases effectiveness; may delay weight gain; difficult to use with dentures, partials, or fillings FDA pregnancy category C Side effects: Gastrointestinal distress; mouth or throat irritation	$40 ($52) for 100 pieces
Nicotine inhaler† (Nicotrol)	One dose consists of one inhalation Recommended dosage is six to 16 cartridges per day; each cartridge delivers 4 mg of nicotine over 80 inhalations	Prescription Eating or drinking acidic foods or beverages within 30 minutes of use decreases effectiveness FDA pregnancy category D Side effects: Mouth or throat irritation (40 percent), coughing (32 percent), rhinitis (23 percent)	NA ($213) for 168 10-mg cartridges
Nicotine lozenge† (Nicorette)	Heavy smokers: 4 mg Light smokers: 2 mg Maximum: 20 lozenges per day	Over the counter May delay weight gain; should be taken one at a time and dissolved in the mouth, not chewed or swallowed; eating or drinking acidic foods or beverages within 30 minutes of use decreases effectiveness; contains 25 percent more nicotine than gum FDA pregnancy category D Side effects: Nausea, heartburn, headache	$47 ($58) for 108 lozenges
Nicotine patch† (Nicoderm CQ [24-hour patch], Nicotrol [16-hour patch; not available in the United States])	Doses vary and should be tapered as therapy progresses Heavy smokers: 21 mg per day (initial dosage) Light smokers or those weighing less than 100 lb (45 kg): 10 to 14 mg per day (initial dosage)	Over the counter Treatment of up to eight weeks has been shown to be as effective as longer treatments; site of patch should be changed daily; 16- and 24-hour patches have comparable effectiveness; adolescents may require lower starting dosages because of body habitus and overall smoking patterns (e.g., less than one-half pack per day) FDA pregnancy category D Side effects: Skin reactions (up to 50 percent), headaches, insomnia (decreased if patient removes patch at night)	24-hour patch: $32 ($54) for 14 patches
Nasal spray† (Nicotrol NS)	One dose consists of two 0.5-mg sprays (one in each nostril) Initial dosage is one or two doses per hour (minimum of eight doses per day), increasing as needed for symptom relief Maximum: 40 doses per day (five doses per hour)	Prescription Dependence potential is intermediate between other nicotine replacement therapies and cigarettes FDA pregnancy category D Side effects: Moderate to severe nasal irritation within the first two days (94 percent) that often continues throughout use	NA ($207) for 40 mL
Bupropion, sustained release (Zyban)	150 mg in the morning for three days, then increased to 150 mg twice per day Begin therapy one to two weeks before the quit date, continue until 12 weeks to six months after the quit date	Prescription Can be combined with a nicotine replacement therapy for increased effectiveness; may be beneficial for patients with a history of depression; insufficient evidence to be a first-line therapy for adolescents FDA pregnancy category C Side effects: Insomnia (35 to 40 percent), dry mouth (10 percent) Contraindicated in persons with a history of seizure disorder or an eating disorder, and in those who have used a monoamine oxidase inhibitor in the past 14 days FDA boxed warning: May increase suicidality in patients with depression	$106 ($210) for 60 tablets
Varenicline (Chantix)	Days 1 to 3: 0.5 mg once per day Days 4 to 7: 0.5 mg twice per day Day 8 to end of treatment: 1 mg twice per day Begin therapy one week before quit date and continue for 12 weeks; an additional 12 weeks can be added if quit attempt is successful to increase chances of long-term abstinence	Prescription Should not be combined with a nicotine replacement therapy; the safety of combining varenicline and bupropion has not been established; insufficient evidence to be a first-line therapy for adolescents FDA pregnancy category C Side effects: Headache, nausea (dose related), insomnia, abnormal dreams, flatulence Increased risk of cardiovascular events in smokers with cardiovascular disease should be discussed with patients[30] FDA boxed warning: May cause serious neuropsychiatric symptoms in patients, including changes in behavior, hostility, agitation, depressed mood, suicidal thoughts and behavior, and attempted suicide; patient should be monitored closely	NA ($191) for 60 tablets

FDA = U.S. Food and Drug Administration; NA = not available.

**—Estimated retail price based on information obtained at http://www.drugstore.com (accessed December 6, 2011). Generic prices listed first, brand prices listed in parentheses.*

†—Nicotine replacement therapies should be used with caution in the immediate (within two weeks) postmyocardial infarction period, in patients with serious cardiac arrhythmias, and in patients with serious or worsening angina pectoris.

Information from references 4 and 30.

COMMUNICATION SKILLS

- Doctor-patient communication is not limited to the extraction of the history from the patient.
- Doctors are expected to take the patient as a whole and attend to their needs, fears and concerns during the consultation, adopting a patient-centred attitude.
- The role of the physician communicator is redefined; the ability of a doctor to provide comfort through their presence and their words is considered to be a fundamental component of good medical care.

THE ART OF COMMUNICATION

- Effective communication is an essential skill in general practice consultations.
- Patients frequently experience psychological distress and require empathy and highly skilled communication.
- The technical aspects of good communication can be known but the art of communication is the development of these skills and finding a style of communication that suits the clinician and produces benefits for the patient and doctor.
- In Australia : statistics reveal that almost two-thirds of the problems managed were done so without pharmacological treatment.
- The two most frequent clinical treatments in consultations are advice and counselling.
- Mental health consultations are common.
- Depression and anxiety feature in the top 10 most common problems managed with clinical treatments, such as advice and counselling

BENEFITS OF EFFECTIVE COMMUNICATION

For patients

1. Increased patient and doctor satisfaction
2. Better adherence to treatment
3. Improved follow up
4. Decreased litigation

Evidence can give us the knowledge that exercise is at least as beneficial as antidepressants in treating depression. However, it will be the clinician's skill in communicating this knowledge and motivating the patient to exercise that will produce the benefit.

The skills and knowledge to diagnose diabetes and its complications can be highly developed, but without the communication skills that take a patient-centred approach to understand what this illness means to the patient, there is likely to be little understanding of the patient beyond knowing they have a disease.

5. Reduced morbidity
6. Improved quality of life for patients

For doctors

7. A study of primary care physicians found that undertaking an intensive education program in mindful communication improved patient outcomes and personal well-being for the doctors.
8. The medical literature also provides reassuring evidence that an effective patient-centered model does not take more time.

Good communication can improve outcomes for patients and doctors and deserves equal importance as developing clinical knowledge and procedural skill .

COMPONENTS OF EFFECTIVE COMMUNICATION

1. Listening to patients
2. Respecting their views
3. Encouraging open communication about treatment preferences
4. Discussing management options
5. Allowing patients to ask questions
6. Ensuring understanding
7. Being aware of language, cultural and communication issues .

THE SKILLS REQUIRED FOR GOOD COMMUNICATION

- Every doctor can know the guidelines but developing and incorporating these skills into their consultations is an ongoing journey and a lifelong process of refining this art.
- verbal and non-verbal communication skills .

- NON VERBAL :

1. Open, attentive posture
2. eye contact
3. **active Listening** and encouraging the patient to talk using cues such as nodding

- VERBAL :

1. beginning the consultation by encouraging the patient to tell their story without interruption .
2. asking further, open-ended questions .

- Summarizing and Reflecting what has been heard is important to ensure a shared understanding.

-Empathy

-Consideration of the patient's perspective

-Development of trust and ensuring understanding of the patient's story and psychosocial factors of health .

- We are far more influenced by what we see than by what we hear
- 93% of all communication is non-verbal (38% tone of voice & 55% body language) & 7% words

ESTABLISHING RELATIONSHIP WITH PATIENT

The opening of the consultation is the key to putting patient at ease. Factors enhancing relationship:

1) Setting of consultation including accessibility, waiting time…..
2) Appearance of doctor
3) Greeting Pt. by name, rising to meet pt., shaking hand, show where to sit & engage in most preliminary informal chat
4) Demonstrate interest (for patient as a whole and his problem)
5) Encourage open communication
6) We are far more influenced by what we see than by what we hear
7) 93% of all communication is non-verbal (38% tone of voice & 55% body language) & 7% words

DEALING WITH ANGER

1. Consider avoiding confrontation
2. Facilitate discussion
3. Ventilate feelings
4. Explore reasons
5. Refer / Investigate

DEALING WITH CONFLICT OR ETHICALLY REQUEST

1. Consider whether to agree or disagree
2. Discuss with partners
3. Refer
4. Defer
5. Bargain / Negotiate
6. Educate
7. Counsel

DEALING WITH GRIEF

1. Consider facilitating expression
2. Help Pt. understanding of reaction
3. Educate what to expect
4. Identify problems and offer solutions
5. Inspire hope but not false one
6. Identify positive achievement

COMMUNICATION WHEN THINGS GO WRONG

- Acknowledge your mistake to the patient or family.
  -This is what patients want, and it has the likelihood of decreasing the risk of litigation.
  -acknowledgement does something for the physician. Confession, which has been practiced throughout history as part of many religions, is a vital component of learning from our mistakes and beginning the process of healing.
  -The opposite approach, denying or defending our mistakes and our feelings, is destructive not only for the patient but also for ourselves.
- Be prepared to say SORRY.
- Full and proper communication with patient is important.

OBESITY

In 2012, the U.S. Preventive Services Task Force (USPSTF) issued the recommendation that all adults be screened for obesity, and that patients with a body mass index (BMI) of 30 kg/m2 or greater be offered intensive, multicomponent behavioral interventions.

The prevalence of obesity exceeds 30% in adults and is associated with increased risk of such serious health problems as cardiovascular disease, type 2 diabetes, and various types of cancer.

THE LEADING CAUSES OF DEATH IN OBESE ADULTS:

- ✓ ***ischemic heart disease***
- ✓ ***diabetes***
- ✓ ***respiratory diseases***
- ✓ ***cancer (i.e., liver, kidney, breast, endometrial, prostate, and colon).***

BRIDGING THE GAP

Many factors complicate efforts to address overweight, obesity, and the promotion of healthier diets and lifestyles

- ✓ ***Insufficient time during visits for screening and counseling***
- ✓ ***Lack of available referral services for patients***
- ✓ ***Perception that patients will not be willing or able to make lifestyle changes***
- ✓ ***Poor reimbursement for nutrition and weight-management counseling***
- ✓ ***Reluctance to discuss weight among physicians who are themselves overweight***
- ✓ ***Uncertainty about whether interventions will have a positive impact***

Overweight and obesity are chronic diseases with behavioral origins that can be traced back to childhood. Because family physicians see patients of all ages and often care for entire families, they are well positioned to help turn the tide on the obesity epidemic

Overweight: is defined as a body mass index (BMI) in the 25 to 29 kg/m2 range, whereas obesity is a BMI in excess of 30 kg/m2.

Although BMI correlates with the amount of body fat, it must be recognized that BMI does not **directly** measure body fat, nor does it **differentiate** fat from muscle.

Nevertheless, BMI is recommended for use in clinical practice as a practical way to identify individuals who are overweight or obese. Furthermore, calculating BMI is still a good way to evaluate changes over time, because incremental increases most likely represent gains in body fat.

Recognizing that BMI is just one indicator of potential health risks associated with being overweight or obese, the National Heart, Lung and Blood Institute (NHLBI) recommends that physicians also look at the following factors:

1) Risk factors for diseases associated with obesity, such as high blood pressure and physical inactivity

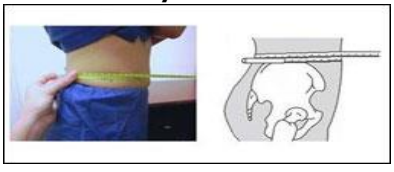
2) Waist circumference as a measure of abdominal adiposity

WAIST CIRCUMFERENCE

Abdominal adiposity is an important independent risk factor for cardiovascular disease, type 2 diabetes, dyslipidemia, and hypertension.

The NHLBI defines abdominal obesity as:

- ✓ *Waist circumference greater than **40 in (102 cm) in men***
- ✓ *Waist circumference greater than **35 in (88 cm) in women***



Individuals with larger waist circumferences have more than a fivefold greater risk of multiple cardiometabolic risk factors, even after adjusting for BMI, compared with individuals with waist measurements in the normal range

Abdominal obesity is also one of five diagnostic criteria for metabolic syndrome

Additional Evaluation

Most cases of obesity are not due to a medical disorder, but rather to a combination of hereditary predisposition and lifestyle factors. Nevertheless, the initial evaluation should include a review of the medication list and a thorough medical history, including age at onset of weight gain, previous weight-loss efforts, dietary and exercise habits, and history of smoking

Medications That Promote Weight Gain

Assessment of the obese patient should include a complete medication history. Many agents, including beta blockers, corticosteroids, diabetes drugs, and psychoactive drugs, are known to cause weight gain. Most of these medications cause weight gain by increasing appetite. Prescribing these medications may be unavoidable, but patients should be told that weight gain is a side effect and encouraged to take steps to prevent it (e.g., increase physical activity).

Anticonvulsants	Antihypertensives	Antipsychotics	Corticosteroids
Valproic acid	Clonidine	Chlorpromazine	**Psychotropics**
Carbamazepine	Guanabenz	Thiothixene	Lithium
Antidepressants	Methyldopa	Haloperidol	**Sulfonylureas**
Amitriptyline	Prazosin	Olanzapine	Glipizide
Imipramine	Terazosin	Clozapine	Glyburide
Phenelzine	Propranolol	Risperidone	
	Nisoldipine	Quetiapine	

Adapted from Kolasa KM, Collier DN, Cable K. Weight loss strategies that really work. J Fam Pract. *2010;59(7):378-385.*

METABOLIC SYNDROME

Metabolic syndrome is a **constellation** of risk factors

The predominant underlying risk factors for metabolic syndrome are **abdominal obesity** and **insulin resistance**.

Informing a patient that he or she has metabolic syndrome can generate a valuable counseling opportunity

APPROACH TO MANAGEMENT

Although significant weight loss may be ideal, even a modest reduction in weight (5% to 10% of total body weight) can have significant health benefits.

Diabetes Prevention Program (DPP), a rigorously conducted randomized trial that compared usual care, metformin use (850 mg two times per day), and intensive lifestyle modification in more than 3,000 individuals with impaired glucose tolerance. In the trial, intensive lifestyle modification decreased progression to diabetes by nearly 60% while metformin resulted in a 31% decrease, compared with usual care

BEHAVIORAL TREATMENT

The goal of behavioral therapy is to enable patients to reduce and manage their weight by monitoring and modifying their food intake, increasing their physical activity level, and recognizing and controlling cues that

Table 3.
Diagnostic Criteria for Metabolic Syndrome*

Measure (any 3 of 5 criteria constitute diagnosis of metabolic syndrome)	Categorical Cut Points
Elevated waist circumference	>102 cm (>40 in) in men >88 cm (>35 in) in women
Elevated TG	>150 mg/dL (1.7 mmol/L) or drug treatment for elevated TG
Reduced HDL-C	<40 mg/dL (1.03 mmol/L) in men <50 mg/dL (1.3 mmol/L) in women or drug treatment for reduced HDL-C
Elevated BP	>130 mm Hg systolic >85 mm Hg diastolic or drug treatment for hypertension
Elevated fasting glucose (or treatment for elevated fasting glucose)	>100 mg/dL (5.6 mmol/L) or drug treatment for elevated glucose

BP = blood pressure; HDL-C = high-density lipoprotein cholesterol; TG = triglycerides.

**Three of the criteria must be present to make the diagnosis.*

Reprinted with permission from Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome. Circulation. *2005; 112(17):2735-2752.*

trigger overeating.

Self-Monitoring

Stimulus Control

Table 4.
The 5 A's for Evaluation and Treatment of Obesity

Assess	Severity of obesity with calculated BMI, waist circumference, and comorbidities
	Food intake and physical activity in context of health risks and appropriate dietary approach
	Medications that affect weight or satiety
	Readiness to change behavior and stage of change
Advise	Diagnosis of overweight, obese, or severe obesity
	Caloric deficit needed for weight loss
	Various types of diets that lead to weight loss and ease of adherence
	Appropriateness, cost, and effectiveness of meal replacements, dietary supplements, over-the-counter weight aids, medications, surgery
	Importance of self-monitoring
Agree	If patient is not ready, discuss at another visit
	If patient is motivated and ready to change, develop treatment plan
	If patient chooses diet, physical activity, and/or medication, set weight-loss goal at 10% from baseline
	If patient is a potential candidate for surgery, review options
Assist	Provide a diet plan, physical activity guide, and behavior-modification guide
	Provide Web resources based on patient interest and need
	Identify method for self-monitoring (e.g., diary)
	Review food and activity diary on follow-up (reassess if initial goal is not met)
Arrange	Follow-up appointments to meet patient needs
	Referral to registered dietitian and/or behavioral specialist for individual counseling/monitoring or weight-management class
	Referral to surgical program
	Maintenance counseling to prevent relapse or weight regain

BMI = body mass index.

Reprinted with permission from Kolasa KM, Collier DN, Caleb K. Weight loss strategies that really work. J Fam Pract. 2010;59(7):378-385.

Table 2. Motivational Interviewing Techniques

Technique	*Example*	*Rationale*
Ask permission to discuss behavior-change topic	"Would it be okay if we talked about your weight today?"	When patient gives permission, he or she is more open to the conversation
Show empathy	"Losing weight is very challenging."	Aids in building rapport, particularly in difficult discussions
Scale motivation (0 = low to 10 = high)	"On a scale of 0 to 10, with 10 being the highest, how motivated are you to try to lose weight?"	Assesses motivation to change; if very low, the patient may not be ready for change; if high, additional intervention strategies may be successful
Scale confidence (0 = low to 10 = high)	"On a scale of 0 to 10, with 10 being the highest, how confident are you that you can lose weight?"	Identifies need for interventions to overcome obstacles
Inquire about the scores on above scales	"Why did you choose 3 instead of 2? What would help you move from 3 to 4?"	Furthers the conversation on thinking about behavior change
Use decisional balance technique (explore pros and cons of change vs. no change)	"What are the pros of losing weight?" "What are the pros of not losing weight?" "What are the cons of losing weight?" "What are the cons of not losing weight?"	Helps patient and physician understand barriers to and motivators for change
Listen for change talk and reinforce it; let the patient take ownership by generating ideas for change	Patient: "I think I could try to walk more." Physician: "That's a fantastic idea that will help you move toward your goal."	Provides encouragement and helps promote confidence in patients

Information from references 11 and 12.

NUTRITION COUNSELING :

National Heart, Lung and Blood Institute guidelines suggest that patients who want to lose weight reduce their caloric intake by 500 to 1,000 kcal per day to produce a weight loss of (0.45 to 0.90 kg) per week

PHYSICAL ACTIVITY:

- Physicians should routinely recommend regular physical activity to all patients, not only to those who are overweight or obese
- The 2008 Physical Activity Guidelines for Americans recommend that adults perform at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity aerobic activity per week (or an equivalent combination of these).
- Aerobic activity should be performed for at least 10 minutes per session and should be spread throughout the week.
- Adding physical activity to calorie restriction may result in modest improvements in weight loss.4 Physical activity alone, however, has not been shown to be sufficient in producing significant weight loss, except at very high intensity levels.
- even without weight loss, however, exercise can mitigate the damaging effects of obesity and a sedentary lifestyle. Increasingly, "**sitting time**" is being recognized as an independent risk factor for the development of metabolic risk factors.
- Physical exercise and activity are particularly important for maintaining weight loss over the long term (and for preserving lean body mass during dieting).

Maintenance of weight loss has a graded relationship to the amount of exercise that individuals need after weight loss. Thus, patients who have lost considerable weight may need to engage in higher amounts (more than 300 minutes a week) or more vigorous exercise to maintain their weight loss.

PHARMACOTHERAPY

Prescription anti-obesity drugs can be useful adjuncts to diet and exercise for obese adults who have failed to achieve weight loss with diet and exercise. Prescription weight-loss drugs are approved for patients who meet the following criteria:

- **BMI of 30 kg/m2 or greater**
- **BMI of 27 kg/m2 or greater and an obesity-related condition (such as hypertension, type 2 diabetes, or dyslipidemia)**

Table 6. Anti-obesity Medications Approved for Long-term Use

Drug	Mechanism of Action	Possible Adverse Effects
Lorcaserin (Belviq)	Decreases appetite, increases feeling of fullness	Headache, dizziness, fatigue, nausea, dry mouth, constipation
Orlistat (Xenical)	Blocks absorption of fat	Intestinal cramps, gas, diarrhea, oily spotting
Phentermine and topiramate extended-release (Qsymia)	Decreases appetite, increases feeling of fullness	Increased heart rate, birth defects, tingling of hands and feet, insomnia, dizziness, constipation, dry mouth

Adapted from Prescription medications for the treatment of obesity. win.niddk.nih.gov/publications/prescription.htm; Accessed April 17, 2013. Bray GA. Drug therapy of obesity. www.UpToDate.com. Accessed March 1, 2013; Allison DB, Gadde KM, Garvey WT, et al. Controlled-release phentermine/topiramate in severely obese adults. Obesity. *2012;20(2):330-342.*

4) liraglutide (victosa) is glucoge like peptide 1 receptor agonist, contraindicates in personal or family history of medullary thyroid cancer or multiple endocrine neoplasia type 2 , side effects diarrhea, headache, hypoglycemia dizziness and constipation

5) naltrexone/bupropion : contraindication seizure, uncontrolled htn abrupt discontinuation of alcohol, barbiturates or antiepileptic, anorexia nerviousa or bulimia

BARIATRIC SURGERY

Bariatric surgery may be considered in adults who have not achieved weight loss with dietary or other treatments and who have a BMI of **40 kg/m2** or greater, or for those who have a BMI of **35** kg/m2 or greater with significant obesity-related comorbidities (e.g., severe hypertension, type 2 diabetes, obstructive sleep apnea).

Bariatric surgery may also benefit patients with obesity-related comorbidities who have a BMI of 35 kg/m2 or lower, but it is not routinely recommended for these patients.

Multiple studies have demonstrated that bariatric surgery produces substantial and sustained weight loss, and results in amelioration of obesity-related comorbidities, compared with usual care. Bariatric surgery also appears to improve long-term survival. Perhaps just as important, bariatric surgery has the potential to dramatically improve a patient's quality of life.

Numerous bariatric procedures are in use

Restrictive procedures limit the size of the stomach. examples include laparoscopic adjustable gastric banding and vertical sleeve gastrectomy.

Malabsorptive procedures restrict the size of the stomach to some extent but also involve bypassing a portion of the small intestine. Roux-en-Y gastric bypass is an example of this type of procedure.

it is essential to emphasize that bariatric surgery is not a magic bullet. Following surgery, a significant number of patients fail to achieve optimal weight loss and/or regain weight. Some studies suggest that these results occur, at least in part, because patients return to or develop problematic dietary patterns.

Sustained changes in diet and exercise habits are essential following bariatric surgery.

OVERWEIGHT AND OBESITY IN CHILDREN

(BMI at or above the 95th percentile for age and sex). Childhood obesity causes health problems, such as elevated cholesterol and blood pressure levels, as well as socialpsychologic difficulties for children. It also predisposes children to obesity and significant morbidity in adulthood. The USPSTF and the American Academy of Family Physicians (AAFP) recommend that physicians screen children ages 6 years or older for obesity and offer comprehensive, intensive behavioral interventions to promote improvement in weight status. The American Academy of Pediatrics (AAP) recommends that BMI be calculated and plotted annually.

REFERENCES

1) The 5 minute clinical consult 2019

2) JNC 8 Hypertension Guideline

3) Ambulatory Best Practice Group Recommended Screening Guidelines for Adults

4) ADA 2019 / Abridged ADA guidelines for primary care providers 2019

5) 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease

6) GINA guidelines

7) Murtagh's general practice 7e

8) American Academy of Family Physicians (AAFP)

9) Medscape

10) CDC/ Recommended Adult Immunization Schedule for ages 19 years or older

11) Oscestop

12) Kaplan internal medicine 2019