Subtle Signs and Symptoms of Illness and Injury

Developmental Disabilities Support Division

Resource Packet F

Injuries

Falls, Pain, Seizures, and Dementia

Required for:
RN, LPN, SLP, PT, OT, BSC,
and
Optional for RD/LD/LN and Other

NEW MEXICO
Department of Health
Developmental Disabilities Supports Division

2021

Points To Remember About Osteoporosis

- Osteoporosis is a disease that causes bones to become weak and brittle. This increases your risk of broken bones (fractures).
- Osteoporosis is a “silent” disease because you may not have symptoms. You may not even know you have the disease until you break a bone.
- You can take steps to help prevent osteoporosis and broken bones by doing weight-bearing exercises or lifting weights, eating a well-balanced diet rich in calcium and vitamin D, not drinking too much alcohol, not smoking, and taking your medications, if prescribed.
- Take steps to reduce your risk of falling, which can cause broken bones. Keep your floors free of clutter, wear shoes with nonslip soles, be careful on icy, wet, or polished surfaces, and use a cane or walker to keep you stable and provide support.

What is osteoporosis?

Osteoporosis is a disease that causes bones to become weak and brittle. This increases your risk of broken bones (fractures).

Osteoporosis is a “silent” disease because you may not have symptoms. You may not even know you have the disease until you break a bone. Breaks can occur in any bone but happen most often in:

- Hip bones.
- Vertebrae in the spine.
- Wrist.

You can take steps to help prevent osteoporosis and broken bones by:

- Doing weight-bearing exercises, such as walking or dancing, and lifting weights.
- Not drinking too much alcohol.
- Quitting smoking, or not starting if you don’t smoke.
- Taking your medications, if prescribed.
- Eating a well-balanced diet rich in calcium and vitamin D.

Who gets osteoporosis?

Osteoporosis affects women and men of all races and ethnic groups. Osteoporosis can occur at any age, although you are at greater risk as you get older. For many women, the disease begins to develop a year or two before menopause.

- Osteoporosis is most common in non-Hispanic white women and Asian women.
- African American and Hispanic women have a lower risk of developing osteoporosis, but they are still at significant risk.
- Among men, osteoporosis is more common in non-Hispanic whites.

Because women get osteoporosis more than men, many men think they won’t get the disease. But older men and women are at risk for the disease.

What are the symptoms of osteoporosis?

Osteoporosis is called a “silent” disease” because you typically have no symptoms until a bone breaks or one or more vertebrae in the spine collapse. Symptoms of broken vertebrae include severe back pain, loss of height, or a stooped or hunched posture.

Bones affected by osteoporosis may break very easily or as the result of:

- Minor falls that would not normally cause a break in a healthy bone.
- Normal stresses such as bending, lifting, or even coughing.

What causes osteoporosis?

Osteoporosis happens when new bone tissue is not made as fast as old bone tissue is lost. When this happens, too much bone is lost and the bones become weak.

Certain factors may make you more at risk of developing the disease. There are some factors that you cannot change, and others that you may be able to change. Factors that may increase your risk for osteoporosis include:

- **Sex.** If you are a woman, you are at greater risk for developing osteoporosis. However, men are still at risk, especially after the age of 70.

- **Age.** As you age, your bones can get weaker.
- **Body size.** Slender, thin-boned women and men are at greater risk to develop osteoporosis.
- **Race.** White and Asian women are at highest risk. African American and Mexican American women have a lower risk.
- **Family history.** Your risk for osteoporosis and broken bones may increase if one of your parents has a history of osteoporosis or hip fracture.
- **Changes to hormones.** Low levels of certain hormones can increase your chances of developing osteoporosis.
- **Diet.** A diet low in calcium and vitamin D can increase your risk for osteoporosis and broken bones. Dieting too much or getting too little protein may also increase your risk for bone loss and osteoporosis.
- **Other medical conditions.** Some medical conditions can increase the risk of osteoporosis.
- **Medications.** Long-term use of certain medications may make you more likely to develop bone loss and osteoporosis.
- **Lifestyle.** A healthy lifestyle can be important for keeping bones strong. Lifestyle changes that may cause bone loss include:
  - Not getting enough exercise and being inactive for long periods of time.
  - Long-term heavy drinking of alcohol.
  - Smoking.

Is there a test for osteoporosis?

If you are a woman over age 65 or if you are a woman of any age who has factors that increase the chance of developing osteoporosis, your doctor likely will screen you for osteoporosis.

Younger women and older men should talk to their doctor to find out if they have risk factors for osteoporosis.

During your visit with your doctor, remember to talk about:

- Any broken bones you have had.
- Your diet, exercise, alcohol use, and smoking history.
- Current or past medical conditions and medications that could lead to low bone mass and increased risk of broken bones.
- Your family history of osteoporosis and other diseases.
- For women, your menstrual history.

The doctor may also perform a physical exam that includes checking for:

- Loss of height and weight.
- Changes in posture.
- Balance and the way you walk.
- How strong your muscles are, such as if you can stand from sitting without using your arms.

In addition, your doctor may order a test that measures your bone mineral density in an area of your bone. This test measures how much calcium and other minerals are in a specific area of your bone, usually your spine and hip.

How is osteoporosis treated?

The goals for treating osteoporosis are to slow or stop bone loss and to prevent broken bones. Your doctor may recommend:

- Eating a healthy diet.
- Exercising.
- Making healthy choices about smoking and alcohol.
- Working to prevent falls to help prevent fractures.
- Taking medications.

If you develop osteoporosis from another condition, work with your doctor to treat the underlying cause.

Nutrition

An important part of treating osteoporosis is eating a healthy, balanced diet, which includes:

- Plenty of fruits and vegetables.
- An appropriate amount of calories for your age, height, and weight. Talk to your doctor about the amount of calories you need each day to keep a healthy weight.
- Foods and liquids that include calcium, vitamin D, and protein. Good sources of calcium include:

- Low-fat dairy products.
- Dark green leafy vegetables, such as bok choy, collards, and turnip greens.
- Broccoli.
- Sardines and salmon with bones.
- Calcium-fortified foods such as soymilk, tofu, orange juice, cereals, and breads.

Vitamin D helps your body absorb calcium. Some foods naturally contain enough vitamin D, including fatty fish, fish oils, egg yolks, and liver. Other foods have added vitamin D, including milk and cereals.

Lifestyle

You can make some choices to keep your bones healthy:

- Avoid secondhand smoke, and if you smoke, quit.
- If you drink alcohol, only have one drink a day if you're a woman and two drinks a day if you're a man.
- Visit your doctor for regular checkups and ask about anything that may affect your bone health or increase your chance of falling, such as medications or other medical conditions.

Exercise

Exercise is an important part of an osteoporosis treatment program. During childhood and adulthood, exercises such as walking, dancing, or weight lifting can make bones stronger. For older adults, regular exercise can help:

- Keep muscles strong and improve coordination and balance. This can help lower your chance of falling.
- Keep your independence.
- Manage your daily living tasks.

If you have osteoporosis, you should not do high-impact exercise. Work with a physical therapist or rehabilitation medicine specialist to figure out an exercise program that works for you.

Medications

Your doctor may prescribe medications for osteoporosis. Your doctor will discuss the best option for you, thinking about your age, sex, general health, and the amount of bone you have lost.

Who treats osteoporosis?

Health care providers who treat osteoporosis include:

- Endocrinologists, who treat problems related to the glands and hormones.
- Geriatricians, who specialize in caring for all aspects of health in older people.
- Gynecologists, who specialize in diagnosing and treating conditions of the reproductive system of women.
- Nurse educators, who specialize in helping people understand their overall condition and set up their treatment plans.

- Occupational therapists, who teach ways to protect joints, minimize pain, perform activities of daily living, and conserve energy.
- Orthopaedists, who specialize in the treatment of and surgery for bone and joint diseases or injuries.
- Physiatrists (doctors specializing in physical medicine and rehabilitation).
- Physical therapists, who help to improve joint function.
- Primary care providers, such as a family physician or internal medicine specialist.
- Rheumatologists, who specialize in arthritis and other diseases of the bones, joints, and muscles.

Living with osteoporosis

In addition to the treatments your doctor recommends, the following tips can help you manage osteoporosis, prevent broken bones, and prevent falls.

Broken bones can cause other medical problems and take away your independence. Falls increase your chances of breaking a bone in the hip, wrist, spine, or other bone. Taking steps to prevent falls both inside and outside of the house can help prevent broken bones. It is important to tell your doctor if you have had problems with falling.

Here are some tips to help prevent falls outdoors and when you are away from home:

- Use a cane or walker to keep you stable and provide support.
- Wear shoes that provide support and have thin nonslip soles.
- Walk on grass when sidewalks are slippery.
- Stop at curbs and check their height before stepping up or down.

Some ways to help prevent falls indoors are:

- Keep rooms free of clutter, especially on floors.
- Use plastic or carpet runners on slippery floors.
- Wear shoes, even when indoors.
- If you have a pet, be aware of where they are to avoid tripping over them.
- Do not walk in socks, stockings, or slippers.
- Be careful on highly polished floors that are slick.
- Be sure carpets and area rugs have skid-proof backing or are tacked to the floor.
- Be sure stairs are well lit and have rails on both sides.
- Install grab bars on bathroom walls near the tub, shower, and toilet.
- Use a rubber bathmat or slip-proof seat in the shower or tub.
- Keep your home well lit, and use a nightlight.

- Use a sturdy stepstool with a handrail and wide steps.
- Keep a cordless phone or cell phone with you.
- Consider having a personal emergency-response system; you can use it to call for help if you fall.

Other tips that can help you manage your osteoporosis include:

- Talking with other people who have osteoporosis.
- Reaching out to family and friends for support.
- Learning about the disease and treatments to help you make decisions about your care.

For more info

U.S. Food and Drug Administration

Toll free: 888-INFO-FDA (888-463-6332)
Website: https://www.fda.gov
Drugs@FDA is a searchable catalog of FDA-approved drug products.

NIH Osteoporosis and Related Bone Diseases
National Resource Center
A Service Provided by the National Institutes of Health

Preventing Falls and Related Fractures

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Bone Diseases
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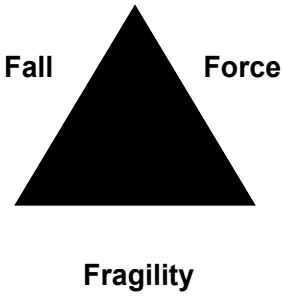
Falls are serious at any age, and breaking a bone after a fall becomes more likely as a person ages. Many of us know someone who has fallen and broken a bone. While healing, the fracture limits the person's activities and sometimes requires surgery. Often, the person wears a heavy cast to support the broken bone and needs physical therapy to resume normal activities. People are often unaware of the frequent link between a broken bone and osteoporosis. Known as a silent disease because it progresses without symptoms, osteoporosis involves the gradual loss of bone tissue or bone density and results in bones so fragile they break under the slightest strain. Consequently, falls are especially dangerous for people who are unaware that they have low bone density. If the patient and the doctor fail to connect the broken bone to osteoporosis, the chance to make a diagnosis with a bone density test and begin a prevention or treatment program is lost. Bone loss continues, and other bones may break.

Even though bones do not break after every fall, the person who has fallen and broken a bone nearly always becomes fearful of falling again. As a result, she or he may limit activities for the sake of "safety." Among Americans age 65 and older, fall-related injuries are the leading cause of accidental death.

This publication explores the components of the **Fracture Triangle** and offers tips for reducing the chances of fall-related fractures that result from low bone mass and osteoporosis. If one of the following three factors is modified, the chances of breaking a bone are greatly reduced:



The **fall** itself

The **force** and direction of the fall

The **fragility** of the bone(s) that take the impact

The fall itself

Several factors can lead to a fall. Loss of footing or traction is a common cause of falls. Loss of footing occurs when there is less than total contact between a person's foot and the ground or floor. Loss of traction occurs when a person's feet slip on wet or slippery ground or floor. Other examples of loss of traction include tripping, especially over uneven surfaces such as sidewalks, curbs, or floor elevations that result from carpeting, risers, or scatter rugs. Loss of footing also happens from using household items intended for other purposes – for example, climbing on kitchen chairs or balancing on boxes or books to increase height.

A fall may occur because a person's reflexes have changed. As people age, reflexes slow down. Reflexes are automatic responses to stimuli in the environment. Examples of reflexes include quickly slamming on the car brakes when a child runs into the street or quickly moving out of the way when something accidentally falls. Aging slows a person's reaction time and makes it harder to regain one's balance following a sudden movement or shift of body weight.

Changes in muscle mass and body fat also can play a role in falls. As people get older, they lose muscle mass because they have become less active over time. Loss of muscle mass, especially in the legs, reduces a person's strength to the point where she or he is often unable to get up from a chair without assistance. In addition, as people age, they lose body fat that has cushioned and protected bony areas, such as the hips. This loss of cushioning also affects the soles of the feet, which upsets the person's ability to balance. The gradual loss of muscle strength, which is common in older people but not inevitable, also plays a role in falling. Muscle-strengthening exercises can help people regain their balance, level of activity, and alertness no matter what their age.

Improving balance

- Do muscle-strengthening exercises.
- Obtain maximum vision correction.
- Practice using bifocal or trifocal glasses.
- Practice balance exercises daily.

Changes in vision also increase the risk of falling. Diminished vision can be corrected with glasses. However, often these glasses are bifocal or trifocal so that when the person looks down through the lower half of her or his glasses, depth perception is altered. This makes it easy to lose one's balance and fall. To prevent this from happening, people who wear bifocals or trifocals must practice looking straight ahead and lowering their head. For many other older people, vision changes cannot be corrected completely, making even the home environment hazardous.

As people get older, they also are more likely to have a variety of chronic medical conditions that often require taking several medications. People with chronic illnesses that affect their circulation, sensation, mobility, or mental alertness as well as those taking some types of medications (see table below) are more likely to fall as a result of drug-related side effects such as dizziness, confusion, disorientation, or slowed reflexes.

Drinking alcoholic beverages also increases the risk of falling. Alcohol slows reflexes and response time; causes dizziness, sleepiness, or lightheadedness; alters balance; and encourages risky behaviors that can lead to falls.

The force and direction of a fall

The force of a fall (how hard a person lands) plays a major role in determining whether or not a person will break a bone. For example, the greater the distance of the hip bone to the floor, the greater the risk of fracturing a hip, so tall people appear to have an increased risk of fracture when they fall. The angle at which a person falls also is important. For example, falling sideways or straight down is more risky than falling backward.

Medications that may increase the risk of falling

- Blood pressure pills.
- Heart medicines.
- Diuretics or water pills.
- Muscle relaxants or tranquilizers.

Protective responses, such as reflexes and changes in posture that break the fall, can reduce the risk of fracturing a bone. Individuals who land on their hands or grab an object on their descent are less likely to fracture their hip, but they may fracture their wrist or arm. Although these fractures are painful and interfere with daily activities, they do not carry the high risks that a hip fracture does.

The type of surface on which a person lands also can affect whether or not a bone breaks. Landing on a soft surface is less likely to cause a fracture than landing on a hard surface.

Preliminary research suggests that by wearing trochanteric (hip) padding, people can decrease the chances of fracturing a hip after a fall. The energy created by the fall is distributed throughout the pad, lessening the impact to the hip. Further research is needed to fully evaluate the role of these devices in decreasing the risk of a hip fracture following a fall.

Bone fragility

Although most serious falls happen when people are older, steps to prevent and treat bone loss and falls can never begin too early. Many people begin adulthood with less than optimal bone mass, so the fact that bone mass or density is lost slowly over time puts them at increased risk for fractures.

Bones that once were strong become so fragile and thin that they break easily. Activities that once were done without a second thought are now avoided for fear that they will lead to another fracture.

Did you know?

- Being tall appears to increase your risk of a hip fracture.
- How you land increases your fracture risk.
- Catching yourself so you land on your hands or grabbing onto an object as you fall can prevent a hip fracture.

Prevention of falls and fractures

Safety first to prevent falls. At any age, people can change their environments to reduce their risk of falling and breaking a bone.

Outdoor safety tips:

- In nasty weather, use a walker or cane for added stability.
- Wear warm boots with rubber soles for added traction.
- Look carefully at floor surfaces in public buildings. Many floors are made of highly polished marble or tile that can be very slippery. If floors have plastic or carpet runners in place, stay on them whenever possible.
- Identify community services that can provide assistance, such as 24-hour pharmacies and grocery stores that take orders over the phone and deliver. It is especially important to use these services in bad weather.
- Use a shoulder bag, fanny pack, or backpack to leave hands free.
- Stop at curbs and check their height before stepping up or down. Be cautious at curbs that have been cut away to allow access for bikes or wheelchairs. The incline up or down may lead to a fall.

Steps to prevent fragile bones

- Consume adequate amounts of calcium and vitamin D (see “Recommended Calcium and Vitamin D Intake” chart).
- Exercise several times a week.
- Ask your doctor about a bone mineral density test.
- Ask about medications to slow bone loss and reduce fracture risk.

Indoor safety tips:

- Keep all rooms free from clutter, especially the floors.
- Keep floor surfaces smooth but not slippery. When entering rooms, be aware of differences in floor levels and thresholds.
- Wear supportive, low-heeled shoes, even at home. Avoid walking around in socks, stockings, or floppy, backless slippers.
- Check that all carpets and area rugs have skid-proof backing or are tacked to the floor, including carpeting on stairs.
- Keep electrical and telephone cords and wires out of walkways.
- Be sure that all stairwells are adequately lit and that stairs have handrails on both sides. Consider placing fluorescent tape on the edges of the top and bottom steps.
- For optimal safety, install grab bars on bathroom walls beside tubs, showers, and toilets. If you are unstable on your feet, consider using a plastic chair with a back and nonskid leg tips in the shower.
- Use a rubber bath mat in the shower or tub.
- Keep a flashlight with fresh batteries beside your bed.
- Add ceiling fixtures to rooms lit by lamps only, or install lamps that can be turned on by a switch near the entry point into the room. Another option is to install voice- or sound-activated lamps.
- Use bright light bulbs in your home.
- If you must use a step-stool for hard-to-reach areas, use a sturdy one with a handrail and wide steps. A better option is to reorganize work and storage areas to minimize the need for stooping or excessive reaching.
- Consider purchasing a portable phone that you can take with you from room to room. It provides security because you can answer the phone without rushing for it and you can call for help should an accident occur
- Don't let prescriptions run low. Always keep at least 1 week's worth of medications on hand at home. Check prescriptions with your doctor and pharmacist to see if they may be increasing your risk of falling. If you take multiple medications, check with your doctor and pharmacist about possible interactions between the different medications.
- Arrange with a family member or friend for daily contact. Try to have at least one person who knows where you are.
- If you live alone, you may wish to contract with a monitoring company that will respond to your call 24 hours a day.
- Watch yourself in a mirror. Does your body lean or sway back and forth or side to side? People with decreased ability to balance often have a high degree of body sway and are more likely to fall.

Reducing the force of a fall

Take steps to lessen your chances of breaking a bone in the event that you do fall:

- Remember that falling sideways or straight down is more likely to result in a hip fracture than falling in other directions. If possible, try to fall forward or to land on your buttocks.
- If possible, land on your hands or use objects around you to break a fall.
- Walk carefully, especially on hard surfaces.
- When possible, wear protective clothing for padding.

Practice balance exercises every day

- While holding the back of a chair, sink, or countertop, practice standing on one leg at a time for a minute. Gradually increase the time. Try balancing with your eyes closed. Try balancing without holding on.
- While holding the back of a chair, sink, or countertop, practice standing on your toes, then rock back to balance on your heels. Hold each position for a count of 10.
- While holding the back of chair, sink, or countertop with both hands, make a big circle to the left with hips, then repeat to the right. Do not move your shoulders or feet. Repeat five times.

Decreasing bone fragility

Individuals can protect bone health by following osteoporosis prevention and treatment strategies:

- Consume a calcium-rich diet that provides between 1,000 mg (milligrams) daily for men and women up to age 50. Women over age 50 and men over age 70 should increase their intake to 1,200 mg daily from a combination of foods and supplements.
- Obtain 600 IU (International Units) of vitamin D daily up to age 70. Men and women over age 70 should increase their uptake to 800 IU daily.
- Participate in weight-bearing and resistance-training exercises most days, preferably daily.
- Talk with your doctor about having a bone mineral density (BMD) test. The most widely recognized BMD test is called a dual-energy x-ray absorptiometry, or DXA test. It is painless, a bit like having an x-ray, but with much less exposure to radiation. It can measure bone density at your hip and spine.
- Talk with your doctor about possibly beginning a medication approved by the U.S. Food and Drug Administration for osteoporosis to stop bone loss, improve bone density, and reduce fracture risk.

People need to know whether they are at risk for developing osteoporosis or whether they have lost so much bone that they already have osteoporosis. Although risk factors can alert a person to the possibility of low bone density, only a BMD test can measure current bone density, diagnose osteoporosis, and determine fracture risk. Many different techniques measure bone mineral density painlessly and safely. Most of them involve machines that use extremely low levels of radiation to complete their readings. Sometimes, ultrasound machines, which rely on sound waves, are used instead.

Individuals may wish to have a BMD test to determine current bone health. Today, Medicare and many private insurance carriers cover bone density tests to detect osteoporosis for individuals who meet certain criteria. Talk with your doctor about whether or not this test would be appropriate for you. Falls are serious, but simple, inexpensive steps can be taken to reduce your risk of falling and of breaking a bone if you do fall.

Recommended calcium and vitamin D intakes

Life-stage group	Calcium mg/day	Vitamin D IU/day
Infants 0 to 6 months	200	400
Infants 6 to 12 months	260	400
1 to 3 years old	700	600
4 to 8 years old	1,000	600
9 to 13 years old	1,300	600
14 to 18 years old	1,300	600
19 to 30 years old	1,000	600
31 to 50 years old	1,000	600
51- to 70-year-old males	1,000	600
51- to 70-year-old females	1,200	600
>70 years old	1,200	800
14 to 18 years old, pregnant/lactating	1,300	600
19 to 50 years old, pregnant/lactating	1,000	600

Source: Food and Nutrition Board, Institute of Medicine, National Academy of Sciences, 2010.

Resources

For additional information on osteoporosis, contact the:
NIH Osteoporosis and Related Bone Diseases National Resource Center
Website: https://www.bones.nih.gov

For additional information on fall prevention, contact the:
National Institute on Aging Information Center
Website: https://www.nia.nih.gov

The National Institutes of Health Osteoporosis and Related Bone Diseases ~ National Resource Center acknowledges the assistance of the National Osteoporosis Foundation in the preparation of this publication.

For your information

For updates and for any questions about any medications you are taking, please contact the U.S. Food and Drug Administration toll free at 888-INFO-FDA (463-6332) or visit its website at https://www.fda.gov. For additional information on specific medications, visit Drugs@FDA at https://www.accessdata.fda.gov/scripts/cder/daf. Drugs@FDA is a searchable catalog of FDA-approved drug products.

NIH Publication No. 18–7892

Ride Safe

Information to help you travel more safely in motor vehicles while seated in your wheelchair

wc-transportation-safety.umtri.umich.edu

UMTRI TRANSPORTATION RESEARCH INSTITUTE
UNIVERSITY OF MICHIGAN

When traveling in a motor vehicle, it is generally safest for wheelchair users to transfer to a vehicle seat and use the vehicle seatbelt system or a child safety seat that complies with federal safety standards. The wheelchair should then be stored and secured in the vehicle.

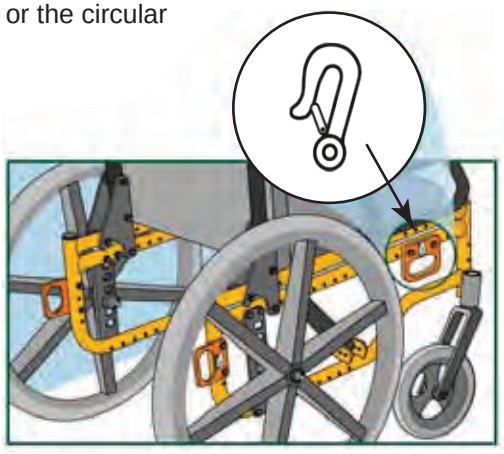
If transferring is not feasible or practical, it is very important to secure the wheelchair to the vehicle facing forward and to use crash-tested seatbelts for the wheelchair-seated rider.

1 START WITH THE RIGHT EQUIPMENT

The Wheelchair

▼ **It is best if you have a wheelchair that has been designed and tested for use as a seat in motor vehicles, often referred to as a WC19 wheelchair.** These wheelchairs comply with ANSI/RESNA WC19, a voluntary standard developed by safety and rehabilitation experts. Wheelchairs that meet the requirements of the WC19 standard will be labeled with words or the circular logo shown.

▼ Most importantly, a WC19 wheelchair has four, crash-tested securement points where tiedown straps and hooks can be easily attached. These points are clearly marked with a hook symbol.



▼ If a WC19 wheelchair is not available, the next best choice is a wheelchair with an accessible metal frame where tiedown straps and hooks can be attached at frame junctions.

The Wheelchair Tiedown and Occupant Restraint System (WTORS)

▼ It is important to use a complete WTORS to secure the wheelchair and provide the wheelchair occupant with a properly fitting lap and shoulder belt system.

▼ **Always use a WTORS that has been crash tested and labeled as complying with ANSI/RESNA WC18,** a voluntary standard developed by safety and rehabilitation experts. The most common type of wheelchair tiedown uses four straps to secure the wheelchair to the vehicle. Although it requires someone other than the wheelchair rider to secure and release the wheelchair, this tiedown can secure a wide range of WC19 and non-WC19 wheelchairs.

▼ To protect the rider during a crash or sudden braking, a seatbelt system with **both** lap and shoulder belts must be used. This will decrease the likelihood of injury caused by contact with the vehicle interior.

2 SECURE THE WHEELCHAIR

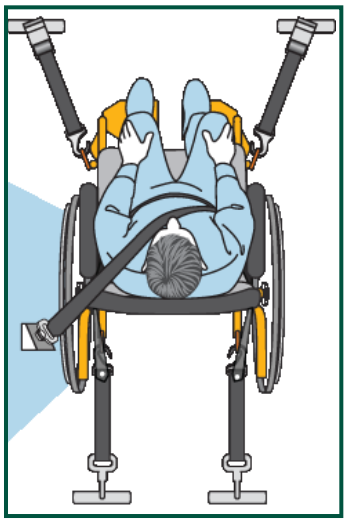
Four-Point Tiedowns

▼ Always position the wheelchair and rider facing forward in the vehicle.

▼ When securing a WC19 wheelchair, attach the four tiedown straps or hooks to the securement points provided on the wheelchair. Tighten the straps to remove all slack.

▼ If you do not have a WC19 wheelchair, it is best to attach the tiedown hooks or straps to welded junctions of the frame or to other structural areas where the frame is fastened together with hardened steel bolts -- often indicated by six raised lines or bumps on the bolt head.

▼ **Do not attach tiedowns to adjustable, moving, or removable parts of the wheelchair such as arm supports, foot supports, and wheels.**

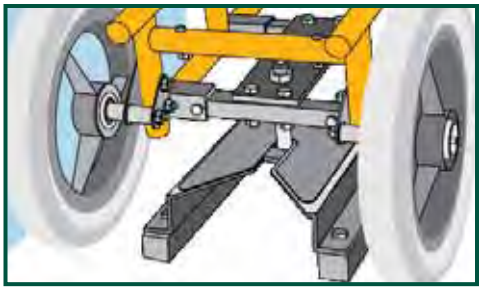


▼ When securing non-WC19 wheelchairs, choose structural securement points as cose to the seat surface as possible to provide greater wheelchair stability during travel. It is best if the rear securement points are high enough to result in angles of the rear tiedown straps between 30 and 45 degrees to the horizontal.

▼ If you have a non-WC19 wheelchair with a tilt seat, make sure to attach both the front and rear straps to either the seat frame or to the base frame. Mixing wheelchair securement points between the seat and base can result in the tiedown straps becoming slack if the angle of the seat changes during a crash.

▼ It is best if floor anchor points for rear tiedown straps are located directly behind the rear securement points on the wheelchair. If possible, the front tiedown straps should anchor to the floor at points that are spaced wider than the wheelchair to increase stability during travel.

Other Methods of Wheelchair Securement



▼ In some cases, wheelchairs can also be secured using a docking tiedown device. This method is mostly used in private vehicles since it requires added adaptor hardware on the wheelchair frame that will engage with the docking tiedown device in the vehicle. Docking securement devices allow the wheelchair rider to secure and release the wheelchair without assistance.

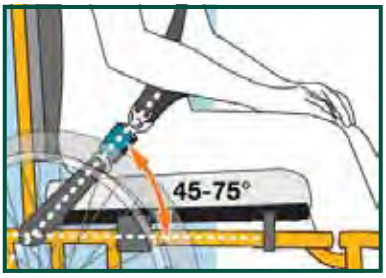
▼ If you plan to secure your wheelchair with a docking tiedown device, you should check with the WTORS or wheelchair manufacturer to ensure that your wheelchair model has been successfully crash tested with their system.

▼ Clamp-type securement devices are not recommended since they do not provide effective wheelchair securement in frontal crash testing.

3 PROTECT THE WHEELCHAIR RIDER

▼ In addition to securing the wheelchair, **it is very important to provide effective restraint for the wheelchair user with a crash-tested lap and shoulder belt or with a child restraint harness.** Postural support belts attached to the wheelchair are **not** strong enough to withstand crash forces and are usually not positioned correctly to protect the person safely in a crash.

▼ The lap belt should be placed low across the front of the pelvis on the upper thighs, not on the abdomen. When possible, the lap belt should be angled between 45 and 75 degrees to the horizontal when viewed from the side. Some wheelchair features, like armrests, can interfere with good belt fit. To avoid placing the lap belt over the armrest and to keep the lap belt low on the pelvis, it may be necessary to insert the belt between the armrest and the seatback, or through openings between the backrest and seat.



▼ A diagonal shoulder belt should cross the middle of the shoulder and the center of the chest, and should connect to the lap belt near the hip of the wheelchair rider. The upper shoulder-belt anchor point or guide should be anchored above and behind the top of the occupant's shoulder, so that the belt is in good contact with the shoulder and chest while traveling.

▼ Newer WC19 wheelchairs offer the option of a crash-tested lap belt that is anchored to the wheelchair frame. If the wheelchair has an onboard crash-tested lap belt, complete the belt system by attaching the lower end of a shoulder belt to the lap belt. Crash-tested wheelchair-anchored lap belts will be labeled to indicate that they comply with WC19.

Other Important Points

- Read and follow all manufacturers' instructions.
- It is best to ride with the wheelchair backrest positioned at an angle of 30 degrees or less to the vertical. If a greater recline angle is needed, the shoulder belt anchor point should be moved rearward along the vehicle sidewall so the belt maintains contact with the rider's shoulder and chest.
- Maximize the clear space around the rider to reduce the possibility of contact with vehicle components and other passengers in a crash. Cover rigid vehicle components that are close to the rider with dense padding.
- Check wheelchair and WTORS equipment regularly and replace worn components. If involved in a vehicle crash, check with the manufacturer to determine if the equipment needs to be replaced. Keep WTORS anchorage track free of debris.
- If possible, remove hard trays and secure them in the vehicle to reduce the chance of rider injury from contact with the tray. Consider the use of foam trays instead of rigid trays during transit. If it is not possible to remove a hard tray, place dense padding between the rider and the edge of the tray and make sure that the tray is securely attached to the wheelchair so it will not break loose and cause injury to other occupants in a crash.
- A properly positioned headrest may help protect the neck in a rear impact.
- If it is necessary to use a head and neck support during travel, choose a soft, light, neck collar because stiff collars and head straps are more likely to cause neck injury in a crash. The soft collar should not be attached to the seating system.
- Secure medical and other equipment to the wheelchair or vehicle to prevent it from breaking loose and causing injuries in a crash.
- Seating systems can be crashed tested to ANSI/RESNA WC20 and then used with a WC19-compliant frame to create a crashworthy wheelchair.

RESOURCES

Organizations

University of Michigan Transportation Research Institute
www.umtri.umich.edu
wc-transportation-safety.umtri.umich.edu

RESNA Rehabilitation Engineering and Assistive Technology Society of North America
www.resna.org

Wheelchair and Seating Manufacturers
(Ask for Products that have been Successfully Tested to WC19 and/or WC20)

ADI - Accessible Designs, Inc.
adirides.com; 888-684-2234

Amylior Inc.
www.amysystems.com; 888-453-0311

Bergeron Health Care
www.specialtomato.com; 866-529-8407

Broda Seating
www.brodaseating.com; 844-552-7632

The Comfort Company
www.comfortcompany.com; 800-564-9248

Convaid / R82
www.convaid.com; 844-876-6245

Drive Medical / Inspired by Drive
www.drivemedical.com; 877-224-0946
www.inspiredbydrive.com; 800-454-6612

Dynamic Health Care Solutions
www.dynamichcs.com; 866-875-2877

Eurovema AB
www.eurovema.se; +46-371-390-100

Freedom Designs
www.freedomdesigns.com; 800-331-8551

Gunnell
www.gunnell-inc.com; 800-551-0055

Harris Medical LLC
Eztransportchair.com; 954-609-4214

Hoggi
www.hoggi.de; +49 2623 92499-0
or 877-767-9462

Icon Wheelchairs, Inc.
www.iconwheelchairs.com; 888-461-5759

Innovative Products
www.mobility4kids.com; 800-950-5185

Invacare
www.invacare.com; 800-333-6900

Ki Mobility
www.kimobility.com; 800-981-1540

Leggero, LLC
leggero.us; 844-503-KIDS (5437)

Maple Leaf Wheelchair
www.mapleleafwheelchair.ca; 905-564-2250

Medifab / Spex
www.spexseating.com; +64 3 307 9790

Merits Health Products, Inc.
meritshealth.com; 800-963-7487

Metalcraft Industries
www.metalcraft-industries.com; 888-399-3232

Motion Composites
www.motioncomposites.com; 866-650-6555

Motion Concepts
www.motionconcepts.com; 888-433-6818

NuTec Rehab
www.nutecrehab.com; 888-448-0093

Permobil / TiLite / ROHO
www.permobil.com; 800-736-0925

Pride Mobility Products Corp.
www.pridemobility.com; 800-800-8586

Product Design Group
www.pdgmobility.com; 888-858-4422

Rolapal Ltd
www.rolapal.co.nz; +64 9 634 2300

Stealth Products
www.stealthproducts.com; 800-965-9229

Sunrise Medical
www.sunrisemedical.com; 800-333-4000

Therafin Corporation
www.therafin.com; 800-843-7234

V-Trak
www.v-trak.com; 866-632-1755

Varilite
www.varilite.com; 800-827-4548

WHILL, Inc.
whill.us; 844-699-4455

XPlore Mobility
www.xploremobility.com; 888-575-9225

Wheelchair Tiedown and Occupant Restraint Manufacturers (Ask for Products that Comply with WC18)

B&D Independence
bdindependence.com; 618-262-7117

EZ-Lock
www.ezlock.net; 225-214-4620

New Haven
www.safehaven-usa.com; 800-421-8700

Orthosafe
www.orthosafe.com; 609-587-9444

Q'Straint
www.qstraint.com; 800-987-9987

SureLok
www.sure-lok.com; 866-787-3565

GLOSSARY OF TERMS

Anchor point: The location on a vehicle, wheelchair, or wheelchair tiedown where a belt-restraint or wheelchair-tiedown anchorage is attached.

ANSI/RESNA WC18 (SECTION 18 RESNA WC-4:2017): A voluntary standard for WTORS.
NOTE: ISO 10542 is an international standard that is comparable with WC18.

ANSI/RESNA WC19 (SECTION 19 RESNA WC-4:2017): A voluntary standard for wheelchairs designed for use as a seat when traveling in a motor vehicle.
NOTE: ISO 7176-19 is an international wheelchair standard that is comparable with WC19.

ANSI/RESNA WC20 (SECTION 20 RESNA WC-4:2017): A voluntary standard for wheelchair seating systems designed or used as part of a wheelchair when traveling in a motor vehicle.
NOTE: ISO 16840-4 is an international wheelchair standard that is comparable with WC20.

SAE Recommended Practice J2249: A Society of Automotive Engineers Recommended Practice for WTORS that has been replaced by ANSI/RESNA WC18.
NOTE: WC18 is an enhanced version of this standard and ISO 10542 is a similar international standard.

Belt: A length of energy-absorbing webbing material used in occupant restraint systems.

Docking tiedown: A method for securing wheelchairs where portions of the wheelchair frame, or add-on brackets fastened to the wheelchair frame, engage with a securement device anchored to the vehicle.

Four-point strap-type tiedown: A method for securing a wheelchair where four straps are attached to the wheelchair at four separate securement points and attached to the vehicle at four separate anchor points.

Occupant restraint: A system or device designed to protect a motor vehicle occupant in a crash by keeping them in the seat and minimizing contact with objects inside or outside the vehicle.

Postural support: A padded component and/or belt used to help maintain a person in a desired position during normal wheelchair use. In general postural supports are **not** designed to provide effective occupant restraint in a motor vehicle crash.

Securement points: Specific structural points on the wheelchair base or seat frame that are designed for attachment of wheelchair tiedown straps.

Strap: A length of webbing material used in wheelchair tiedown systems.

WC19 wheelchair: A crash-tested wheelchair with four clearly identified securement points that meets ANSI/RESNA WC19.

WC20 seating system: A crash-tested seating system and its attachment hardware that meets ANSI/RESNA WC20 and is used with a WC19 compliant frame to create a crashworthy wheelchair.

Wheelchair tiedown and occupant-restraint system (WTORS): A complete system for securing the occupied wheelchair and a belt-type restraint system for limiting occupant movement in a motor vehicle crash.

University of Michigan Transportation Research Institute

University of Michigan Health System

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2018

GLASGOW COMA SCALE : Do it this way

GCS at 40 | EYES VERBAL MOTOR

Institute of Neurological Sciences NHS Greater Glasgow and Clyde

CHECK

For factors Interfering with communication,
ability to respond and other injuries

OBSERVE

Eye opening , content of speech and movements of right and left sides

STIMULATE

Sound: spoken or shouted request
Physical: Pressure on finger tip, trapezius or supraorbital notch

RATE

Assign according to highest response observed

Eye opening

Criterion	Observed	Rating	Score
Open before stimulus	✔	Spontaneous	4
After spoken or shouted request	✔	To sound	3
After finger tip stimulus	✔	To pressure	2
No opening at any time, no interfering factor	✔	None	1
Closed by local factor	✔	Non testable	NT

Verbal response

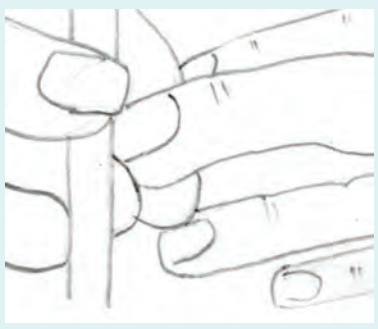
Criterion	Observed	Rating	Score
Correctly gives name, place and date	✔	Orientated	5
Not orientated but communication coherently	✔	Confused	4
Intelligible single words	✔	Words	3
Only moans / groans	✔	Sounds	2
No audible response, no interfering factor	✔	None	1
Factor interferring with communication	✔	Non testable	NT

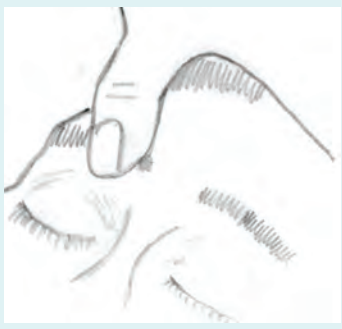
Best motor response

Criterion	Observed	Rating	Score
Obey 2-part request	✔	Obeys commands	6
Brings hand above clavicle to stimulus on head neck	✔	Localising	5
Bends arm at elbow rapidly but features not predominantly abnormal	✔	Normal flexion	4
Bends arm at elbow, features clearly predominantly abnormal	✔	Abnormal flexion	3
Extends arm at elbow	✔	Extension	2
No movement in arms / legs, no interfering factor	✔	None	1
Paralysed or other limiting factor	✔	Non testable	NT

Sites For Physical Stimulation

Finger tip pressure | Trapezius Pinch | Supraorbital notch





Features of Flexion Responses

Modified with permission from Van Der Naalt 2004
Ned Tijdschr Geneeskd

Abnormal Flexion

Slow Sterotyped
Arm across chest
Forearm rotates
Thumb clenched
Leg extends

Normal flexion

Rapid
Variable
Arm away from body

For further information and video demonstration visit www.glasgowcomascale.org

Graphic design by Margaret Frej based on layout and illustrations from Medical Illustration M I • 268093

Everything You Need to Know About Pain

Medically reviewed by Deborah Weatherspoon, Ph.D., R.N., CRNA — Writtenby Amber Erickson Gabbey on April 5, 2021

What is pain?

- Pain is a general term that describes uncomfortable sensations in the body. It stems from activation of the nervous system.
- Pain can range from annoying to debilitating. It may feel like a sharp stab or dull ache. It may also be described as throbbing, pinching, stinging, burning, or sore.
- Pain may be consistent, it may start and stop frequently, or it may occur only under some conditions. It may be acute, developing suddenly and lasting for a short period of time. Or it may be chronic, with ongoing sensations that last or return repeatedly over several months or years.
- Pain may be localized, affecting a specific part of your body. Or it may be generalized, such as the overall body aches associated with the flu.
- People respond to pain differently. Some people have a high tolerance for pain, while others have a low tolerance. Pain is highly subjective.
- Pain lets us know when something is wrong and gives us hints about the cause. Some pain is easy to diagnose and can be managed at home. Other types of pain are signs of serious health conditions that require medical attention to treat.

What causes pain?

- In some cases, pain is clearly caused by a specific injury or medical condition. In other cases, the cause of the pain may be less obvious or unknown.
- Some common causes of pain include:
    - headache
    - toothache
    - sore throat
    - stomachache or cramps muscle cramps or strains cuts, burns, or bruises bone fractures
- Many illnesses or disorders, such as the flu, arthritis, endometriosis, and fibromyalgia, can cause pain. Depending on the underlying cause, you may develop other symptoms as well. For example, these may include fatigue, swelling, nausea, vomiting, or mood changes.

Types of pain

There are several different types of pain. It's possible to experience more than one type at the same time. If you're in pain, identifying the type of pain may help your healthcare professional narrow down the potential causes and develop a treatment plan.

- Acute pain
    - Acute pain develops over a short period of time. It tends to occur suddenly, often as a result of a known injury, illness, or medical procedure.
    - For example, acute pain may result from:
        - injuries such as cuts, burns, muscle strains, or bone fractures

        - illnesses such as food poisoning, strep throat, or appendicitis
        - medical procedures such as injections, dental work, or surgery
    - Acute pain tends to be sharp, rather than dull. It usually goes away within a few days, weeks, or months, after the cause has been treated or resolved.
- Chronic pain
    - Chronic pain lasts, or comes and goes, over multiple months or years.
    - It may result from a variety of health conditions, such as arthritis, fibromyalgia, chronic migraine, or cancer.
    - Some people also experience chronic pain following an injury, even after the initial injury has healed.
    - In some cases, the cause of chronic pain is hard to identify. Some people experience chronic pain when there's no other evidence of underlying injury or illness. This is known as *functional pain.*
    - The National Health Interview Survey found that in 2019, roughly 1 in 5 adults in the United States had chronic pain. More than 7% had chronic pain that frequently limited their activities at work or in wider life.
- Nociceptive pain
    - Nociceptive pain is caused by tissue damage.
        - For example, it may result from injuries such as cuts, burns, bruises, or fractures.
        - It may also result from certain health conditions that cause tissue inflammation and damage, such as arthritis, osteoporosis, or inflammatory bowel disease (IBD).
    - When nociceptive pain develops in your skin, muscles, ligaments, tendons, joints, or bones, it's known as *somatic pain*. When it develops in your internal organs, it's known as visceral pain.
    - Nociceptive pain may be acute or chronic, depending on the underlying cause. It may feel achy, throbbing, or sharp.
    - Nociceptive pain affects almost everyone at some point in their lifetime.
- Neuropathic pain
    - Neuropathic pain results from nerve damage, which may be caused by a variety of injuries and illnesses. For example, you may experience neuropathic pain if one of the discs in your spine slips out of place and puts pressure on a nerve.
    - You may also develop neuropathic pain because of certain illnesses, such as shingles, diabetes, multiple sclerosis, or cancer.
    - One study in the United States found that 10% of adults experience pain that's likely neuropathic. It tends to be chronic, but acute neuropathic pain may also occur.
- Functional pain
    - Functional pain is pain that's caused by no obvious injury or damage to your body. It tends to be chronic, although acute functional pain may also develop.

    - More than 15% of the world's population has a functional pain syndrome, report researchers in BJA Education.
    - Examples of functional pain syndromes include:
        - fibromyalgia, which causes widespread pain throughout the body
        - irritable bowel syndrome (IBS), which causes abdominal pain
        - temporomandibular dysfunction, which causes jaw pain
        - chronic cardiac chest pain, which causes chest pain

<u>When to seek help</u>

Seek medical attention for your pain if it's:

- the result of an injury or accident that may have caused substantial damage to your body, including severe or uncontrollable bleeding, broken bones, or head injury
- located in your chest, back, shoulders, neck, or jaw and accompanied by other potential signs or symptoms of a heart attack, such as pressure in your chest, shortness of breath, dizziness, weakness, cold sweats, nausea, or vomiting
- interfering with your day-to-day life, including your ability to sleep, work, or take part in other activities that are important to you

<u>How is pain diagnosed?</u>

- If you seek medical attention for your pain, your healthcare professional will first do a physical examination and ask you some questions.
- Be prepared to describe the pain specifically, including when it started, when it is most intense, and whether it is mild, moderate, or severe.
- Your doctor may also ask you:
    - how the pain affects your life
    - if you have other symptoms
    - if there are triggers that make the pain worse
    - if you have any diagnosed health conditions
    - if you've had any recent injuries or illnesses
    - if you have recently changed your diet or exercise routine if you're taking medications or supplements
- Depending on your symptoms and medical history, your doctor may order one or more of the following tests to check for potential causes of your pain:
    - blood tests, urine tests, stool tests, or cerebral spinal fluid tests
    - endoscopy to check for signs of damage or other problems in your respiratory, gastrointestinal, urinary, or reproductive tract
    - X-ray, CT scan, MRI scan, or ultrasound scan to check for signs of damage in your muscles, ligaments, tendons, bones, nerves, or internal organs
    - biopsy to collect a sample of tissue for analysis
    - nerve function tests to learn how your nerves are working
    - psychological tests to check for conditions such as depression

- If they can't find any signs of underlying damage that may be causing the pain, you might have a functional pain syndrome. These syndromes are diagnosed based on symptoms, after other potential causes are ruled out.

How is pain treated?

- Treatment for pain depends on the underlying issue or injury that's causing it, if known.
- Acute pain will generally go away once the cause has been treated or resolved.
- Chronic pain can be more difficult to manage, especially if it's *functional pain* that results from an unknown cause.
- If the pain was caused by an injury, it might heal naturally with time, or you might need medication, surgery, or other medical attention.
- If the pain is caused by an infection, it might resolve on its own or you might need medication or other treatments.
- for chronic health condition such as arthritis, cancer, or chronic migraine, the doctor might prescribe medication, surgery, or other therapies to help treat it.
- The healthcare professional might also recommend treatments to ease the pain itself.
- For example, they may recommend or prescribe:
    - over-the-counter pain relievers, such as acetaminophen, aspirin, or
    - prescription anti-inflammatory drugs, such as as corticosteroids or certain types of COX-2 inhibitors
    - opioid medications, which may be prescribed for acute pain following an injury or surgery
    - antidepressant or anti-seizure medications, which may be prescribed for some types of neuropathic pain or functional pain syndromes
    - physical therapy, which may help relieve pain caused by injuries or certain health conditions such as arthritis or multiple sclerosis
    - occupational therapy, which may help you learn how to adapt your daily activities and environments to limit pain
- The doctor may also recommend complementary therapies, such as:
    - biofeedback, in which a therapist uses electronic devices to help the person learn how to consciously control body functions such as breathing
    - acupuncture or acupressure, in which a practitioner stimulates certain pressure points on your body to help relieve chronic pain
    - massage, in which a therapist rubs, kneads, or presses on muscles or other soft tissues to help ease tension and pain
    - meditation, in which you focus your mind in ways intended to relieve stress and tension
    - tai chi or yoga, which combine gentle movements and deep breathing to stretch and stimulate your muscles and ease tension
    - progressive muscle relaxation, in which you consciously tighten and then relax different muscle groups to promote natural relaxation
    - guided imagery, in which you visualize calming images

- The doctor may also recommend lifestyle changes or home remedies to help manage pain.
    - For example, they might encourage you to:
        - apply a towel-wrapped cold pack or ice to reduce painful swelling and inflammation caused by injuries or chronic conditions such as arthritis
        - apply heating pads or take warm baths to ease muscles stiffness, soreness, or cramps
        - limit or avoid certain activities or triggers that make your pain worse
        - take steps to limit and ease stress
        - get regular gentle exercise
        - get enough sleep
- For minor injuries that don't require medical attention, follow the general rule of RICE:
    - ***R**est*. Rest and protect the injured or sore area. Stop, change, or take a break from any activity that may be causing your pain or soreness.
    - ***I**ce*. Cold will reduce pain and swelling. Apply an ice or cold pack right away to prevent or minimize swelling. Apply the ice or cold pack for 10 to 20 minutes, 3 or more times a day. After 48 to 72 hours, if swelling is gone, apply heat to the area that hurts. Do not apply ice or heat directly to the skin. Place a towel over the cold or heat pack before applying it to the skin.
    - ***C**ompression*. Compression, or wrapping the injured or sore area with an elastic bandage (such as an Ace wrap), will help decrease swelling. Don't wrap it too tightly because this can cause more swelling below the affected area. Loosen the bandage if it gets too tight. Signs that the bandage is too tight include numbness, tingling, increased pain, coolness, or swelling in the area below the bandage. Talk to your doctor if you think you need to use a wrap for longer than 48 to 72 hours; a more serious problem may be present.
    - ***E**levation*. Elevate the injured or sore area on pillows while applying ice and anytime you are sitting or lying down. Try to keep the area at or above the level of your heart to help minimize swelling.

The takeaway

- Pain is a sign that something is wrong in your body. It may be caused by a wide variety of injuries, diseases, and functional pain syndromes.
- In general, the most effective way to treat pain is to address the underlying cause if it can be identified. In some cases, the injury or illness causing the pain may heal or resolve on its own. In other cases, you may need medication, surgery, or other therapies to treat the cause. Sometimes, your provider may not be able to identify the cause.
- If you think your pain is caused by a serious injury or illness that requires medical attention to treat, contact your doctor or emergency medical services. Let them know if you've been experiencing pain that interferes with your daily life.
- Your healthcare professional can help you develop a plan to manage the pain.

The Difference Between the Types of Pain: Acute vs. Chronic

When it comes to your pain, there are two different types of pain: acute and chronic. All pain is uncomfortable, unwanted, and unpleasant.

No matter the type of pain, it can range from mild to severe and all pain has the ability to reduce your quality of life and prevent you from living the life you deserve.

The main difference between the two main types of pain, acute and chronic pain, is that acute pain typically has a specific, treatable cause. Chronic pain is not so easily diagnosed because it can be rooted in underlying, "invisible" causes.

Acute Pain

Acute pain is a sudden, sharp pain that lasts less than 6 months. Acute pain acts as a warning to your body that it is unsafe, and its health has been compromised. A common belief is that acute pain is mild and temporary. But in fact, acute pain is very complex.

This type of pain is caused by something specific – a broken bone, burns or cuts, or even labor and childbirth. The pain goes away once the affect area has been treated. Some acute pain is temporary and short-lived. Other times, it can have a longer-lasting effect and cause severe pain.

Therapy for acute pain treats the cause of the pain. However, it can be tricky to diagnose because the symptoms can be start and stop without warning. The pain does not last all day and night, and can produce symptoms that last a few days, a few seconds, or even just a few seconds.

Doctors use the Wong-Baker FACES pain rating scale (below) and have their patients rate their pain on a scale from 1 to 10. This helps the doctor learn about the pain levels and allows them to better assess the situation.

Other test doctors use may include:

- Blood work
- Imaging (MRIs, CT scans, and X-Rays)
- Dye-injection studies
- Nerve Conduction studies

Chronic Pain

Chronic pain is an ongoing pain that last longer than 6 months. This pain is considered a disease state and affects 1 out of 5 American adults. Chronic pain is hard to diagnose and can be misdiagnosed.

Chronic pain is caused by an underlying issue, something surgery may not be able to heal. Pain patients may undergo a variety of treatments to find one, or a combination of many, that helps reduce their pain.

Many types of chronic pain are headaches, arthritis, cancer, nerve pain, back pain, and fibromyalgia. Pain is different for everyone, especially chronic pain. Because of this, treatment options for chronic pain vary and can include everything from a topical cream to surgery.

Oral medications and creams or ointments, such as over-the-counter anti-inflammatories, pain relievers, and creams help reduce pain on a daily basis, but are not meant for long-term use. Treatments without medication like physical therapy is proven to help patients with chronic pain reduce pain, increase quality of living, while minimizing medication dosages. Other treatments include acupuncture and TENS stimulation. TENS units are external pads that provide stimulation around the area of pain and help reduce pain.

Patients who suffer from severe chronic pain are candidates for injections. The injections are a mixture of an anesthetic and steroid that is injected in the area of pain to help alleviate the pain.

- Trigger Point Injections – These injections help relax and soothe tense muscles that are inflamed and painful.
- Facet Joint Injections – These injections help inflamed joints in the spine. The steroid helps reduce pain and joint soreness.
- Epidural Injections – This kind of injection is a strong anti-inflammatory that reduces pain around the spinal cord.

Chronic pain reduces a person's ability to live their lives the same quality and extent that they enjoy. Knowing your options and treatment availability is important to maintaining the lifestyle you deserve and to keep you doing the things you love.

Southern Pain and Neurological https://southernpainclinic.com/contact/

Types of Pain Scales

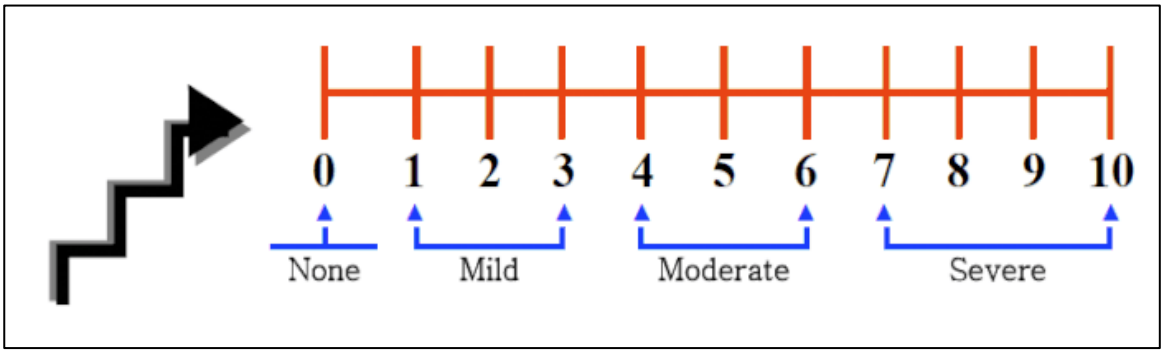
Pain scale results can help guide the diagnostic process, track the progression of a condition, and more. There are at least 10 pain scales in common use, which are described below. They tend to fall into certain categories:

- **Numerical rating scales (NRS)** use numbers to rate pain.
- **Visual analog scales (VAS)** typically ask a patient to mark a place on a scale that aligns with their level of pain.
- **Categorical scales** use words as the primary communication tool and may also incorporate numbers, colors, or relative location to communicate pain.

Numerical scales are more quantitative in nature, but most pain scales have quantitative features and qualitative features.

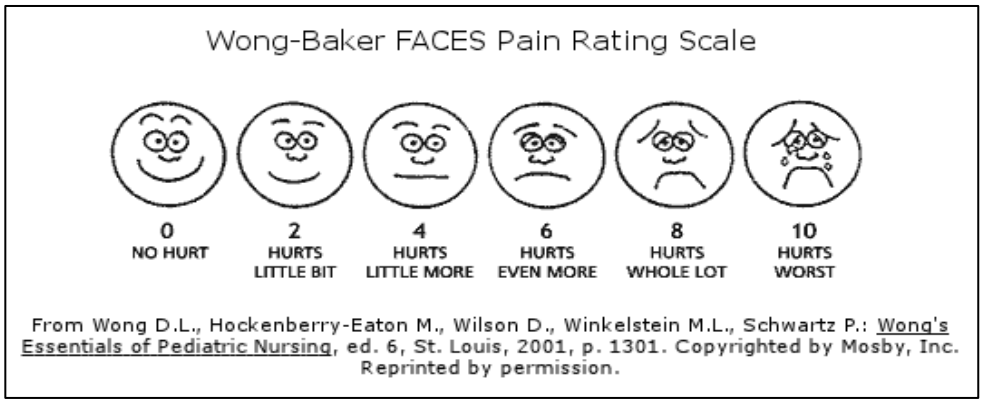
Numerical Rating Pain Scale

- The most used pain scales in health care, the numerical rating scale is designed to be used by those over age 9.
- With the numerical scale, the person has the option to verbally rate their pain from 0 to 10 or to place a mark on a line indicating your level of pain.
- Zero indicates the absence of pain, while 10 represents the most intense pain possible.



Wong-Baker Faces Pain Scale

- The Wong-Baker FACES Pain Scale combines pictures and numbers for pain ratings.
- It can be used in children over the age of 3 and in adults.
- Six faces depict different expressions, ranging from happy to extremely upset. Each is assigned a numerical rating between 0 (smiling) and 10 (crying). If you have pain, you can point to the picture that best represents the degree and intensity of your pain.



FLACC Scale

- FLACC stands for face, legs, activity, crying, and consolability.
- The FLACC pain scale was developed to help medical observers assess the level of pain in children who are too young to cooperate verbally. It can also be used in adults who are unable to communicate.
- The FLACC scale is based on observations, with zero to two points assigned for each of the five areas.
- The overall score is recorded as follows:
    - 0: Relaxed and comfortable
    - 1 to 3: Mild discomfort
    - 4 to 6: Moderate pain
    - 7 to 10: Severe discomfort/pain
- By recording the FLACC score periodically, healthcare providers can gain some sense of whether someone's pain is increasing, decreasing, or stable.

DATE/TIME						
Face 0 - No particular expression or smile 1 - Occasional grimace or frown, withdrawn, disinterested 2 - Frequent to constant quivering chin, clenched jaw						
Legs 0 – Normal position or relaxed 1 – Uneasy, restless, tense 2 – Kicking, or legs drawn up						
Activity 0 – Lying quietly, normal position, moves easily 1 – Squirming, shifting back and forth, tense 2 – Arched, rigid or jerking						
Cry 0 – No cry (awake or asleep) 1 – Moans or whimpers; occasional complaint 2 - Crying steadily, screams or sobs, frequent complaints						
Consolability 0 – Content, relaxed 1 – Reassured by occasional touching, hugging or being talked to, distractible 2 – Difficult to console or comfort						
TOTAL SCORE						

CRIES Scale

- CRIES assesses crying, oxygenation, vital signs, facial expression, and sleeplessness.
- It is often used for infants 6 months old and younger and is widely used in the neonatal intensive care setting.
- This assessment tool is based on observations and objective measurements.
- It is rated by a healthcare professional, such as a nurse or physician.
- Two points are assigned to each parameter, with a rating of 0 for signs of no pain and a rating of 2 for signs of maximal pain.

DATE/TIME						
Crying - Characteristic cry of pain is high pitched. 0 – No cry or cry that is not high-pitched 1 - Cry high pitched but baby is easily consolable 2 - Cry high pitched but baby is inconsolable						
Requires O_2 for SaO_2 < 95% - Babies experiencing pain manifest decreased oxygenation. Consider other causes of hypoxemia, e.g., oversedation, atelectasis, pneumothorax) 0 – No oxygen required 1 – < 30% oxygen required 2 – > 30% oxygen required						
Increased vital signs (BP* and HR*) - Take BP last as this may awaken child making other assessments difficult **0** – Both HR and BP unchanged or less than baseline **1** – HR or BP increased but increase in < 20% of baseline **2** – HR or BP is increased > 20% over baseline.						
Expression - The facial expression most often associated with pain is a grimace. A grimace may be characterized by brow lowering, eyes squeezed shut, deepening naso-labial furrow, or open lips and mouth. **0** – No grimace present **1** – Grimace alone is present **2** – Grimace and non-cry vocalization grunt is present						
Sleepless - Scored based upon the infant's state during the hour preceding this recorded score. 0 – Child has been continuously asleep 1 – Child has awakened at frequent intervals **2** – Child has been awake constantly						
TOTAL SCORE						

COMFORT Scale

The COMFORT Scale is a pain scale that may be used by a healthcare provider when a person cannot describe or rate their pain. Some of the common populations this scale might be used with include:

- Children
- Adults who are cognitively impaired
- Adults whose cognition is temporarily impaired by medication or illness
- People who are sedated in an ICU or operating room setting

The COMFORT Scale provides a pain rating between nine and 45 based on nine different parameters, each rated from one to five:

- **Alertness** is given a score of 1 for deep sleep, 2 for light sleep, 3 for drowsiness, 4 for alertness, and 5 for high alertness.
- **Calmness** is rated with a score of 1 for complete calmness, and higher ratings given for increased anxiety and agitation.
- **Respiratory distress** is rated based on how much a person's breathing reflects pain, with agitated breathing given higher ratings.
- **Crying** is given a score of 1 for no crying, and higher scores for moaning, sobbing, or screaming.
- **Physical movement** is given a score of 0 for no movement, which can be a sign of less pain or of illness. A score of 1 or 2 indicates some movement, and higher scores indicate vigorous movements.
- **Muscle tone** is rated at a score of 3 if it is normal, with lower scores indicating diminished muscle tone and higher scores indicating increased tone or rigidity.
- **Facial tension** is rated at a score of 1 for a completely normal, relaxed face, and given higher ratings for signs of facial muscle strain.
- **Blood pressure and heart rate** are rated with respect to the normal baseline. A score of 1 indicates that these measures are below the baseline (abnormal), and a score of 2 indicates they are at baseline, while higher scores are given for elevated (abnormal) levels.
- *Scale available on the next page.*

COMFORT Scale

	DATE/TIME						
ALERTNESS	1 - Deeply asleep 2 - Lightly asleep 3 - Drowsy 4 - Fully awake and alert 5 - Hyper alert						
CALMNESS	1 - Calm 2 - Slightly anxious 3 - Anxious 4 - Very anxious 5 - Panicky						
RESPIRATORY DISTRESS	1 - No coughing and no spontaneous respiration 2 - Spontaneous respiration with little or no response to ventilation 3 - Occasional cough or resistance to ventilation 4 - Actively breathes against ventilator or coughs regularly 5 - Fights ventilator; coughing or choking						
CRYING	1 - Quiet breathing, no crying 2 - Sobbing or gasping 3 - Moaning 4 - Crying 5 - Screaming						
PHYSICAL MOVEMENT	1 - No movement 2 - Occasional, slight movement 3 - Frequent, slight movements 4 - Vigorous movement 5 - Vigorous movements including torso and head						
MUSCLE TONE	1 - Muscles totally relaxed; no muscle tone 2 - Reduced muscle tone 3 - Normal muscle tone 4 - Increased muscle tone and flexion of fingers and toes 5 - Extreme muscle rigidity and flexion of fingers and toes						
FACIAL TENSION	1 - Facial muscles totally relaxed 2 - Facial muscle tone normal; no facial muscle tension evident 3 - Tension evident in some facial muscles 4 - Tension evident throughout facial muscles 5 - Facial muscles contorted and grimacing						
BLOOD PRESSURE (MAP) BASELINE	1 - Blood pressure below baseline 2 - Blood pressure consistently at baseline 3 - Infrequent elevations of 15% or more above baseline (1-3 during 2 minutes observation) 4 - Frequent elevations of 15% or more above baseline (> 3 during 2 minutes observation) 5 - Sustained elevations of 15% or more						
HEART RATE BASELINE	1 - Heart rate below baseline 2 - Heart rate consistently at baseline 3 - Infrequent elevations of 15% or more above baseline (1-3 during 2 minutes observation) 4 - Frequent elevations of 15% or more above baseline (> 3 during 2 minutes observation) **5** - Sustained elevations of 15% or more						
	TOTAL SCORE						

McGill Pain Scale

- The McGill Pain Questionnaire consists of 78 words that describe pain.
- A person rates their own pain by marking the words that most closely match up to their feelings. Some examples of the words used are tugging, terrifying, cold, sharp, and wretched.
- Once a person has made their selections, a numerical score with a maximum rating of 78 is assigned based on how many words were marked.
- This scale is helpful for adults and children who can read.

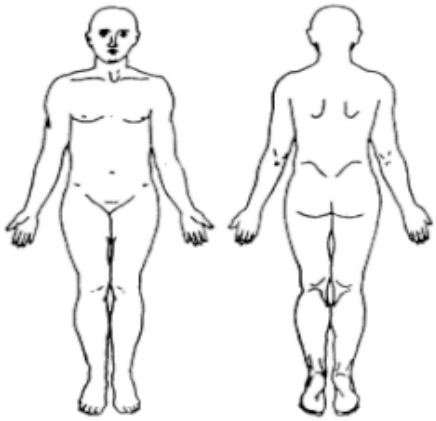
McGill Pain Questionnaire

Patient's Name _____ Date _____ Time _____ am/pm

PRI: S _____ (1-10) A _____ (11-15) E _____ (16) M _____ (17-20) PRI(T) _____ (1-20) PPI _____

1 FLICKERING QUIVERING PULSING THROBBING BEATING POUNDING	11 TIRING EXHAUSTING	
2 JUMPING FLASHING SHOOTING	12 SICKENING SUFFOCATING	
3 PRICKING BORING DRILLING STABBING LANCINATING	13 FEARFUL FRIGHTFUL TERRIFYING	
4 SHARP CUTTING LACERATING	14 PUNISHING GRUELLING CRUEL VICIOUS KILLING	
5 PINCHING PRESSING GNAWING CRAMPING CRUSHING	15 WRETCHED BLINDING	
6 TUGGING PULLING WRENCHING	16 ANNOYING TROUBLESOME MISERABLE INTENSE UNBEARABLE	
7 HOT BURNING SCALDING SEARING	17 SPREADING RADIATING PENETRATING PIERCING	
8 TINGLING ITCHY SMARTING STINGING	18 TIGHT NUMB DRAWING SQUEEZING TEARING	
9 DULL SORE HURTING ACHING HEAVY	19 COOL COLD FREEZING	
10 TENDER TAUT RASPING SPLITTING	20 NAGGING NAUSEATING AGONIZING DREADFUL TORTURING	
	PPI 0 NO PAIN 1 MILD 2 DISCOMFORTING 3 DISTRESSING 4 HORRIBLE 5 EXCRUCIATING	

BRIEF MOMENTARY TRANSIENT	RHYTHMIC PERIODIC INTERMITTENT	CONTINUOUS STEADY CONSTANT



E = EXTERNAL
I = INTERNAL

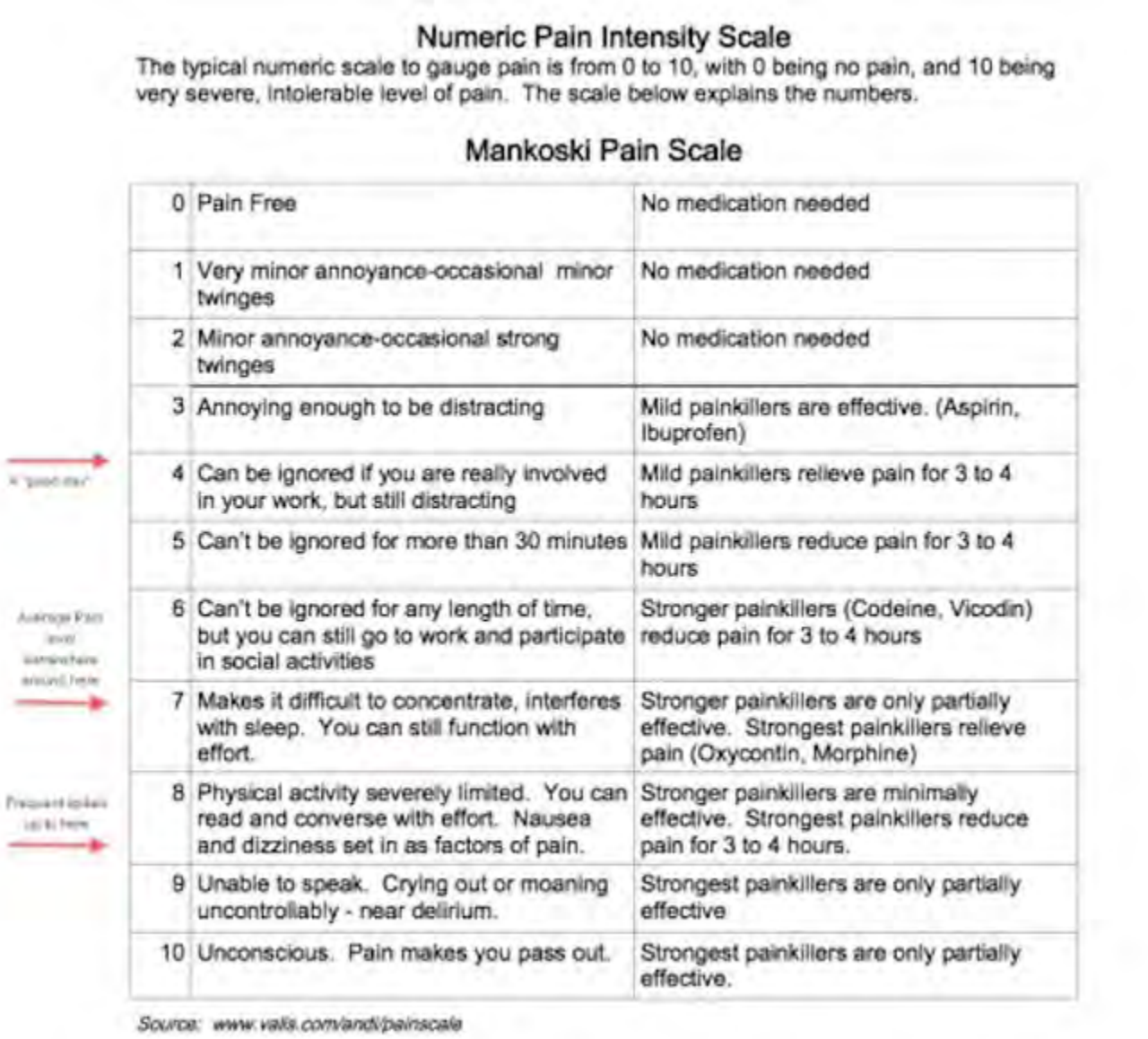
COMMENTS:

Color Analog Scale

- The color analog pain scale uses colors, with red representing severe pain, yellow representing moderate pain, and green representing comfort.
- The colors are usually positioned in a linear format with corresponding numbers or words that describe your pain.
- The color analog scale is often used for children and is considered reliable.

Mankoski Pain Scale

- The Mankoski pain scale uses numbers and corresponding descriptions of pain.
- Descriptions are detailed, including phrases such as "very minor annoyance, occasional minor twinges" or "cannot be ignored for more than 30 minutes."



Numeric Pain Intensity Scale

The typical numeric scale to gauge pain is from 0 to 10, with 0 being no pain, and 10 being very severe, intolerable level of pain. The scale below explains the numbers.

Mankoski Pain Scale

0	Pain Free	No medication needed
1	Very minor annoyance-occasional minor twinges	No medication needed
2	Minor annoyance-occasional strong twinges	No medication needed
3	Annoying enough to be distracting	Mild painkillers are effective. (Aspirin, Ibuprofen)
4	Can be ignored if you are really involved in your work, but still distracting	Mild painkillers relieve pain for 3 to 4 hours
5	Can't be ignored for more than 30 minutes	Mild painkillers reduce pain for 3 to 4 hours
6	Can't be ignored for any length of time, but you can still go to work and participate in social activities	Stronger painkillers (Codeine, Vicodin) reduce pain for 3 to 4 hours
7	Makes it difficult to concentrate, interferes with sleep. You can still function with effort.	Stronger painkillers are only partially effective. Strongest painkillers relieve pain (Oxycontin, Morphine)
8	Physical activity severely limited. You can read and converse with effort. Nausea and dizziness set in as factors of pain.	Stronger painkillers are minimally effective. Strongest painkillers reduce pain for 3 to 4 hours.
9	Unable to speak. Crying out or moaning uncontrollably - near delirium.	Strongest painkillers are only partially effective
10	Unconscious. Pain makes you pass out.	Strongest painkillers are only partially effective.

Source: www.valis.com/andi/painscale

Brief Pain Inventory

The brief pain inventory scale uses a worksheet with 15 questions that ask the person to numerically rate the effect of your pain on categories such as how you relate with other people, walking, and sleep over the last 24 hours.

Date: _____ /_____ /_____ Time: _____

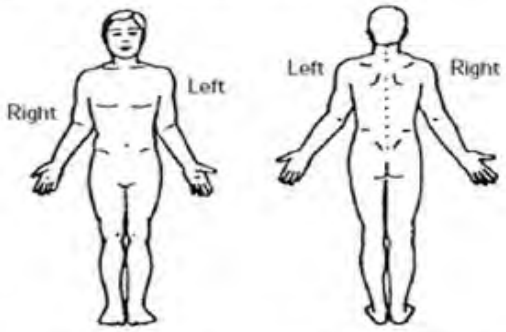
Name: _____ _____ _____

Last First Middle intial

1) Throughout our lives, most of us have had pain from time to time (such as minor headaches, sprains, and toothaches). Have you had pain other than these everyday kinds of pain today?

1. Yes 2. No

2) On the diagram, shade in the areas where you feel pain. Put an X on the area that hurts the most.



3) Please rate your pain by circling the one number that best describes your pain at its **worst** in the past 24 hours.

0 1 2 3 4 5 6 7 8 9 10
No pain — Pain as bad as you can imagine

4) Please rate your pain by circling the one number that best describes your pain at its **least** in the past 24 hours.

0 1 2 3 4 5 6 7 8 9 10
No pain — Pain as bad as you can imagine

5) Please rate your pain by circling the one number that best describes your pain on **average**.

0 1 2 3 4 5 6 7 8 9 10
No pain — Pain as bad as you can imagine

6) Please rate your pain by circling the one number that tells how much pain you have **right now**.

0 1 2 3 4 5 6 7 8 9 10
No pain — Pain as bad as you can imagine

7) What treatments or medications are you receiving for your pain?

_____

_____

8) In the past 24 hours, how much **relief** have pain treatments or medications provided? Please circle the one percentage that most shows how much relief you have received.

0% 10 20 30 40 50 60 70 80 90 100%
No relief — Complete relief

9) Circle the one number that describes how, during the past 24 hours, pain has **interfered** with your:

A. General activity

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

B. Mood

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

C. Walking ability

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

D. Normal work (includes both work outside the home and housework)

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

E. Relations with other people

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

F. Sleep

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

G. Enjoyment of life

0 1 2 3 4 5 6 7 8 9 10
Does not interfere — Completely interferes

Descriptor Differential Scale of Pain Intensity

- This scale uses 12 descriptors, such as faint, strong, intense and, very intense.
- Each word is placed in the middle of its own line with a plus sign at one end of the line and a minus sign at the other end.
- The person marks each line at the level of the word itself, or near the plus or minus sign to convey how well the word describes their pain.

TABLE 2.2. Descriptor Differential Scale of Pain Intensity (DDS-I)

Instructions: Each word represents an amount of sensation. Rate your sensation in relation to each word with a check mark.

Faint
(-) ---------- (+)

Moderate
(-) ---------- (+)

Barely strong
(-) ---------- (+)

Intense
(-) ---------- (+)

Weak
(-) ---------- (+)

Strong
(-) ---------- (+)

Very mild
(-) ---------- (+)

Extremely intense
(-) ---------- (+)

Very weak
(-) ---------- (+)

Slightly intense
(-) ---------- (+)

Very intense
(-) ---------- (+)

Mild
(-) ---------- (+)

Note. Reprinted from Pain, 35, R. H. Gracely and D. M. Kwilosz, The Descriptor Differential Scale: Applying psychophysical principles to clinical pain assessment, 279–288. Copyright 1988, with permission from Elsevier Science.

https://www.verywellhealth.com/pain-scales-assessment-tools-4020329?print

Non-Drug Pain Relief: Imagery

Relaxation helps lessen tension. One way to help decrease pain is to use imagery. Imagery is using your imagination to create a thought or image that will distract you from your pain. Imagery does not replace pain medicine. It works with your pain medicine to help you have better pain relief.

How Imagery Helps

Imagery is used to help reduce stress that can cause muscle tension. It can help relieve tense muscles that may add to the pain. Imagery can be thought of as a focused daydream that uses all of your senses – sight, touch, hearing, smell and taste. Certain images may reduce pain both during imagery and for a period of time afterward. You can imagine and revisit favorite spots in your mind to help you relax, relieve boredom, decrease anxiety and help you sleep.

Directions for Using Imagery

1. Find a quiet room where you can get into a comfortable position to relax. Close your eyes.
2. Do not fold your arms or cross your legs. You may cut off circulation and cause numbness and tingling.
3. Breathe in deeply. Exhale slowly as though you are whistling. Breathe deeply and exhale slowly three times. This will help you relax.
4. Picture in your mind something that is peaceful to you or a place that you have enjoyed visiting. This pleasant image should represent how you picture pain relief. For example:
   - Think of pain as a large boulder that is on a part of your body weighing you down and causing pain. Picture large helium-filled balloons attached to the boulder carrying it away from you and taking away the pain.

- Think of the pain as a thunderstorm with lightning and thunder that rains on your body. Imagine how the pain medicine is like a gentle breeze that blows the rain and thunderclouds away. Instead of rain and thunder, you have sunshine and warmth. The air smells clean and fresh, the rain has watered all the beautiful flowers and the grass is green and lush. There are swans and ducks on a pond. Only a ripple, caused by the ducks' gentle paddling, disturbs the water's peaceful state.

5. Use imagery at least 20 minutes a day.
6. It is best to try imagery before your pain becomes severe, or while you are waiting for your pain medicine to work.
7. If you are keeping a pain management log, write down your use of relaxation and imagery and how it works.

CellPress

Feature Review

Mental Imagery: Functional Mechanisms and Clinical Applications

Joel Pearson,[1,*] Thomas Naselaris,[2] Emily A. Holmes,[3,4] and Stephen M. Kosslyn[5]

Mental imagery research has weathered both disbelief of the phenomenon and inherent methodological limitations. Here we review recent behavioral, brain imaging, and clinical research that has reshaped our understanding of mental imagery. Research supports the claim that visual mental imagery is a depictive internal representation that functions like a weak form of perception. Brain imaging work has demonstrated that neural representations of mental and perceptual images resemble one another as early as the primary visual cortex (V1). Activity patterns in V1 encode mental images and perceptual images via a common set of low-level depictive visual features. Recent translational and clinical research reveals the pivotal role that imagery plays in many mental disorders and suggests how clinicians can utilize imagery in treatment.

Trends

Recent research suggests that visual mental imagery functions as if it were a weak form of perception.

Evidence suggests overlap between visual imagery and visual working memory – those with strong imagery tend to utilize it for mnemonic performance.

Brain imaging work suggests that representations of perceived stimuli and mental images resemble one another as early as V1.

Imagery plays a pivotal role in many mental disorders and clinicians can utilize imagery to treat such disorders.

Mental Imagery

Mental imagery has played a central role in discussions of mental function for thousands of years. Many have argued that it is one of the primary human mental events that allow us to remember, plan for the future, navigate, and make decisions. In addition, mental imagery plays a core role in many mental health disorders and plays an increasingly important role in their treatment.

We use the term 'mental imagery' to refer to representations and the accompanying experience of sensory information without a direct external stimulus. Such representations are recalled from memory and lead one to re-experience a version of the original stimulus or some novel combination of stimuli. Note that not all mental imagery need be voluntary; external events or internal associations also can trigger a mental image, even if one does not want to experience the image at that time [1]. Mental imagery can clearly involve all of the senses, but in this review we focus on visual mental imagery, given that most empirical work has addressed this sensory domain.

Historically, mental imagery research suffered for both practical and theoretical reasons. Methodological constraints caused by imagery's inherently private nature put practical limits on the types of mechanistic investigation that could be performed. Furthermore, the second half of the 20th century saw the rise of behaviorism in psychology. This theoretical orientation rejected the study of internal representations, including mental imagery. The combination of these two impediments is largely responsible for the comparative lack of mental imagery research relative to related topics such as visual attention and visual working memory [2].

Such constraints are now lifting, with increasingly sophisticated research techniques leading to many new discoveries about imagery. In recent years, new objective research methods have

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permitted more direct investigations into the mechanisms and neural substrates of mental imagery. Results from these methods shed light on mental imagery's role in perception, cognition, and mental health. Findings have cemented our understanding of visual mental imagery as a depictive internal representation with strong and unexpected ties to visual perception, effectively ending the so-called 'imagery debate' [3]. Moreover, studies reveal that mental imagery plays a pivotal role in clinical disorders such as anxiety. This upsurge in fundamental and clinical science regarding mental imagery is revealing the central role that mental imagery plays in everyday behavior as well as in human mental function and dysfunction.

Mental Imagery and Weak Perception

Much of the work on imagery and perception in the 1990s and 2000s revealed that imagery shares processing mechanisms with like-modality perception. For example, researchers showed that imagined visual patterns interact with a concurrent perceptual stimulus to boost sensory performance in a detection task [4]. Many studies converged in demonstrating that mental imagery could function much like afferent sensory perception. Imagining oriented lines can induce an orientation aftereffect [5] or imagining a moving stimulus can induce a motion aftereffect on a subsequent perceptual stimulus, much like normal perception [6].

Mental images can also take the place of perceptual stimuli during various types of learning. Perceptual learning typically involves repeated performance of a perceptual detection or discrimination task that leads to increases in performance. However, imagining the crucial components of such a task, instead of actually performing them on a perceptual stimulus, can also enhance performance on the perceptual task [7]. For example, when participants repeatedly imagine a vertical line between two perceptual lines they subsequently perform better in discriminating the distances between three perceptual lines [7]. Similarly, classical conditioning can occur with voluntarily formed visual imagery in place of perceptual stimuli [8]. In both of these examples the imagery-based learning is later tested with perceptual stimuli, which demonstrates generalization from the imagined to the perceptual content.

One important requirement in mental imagery research is to ensure that the effect of visual imagery on concurrent perception is not merely being driven by visual attention. Many studies have demonstrated that applying attention to a particular stimulus, or part of one, can change multiple dimensions of sensory perception. For example, attention alone can increase stimulus contrast, color, or coherency [9–11]. Studies using the '**binocular rivalry**' technique (see Glossary) have demonstrated contrasting effects of imagery and attention. When participants visualize one of two patterns, the imaged pattern has a much higher probability of being perceptually dominant in a subsequent brief single binocular rivalry presentation [2,12,13]. In other words, the content of the mental image primes subsequent dominance in binocular rivalry, just as a weak or low-contrast perceptual stimulus would do. Moreover, these effects grow stronger with longer image generation times, whereas increasing periods of applying attention to a particular stimulus does not modulate this priming effect [12]. Furthermore, particular experimental manipulations can attenuate the priming effect of imagery while leaving intact the effect of prior attention [12]. Thus, imagery can be dissociated from visual attention along at least two different dimensions.

An emerging consensus from multiple behavioral studies is that the influence of prior perceptual stimuli on subsequent perceptual tasks depends on the 'perceptual energy' or strength of the prior stimulus [12,14–16]. Facilitation is more likely if the preceding stimulus is short and/or low contrast, whereas suppression is more likely when a prior stimulus is high contrast and/or is shown for a long duration [12,14–16]. Hence, the facilitative effect of a prior stimulus increases as the strength or presentation duration increases until it reaches a tipping point, when the effect reverses and leads to reduced facilitation and increased suppression (Figure 1) [12,15,16]. Evidence suggests a single continuous mechanism that depends on the visual 'energy' of the

Glossary

Binocular rivalry: a visual phenomenon in which two different patterns are presented, one to each eye; the patterns compete for perceptual dominance, such that during continuous viewing awareness alternates between the two patterns.
Low-level visual features: in this context refers specifically to perceptual visual features such as color, spatial orientation, contrast, and spatial frequency; features of visual stimuli that are largely processed by the early visual cortex.
Multivariate pattern classifiers (MVPCs): also referred to as multivariate decoding; in fMRI, typically the use of spatial patterns (many voxels) to make a prediction or classification regarding some perceptual, cognitive, or behavioral state. The activation of multiple voxels from fMRI data is used as a pattern rather than averaging over a region of interest.
Retinotopic: refers to the mapping of information from the layout of the retina in the eye to the visual cortex. Cells in the visual cortex respond to stimulation of a specific part of visual space, such that two adjacent cells will respond to two adjacent stimuli hitting the retina.
Voxel: in fMRI, the smallest unit of measured data.

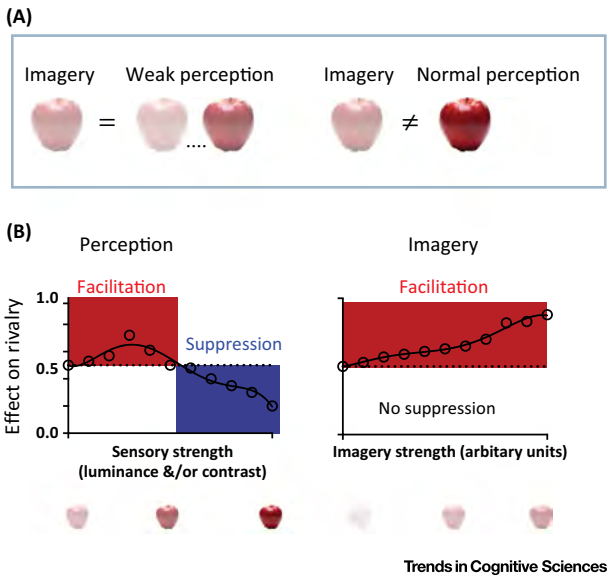

Figure 1. Imagery Resembles a Weak Version of Perception. (A) A useful way to conceptualize mental imagery is as a weak form of sensory perception. (B) A schematic illustration of the effects of prior perceptual stimuli at different strengths and of imagery on subsequent perception. The left graph shows hypothetical data for prior perceptual stimuli at different strengths (e.g., contrasts). Low-contrast prior stimulation facilitates subsequent detection [16] or binocular rivalry dominance [12,15], whereas high-contrast prior stimulation will induce a suppressive aftereffect. By contrast, on the right graph, imagery only facilitates subsequent perception. Overall, imagery acts much like weak perception. Schematic data plots are based on data from [12,15].

prior stimulus; that is, its sensory strength. Behavioral data thus far show that the effect of imagery on subsequent perception is limited to the facilitation range, and not suppression (Figure 1B, right panel) (for a review see [2]).

If mental imagery is conceived of as a type of top-down perception, visual features such as luminance or brightness should also be preserved in imagined representations and should have similar effects on physiology. Indeed a recent study demonstrated exactly that: the brightness of the imagined stimulus had a reliable and predictable effect on pupil constriction, as it does during perception [17].

In addition, brain imaging work has provided compelling evidence that visual mental images arise from activation of the same types of visual features that are activated during visual perception. Several studies have explicitly modeled the representations encoded in activity during perception and then used the model to decode mental images from brain activity. To our knowledge this explicit modeling approach – known as **voxel**-wise modeling and decoding (VM) – was first applied to mental imagery in 2006 [18]. This landmark study designed a voxel-wise model of tuning to **retinotopic** location (i.e., a receptive field model) and then used it to decode mental images of high-contrast blobs in various configurations. Consistent with the evidence for retinotopic organization in mental imagery [19], models of retinotopic tuning during visual perception could be used to identify and even reconstruct mental images of the stimuli (Figure 2).

It could be argued that imagining simple, blob-like stimuli that are optimized to engage area V1 is a special case of mental imagery that may be unrelated to the rich, complex imagery we generate and use daily. However, the representation of **low-level visual features** is conserved even when people visualize complex, multi-object scenes such as photographs and artwork [20]. A model that was tuned to retinotopic location, spatial frequency, and orientation [21] picked out mental images of specific works of art from among thousands of other randomly selected images – and even from other examples of the artists' own work. Performance was lower than that for actual perception, but still much better than that expected by chance. Thus, representations of retinotopy and spatial frequency – quintessential 'visual' representations – are encoded in activity even during rich and complex mental imagery.

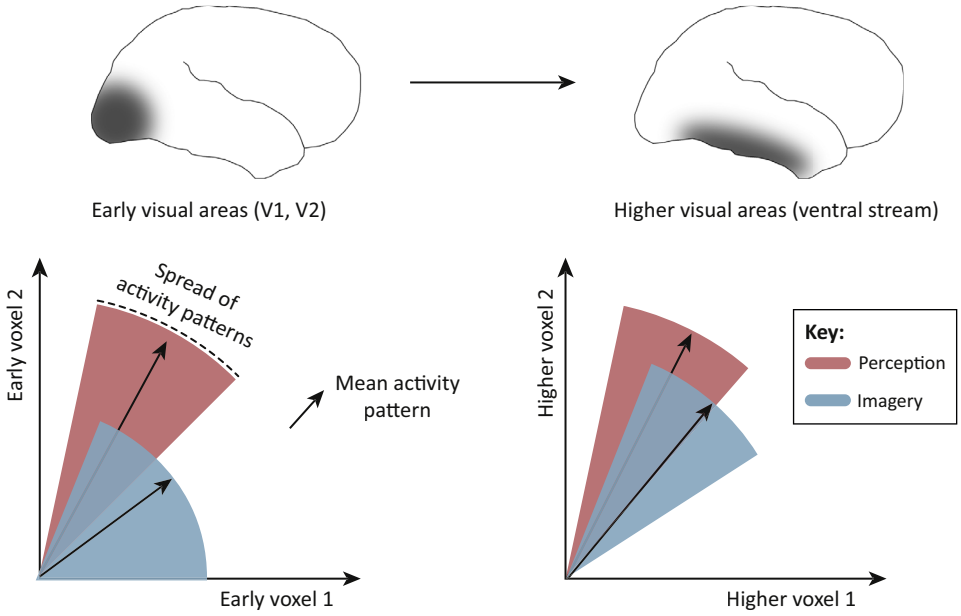

Figure 2. Activity Patterns Evoked by Visual Perception and Visual Mental Imagery Are Increasingly Similar with Ascension of the Processing Hierarchy. This diagram summarizes an organizing principle that is implicit in the fMRI literature on visual mental imagery. Here the ventral stream is coarsely grouped into early visual areas (shaded brain region, left panels) that represent low-level visual features (e.g., edges, textures) and higher-level visual areas (shaded region, right panels) that represent scene-level information and object categories. For purposes of illustration, we consider hypothetical multivoxel populations comprising just two voxels in the early visual cortex (left) and two voxels in the higher visual cortex (right). Activity patterns are represented as vectors in a 2D space in which the axes correspond to the two hypothetical voxels. In the early visual areas, activity associated with mental imagery has a lower signal-to-noise ratio (SNR) than activity associated with perception. This means that the mean activity vector (black arrow) evoked by visualizing a particular stimulus is shorter than the mean activity vector evoked by actually seeing a corresponding stimulus, while the spread of activity patterns around the mean activity vector (arc length) is larger. In the higher visual areas, the SNR associated with mental imagery is not as severely attenuated.

Furthermore, sensitivity to both perceptual orientation and location in visual space has been linked to the anatomy of V1 [22,23]. Likewise, mental imagery precision of spatial orientation and location in retinotopic space are both associated with the size of V1 [24]. In fact, the precision of both mental imagery and visual perception is correlated with the size of area V1, providing further support for the commonalties between the two.

Together, these studies show that activity patterns in the visual cortex are not merely similar across visual mental imagery and perception: activity patterns encode a common set of visual representations. When considered in light of the behavioral evidence reviewed above, these results lend further support to the conceptualization of visual mental imagery as a weak or noisy form of top-down perception that can in some cases take the place of bottom-up perception.

Mental Imagery and Visual Working Memory

Although mental imagery and visual working memory both involve the ability to represent and manipulate visual information, research on the two topics has diverged into two separate literatures that rarely reference one another [25]. Because of the different behavioral measures and tasks used, it has proved challenging to establish the degree of commonality between the two functions.

When participants in visual working memory experiments are asked to describe the strategies they use to complete the memory task, they tend to describe one of two different strategies. One involves creating a mental image to compare with the subsequent test stimuli [26–28]; the other strategy involves picking out particular details of a scene or array and encoding them phonologically or verbally, which is then compared with the test stimuli [26,28,29].

Recent behavioral work supports these subjective reports of different strategies [29,30] (but see [31]). This behavioral work directly compared the sensory strength of mental imagery and different measures of visual working memory. Individuals with stronger mental imagery had greater precision and higher capacity in visual working memory tasks, but not in iconic or verbal working memory tasks [29,30]. Furthermore, only those participants with strong sensory imagery were perturbed by the passive presence of uniform background luminance during visual working memory storage, but not in a verbal working memory task. Importantly, the creation of visual mental images is also perturbed by the presence of uniform passive luminance [12,32]. In addition, in a similar vein to visual imagery, the content of visual working memory can bias perception [33] and can facilitate detection in the neglected hemifield of visual extinction patients [34].

Taken together, these behavioral data suggest that those with relatively strong mental imagery utilize it to perform a visual working memory task whereas those with weaker imagery tend to rely on nonvisual strategies.

Brain imaging work has demonstrated overlap in the neural representation of visual working memory and mental imagery. For example, in one study [35], on some trials, participants were required to hold an oriented grating pattern in visual working memory until their memory performance was tested with a probe stimulus; on other trials, the same participants had to form and rotate a mental image of the same grating in accordance with a given cue. BOLD activity patterns in area V1 enabled accurate decoding of which pattern was being held in visual working memory and in the mental rotation (imagery) condition. When the classifier was trained on data from the working memory condition and then applied to decode data from the imagery condition, performance was just as high [35]. This generalization of decoding from memory to imagery is evidence for commonalities in the spatial pattern of BOLD activity during the two tasks. This in turn is evidence for representational overlap between mental imagery and visual working memory. Recent results also show that both visual working memory capacity and imagery strength and precision are associated with the surface size of V1 [24,36].

The combination of behavioral and brain imaging data shows that, despite clear task differences ('Hold this visual information in memory and we will subsequently test you on it' vs 'Create a mental image of this'), mental imagery and visual working memory can share common neural mechanisms in the sensory cortex. In many tasks, participants have to decide for themselves how best to maximize their memory performance. Depending on the 'mental tools' at hand, this might be with mental imagery or a propositional strategy. Recent work suggests that imagery strength and the neural networks underlying imagery may play a role in how individuals perform such tasks [37].

The key to unlocking the mechanistic relationship between visual imagery and visual working memory may lie in the individual differences across the population in visual representational strength, physiology, and even anatomy [36]. If a subset of the population tends to utilize imagery to aid memory performance, as the evidence suggests, whereas another subset of people who lack strong imagery utilize a different strategy, collapsing across these two groups could induce inconsistencies in visual working memory data. Separating participants into these groups, based the strength of their imagery, may be a good starting point for gaining clarity on the neural machinery used in visual working memory.

Graded, System-Wide Activation of Visual Cortex during Mental Imagery

The human visual system is a constellation of functionally distinct areas. These areas are conceived of as being organized in a hierarchy. Activation in areas toward the top of the hierarchy – so-called 'high-level' visual areas – is sensitive to changes in the semantic content of visual scenery and is invariant to visual detail. These areas are located in the ventral temporal lobe and representations encoded in the activity of these areas become increasingly abstract toward the anterior pole. Areas toward the bottom of the hierarchy – the 'early' visual areas – are located in the occipital cortex and are exquisitely sensitive to visual detail (e.g., retinotopic location, spatial frequency, edges).

Given this organization and the fact that early visual areas both send projections to and receive projections from high-level visual areas, many researchers have predicted that the role of the early visual areas in mental imagery is to flesh out visual detail.

Between 1993 and 2010, at least 20 studies attempted to test this hypothesis by using brain scanning to compare the amplitude of activity in early visual areas during visual mental imagery with the amplitude of activity during perception (or rest). Many studies reported no significant activity above baseline in the early visual cortex during mental imagery [38–45], but a slightly larger number of studies reported significant activity [46–57]. The discrepancy may be explained by differences in experimental factors [58] and variation in the vividness of mental imagery across individuals [55]. Meanwhile, the evidence for activation in high-level areas during imagery is uncontested; studies published over a decade from different groups have shown comparable levels of activity across visual mental imagery and visual perception in high-level visual areas [38,49,59,60]. Although activation in higher areas during mental imagery is more robust than in early visual areas, the vividness of mental images appears to be most tightly coupled to activity in the early visual areas [55]. See Box 1 for other evidence.

However, activation is only part of the picture. Recent studies using **multivariate pattern classifiers (MVPCs)** have shown that the same pattern classifiers that accurately discriminate stimuli by analyzing patterns of activity in visual areas V1 and V2 during perception of simple external stimuli can also discriminate the same stimuli during mental imagery [35,61,62]. This suggests that, although overall levels of activation in V1 during imagery are relatively low, the patterns of activity across imagery and perception in V1 and V2 are similar. This finding again supports the hypothesis of a shared representational format in imagery and perception.

MVPC studies have also revealed similarity in activity patterns across visual perception and imagery in high-level visual areas [59,60,63]. Consistent with activation studies, decoding performance is typically more robust in high-level areas than in early visual areas. Thus, MVPC studies of visual mental imagery support the claim that patterns of activity in perception and imagery become increasingly similar with ascension of the visual hierarchy (see Boxes 1 and 2).

Taken together, the MVPC and activation studies indicate that activity patterns associated with matched external and imagined stimuli begin to resemble one another as early as area V1. The resemblance increases with ascension of the visual hierarchy, although vividness of imagery appears to be most closely associated with early visual areas.

This general picture of how mental imagery engages the visual cortex satisfies many intuitions about how mental images are generated. For example, mental imagery is presumably based on the recall and recombination of memories. Because high-level areas are physically (and synaptically) closer to memory-encoding structures in the medial temporal lobe than are earlier visual areas, it makes sense that the activity patterns associated with perceived and imagined images should more closely resemble one another in high-level than in early visual areas. This may also

Box 1. Brain Damage Studies

Many studies have related measures of imagery to damage in particular parts of the brain. For example, in one study researchers [112] asked patients with unilateral neglect to imagine standing at either end of a well-known piazza in Milan, Italy. The primary symptom of unilateral neglect is that when patients look at a perceptual scene in front of them they tend to neglect one side of space. When asked to describe the piazza in their mind's eye, the patients described landmarks on one side of the square only. To ensure that this was not due to memory deficits, patients were asked to imagine the piazza from the opposite vantage point; the patients could describe the details of the previously neglected side, but now neglected the other side. This result was taken as evidence that imagery and perception share common neural processes at the level of attentional deployment.

Damage to the early visual cortex has also been diagnostic in the role of area V1 in imagery generation. In one study researchers were able to test the size of visualized objects both before and after a unilateral occipital lobe resection for epilepsy treatment [113]. Using a particular method that involves imagining an object and then walking toward it in the mind's eye until the object fills the entire visual field, and then reporting the distance between the individual and the object, researchers are able to infer the maximal image size. Here they found that after surgery the patient's maximal image size shrunk in the horizontal dimension compared with the image size before surgery.

Such data suggest that area V1 plays a functional role in visual mental imagery. However, other studies have demonstrated that it is possible to have intact and even vivid mental imagery, both behaviorally and when assessed using brain imaging, despite near-complete blindness due to cortical grey matter damage in the calcarine sulcus (V1) [114]. Hence, damage to V1 will impair mental images but, even with V1 almost completely gone, mental imagery remains possible (Box 2).

Thus, the results from studies of patients with brain damage are consistent with the results from fMRI and behavioral studies noted in the text: early visual areas can contribute to imagery, but other areas also play key roles. This inference is consistent with the idea that mental images, like visual percepts, rely on representations that are collaboratively constructed by visual areas at all stages of the visual processing hierarchy.

explain why the semantic aspects of mental images tend to be less ambiguous than visual details (e.g., we can know for certain that we are imagining a zebra and not a horse, even if we are not able to imagine the zebra's individual stripes). Lastly, it makes sense that the parts of the visual system responsible for visual detail should be most closely coupled to the visual vividness of mental images.

Box 2. Primary Visual Cortex and Mental Imagery

During visual perception, area V1 is distinguished both by its anatomical location and by the visual features that are encoded in its activity. This area is anatomically privileged because it is a gatekeeper of retinal information into the cortex. It receives more direct connections from the lateral geniculate nucleus than any other part of the visual cortex. However, during mental imagery its proximity to the retina does not make it special. The source of mental imagery is unknown, but it is likely that memory-encoding structures in the medial temporal lobe (MTL) and executive structures in the prefrontal cortex are critical. In addition, area V1 is distinguished by its representation of low-level visual features. Feed-forward models of perception treat these low-level features as the building blocks of object representation. However, feedback models treat them not as foundational for constructing representations of objects but as a tool for error-checking predictions about what objects are present in the immediate environment [115].

The anatomical importance of V1 during mental imagery may be derived from its topographical organization, which allows it to make explicit and accessible geometric properties that are only implicit in representations stored in long-term memory. In other words, the role of V1 in imagery may be determined by the kinds of inferences it allows to be drawn from a mental image. For example, if we want to infer whether a German shepherd dog has pointed or floppy ears [116] we may need to invoke a V1-like representation as a component of our mental image. If we simply want to infer whether an elephant is bigger than a mouse, it may suffice to invoke representations in any one of the many visual and/or parietal areas that are topographically mapped. This idea is perfectly consistent with findings that imply that the extent of V1 activation during mental imagery is task dependent.

In addition, the role of V1 in imagery is likely to vary enormously across individuals. Area V1 may make very different contributions to mental images for different people, depending on how important its representations are to the way in which each person imagines objects and/or scenes [117]. Recent research has documented that the size of area V1 predicts the sensory strength and precision of visual imagery [24]. Such relationships dovetail nicely with capacity limitation theories that propose an interaction between the content and anatomical restrictions due to the 2D layout of structures like V1, which support the representations [118].

Mental Imagery in Mental Disorders and Their Treatment

In a similar time frame to the burgeoning fundamental mechanistic investigations we have discussed so far, mental imagery has also been found to play a pivotal role in many mental and neurological disorders and their treatments. For example, intrusive, emotional mental imagery causes distress across a range of psychiatric disorders, from post-traumatic stress disorder (PTSD) and other anxiety disorders to bipolar disorder and schizophrenia [64]. However, those psychological therapies primarily based on verbal exchanges have historically neglected imagery, primarily focusing on the patient's verbal thoughts.

After a psychologically traumatic event, a significant proportion of people develop PTSD [65]. PTSD – characterized by re-experiencing the traumatic event through unwanted and recurring intrusive memories and nightmares – provides a hallmark illustration of clinically relevant mental imagery. An example of an intrusive memory is re-experiencing a vivid visual and auditory mental image of the moment a red car hit a child on the sidewalk. Such distressing images may be only fleeting and may occur only a handful of times per week, but their impact can be profound. The patient may avoid reminders of the traumatic event such as cars, children, or walking down a street and may feel a sense of current threat and a racing heart. These imagery-based memories are not a mere epiphenomenon of having PTSD but a cognitive mechanism driving the maintenance of the ongoing clinical disorder [66]. In other words, the intrusive images can strongly affect behavior and physiology.

Recent years have witnessed an explosion of research suggesting that mental imagery plays a role across a wide range of mental disorders [64,67–69]. Distressing and unwanted emotional imagery has been shown to occur in many mental disorders and the imagery content matches the core concern of people with the disorder. For example, a patient with arachnophobia (fear of spiders) may report imagery of large, hairy spiders with fangs. A patient with obsessive compulsive disorder (OCD) may have images of contaminated grubs boring into his skin and therefore feel dirty, fuelling the behavior of repeated washing. During conversation, a patient with social phobia (fear of public speaking) may experience concurrent imagery of how she appears to her conversational partner, envisioning herself as red and sweating. A patient with bipolar disorder may have future-oriented imagery and 'flash forward' to a suicidal act [70]. Conversely, in depression, people can report difficulties imagining a positive future [71] (see Figure 3).

The Clinical Relevance of Mental Imagery

Given the centrality of intrusive emotional imagery in such a wide variety of mental disorders, a basic understanding of mental imagery could prove instrumental in the development of new treatments. Potentially critical issues include the relative emotional impact of different representational formats, imagery 'realness', and the perceived likelihood of imagined events occurring.

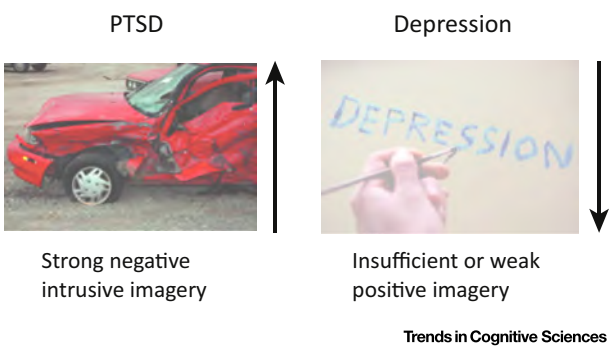

Figure 3. Imagery Is a Key Part of Symptoms in Mental Disorders – From the Intrusive Memories of Trauma in Post-traumatic Stress Disorder (PTSD) to the Lack of Positive Future Imagery in Depression. It presents a cognitive mechanism driving psychopathology, and thus imagery can also be targeted as a process – and harnessed as a tool – in psychological treatment.

Until recently, surprisingly little research had tested the relative impact of picture-like formats versus language-like descriptive formats on emotion; that is, mental imagery versus verbal thought (for an exception see [72]). Recent experiments support the hypothesis that, compared with verbal processing of the same content, mental imagery elicits stronger emotion. For example, in one experiment participants were given negative scenarios with instructions that promoted either verbal processing or mental imagery [73]. Imagery led to a greater increase in anxiety. When presented with positive scenarios, imagery again amplified (positive) emotion [74,75]. Such data are consistent with the finding that emotional memories have more sensory–perceptual features than do non-emotional memories [76].

Other imagery properties are also important. Compared with verbal thoughts of similar content, mental images are rated as more 'real' [77]. Many patients report that their imagery 'feels real' despite having the knowledge that they are not real, the images have a profound impact on their behavior. The apparent realness of clinical imagery seems to add to its power, influencing not only behavior and emotion but also beliefs. Hallucinations in schizophrenia are defined as mental experiences believed to be external percepts. Both schizophrenia [78] and Parkinson's disease [79] involve involuntary sensory hallucinations. In Parkinson's disease the degree of visual hallucination is well predicted by the sensory strength of an individual's voluntary mental imagery [79].

Repeatedly imagining a future event increases its perceived likelihood of occurrence [80]. This simulation heuristic effect also occurs for anxiety-provoking future events [81], increasing anxiety levels. Conversely, imagining an event that supposedly occurred in the past (even if it did not) inflates a person's confidence that the event actually did occur [82].

Imagined rehearsal of an action influences the likelihood that a person will complete that action [83]. Although such promotion of a behavior might be useful when actions are desired such as in sports psychology, one can see how its consequences can be maladaptive in psychopathology; for example, by increasing washing behavior in OCD. Similarly, imagery of a desired substance may contribute to cravings and thereby drive addictive behaviors [84,85]. In depression, imagining suicidal acts may even increase the risk of suicide [86]. Conversely, impaired ability to simulate positive future events is related to depression [87,88] (a disorder characterized by pessimism) whereas trait optimism is associated with greater ability to mentally simulate positive future events [119].

Mental Imagery in Clinical Treatments

These intriguing results on the emotional and behavioral impact of mental imagery offer insights into the development of new treatments for anxiety disorders. It is difficult to treat problematic emotional imagery with purely verbal discussion in therapy: to reduce imagery symptoms effectively, therapeutic techniques should include an imagery-focused component. Mental imagery techniques are currently used in some evidence-based treatments. For example, cognitive behavioral therapy (CBT) often includes 'imaginal exposure', which involves having the patient repeatedly imagine the feared object or context (e.g., contaminated hands) until his or her anxiety level subsides [89]. Imaginal exposure is a key technique, used across anxiety disorders.

Another technique, 'imagery rescripting' [68], aims to transform the imagery content. For example, in social phobia the negative outcome of mental imagery (e.g., performing badly) is changed to a new, more adaptive image such as performing competently [90]. 'Systematic desensitization' uses gradual exposure to images of feared objects or situations, whereby the imagery is paired with an incompatible response to the fear – such as physical relaxation – until the image no longer evokes negative emotion [91]. A form of therapy called 'eye movement desensitization and reprocessing' (EMDR) promotes lateral eye movements during the recall of

emotional memories; this technique appears to dampen the vividness and emotionality of imagery [92].

These imagery-focused therapeutic techniques reduce the powerful impact of dysfunctional imagery on emotion and/or reduce the frequency of associated intrusive imagery. It is noteworthy that imagery-focused CBT, as reviewed in clinical guidelines [120] has the strongest documented impact on treating PTSD and social phobia, with some trials showing success rates of up to 75%.

Future Mental Imagery Treatments

How might mental imagery research lead to future treatment innovations? First, we can import existing imagery techniques to clinical areas where imagery has been neglected. For example, treatments for bipolar disorder have shown little improvement since the discovery of lithium many decades ago. Perhaps advances can be made by leveraging the fact that people with bipolar disorder show high spontaneous use of imagery and intrusive imagery [70,93]. By considering the possible role of imagery in this disorder, new treatments could be devised by importing imagery techniques from those used to treat anxiety disorders. Another example is addressing hopeless, pessimistic future orientation by training patients with depression to generate more adaptive mental imagery and simulate future positive events. An initial randomized controlled trial including computerized positive imagery training in depressed patients showed some promising results [94] (though see also [71]), requiring further research.

Second, basic science studies of mental imagery may inform the development of new imagery treatment techniques by focusing on the depictive, pictorial format of imagery itself. For example, as discussed above, concurrent perception may interfere with image generation [2,12,95]. This finding is consistent with the fact that strategically applied visual tasks, such as the computer game Tetris, performed soon after an experimental trauma (in the time window for memory consolidation), reduce the frequency of intrusive images [96]; this technique has recently been extended to reconsolidation [97]. Such findings may open ways to prevent the accumulation of intrusive images, which is important because we need preventative treatments for PTSD [98]. Linking studies of emotional imagery with neural mechanisms may also be useful [99,100].

Overall, discoveries about mental imagery can contribute to our understanding of the cognitive and neural mechanisms underlying psychopathology and of which mechanisms to target in improving treatments [101]. Even the best treatments do not work for everyone and effective treatments are not yet available for all mental disorders. Science-driven mental imagery treatment techniques could greatly help and may even offer treatment innovations that look little like traditional talking therapies. Such treatments would capitalize on the principle that imagery involves a depictive format with its own set of properties [3] (Box 2).

Concluding Remarks

The many new methods touched on here not only offer new mechanistic insights into mental imagery but also offer new tools for future research. Recent work has demonstrated how imagery can 'stand in' for an afferent visual representation of an external stimulus. Specifically, mental images seem to behave much like weak versions of externally trigged perceptual representations. Functional brain imaging work supports the behavioral evidence by demonstrating that common sets of neural structures are employed during both events. Further, both representations seem to be encoded using a common set of basic visual features, which in many visual areas are organized topographically.

An increasingly important component of imagery research now and in the future is the translation of the fundamental science into the clinic. Clinical research shows that many different mental

Outstanding Questions

How do perception and mental imagery differ? There are clear phenomenological and epistemological differences between external perceptual and mental images, and patterns of activity measured during imagery and perception of the same stimulus are not identical. A first step toward answering this question will be to discover whether imagery-induced neural activity patterns are simply weaker or noisy versions of the activity during the perception of matched external stimuli or whether they encode systematically distorted representations.

Are individuals able to exploit the differences between mental imagery and perception?

How does mental imagery differ from other forms of top-down activity? Visual perception is heavily influenced by working memory, attention, and expectation. Clearly mental imagery is related to these disparate cognitive phenomena, but more work is needed to elucidate the networks and patterns of neural activity that distinguish mental imagery from these and other modes of cognition and perception.

Can mental imagery be involuntary, as clinical theory proposes? Or do individuals simply lack conscious awareness of the voluntary process (e.g., have poor metacognition)? The type of imagery prevalent in many mental disorders is typically described as involuntary, or not under the individual's control (see the clinical section). Little is known about the mechanisms that distinguish voluntary and involuntary imagery.

Can mental images be generated nonconsciously?

What functional mechanisms dictate individual differences in imagery strength?

There are many examples of visual illusions that create a conscious visual experience without a direct stimulus. Might the involuntary nature of such phantom perceptual experience offer a novel way to study the involuntary elements of imagery?

disorders involve symptomatic imagery, and incorporating imagery into behavioral treatments is proving beneficial. Bridges from fundamental research to emotional imagery will be critical for the systemic understanding of mental representations in dysfunction. Similarly, the characteristics and function of mental representations in everyday cognition will help form a fuller understanding of human mental events.

The main functions of mental imagery include simulating possible future scenarios and 'reliving' past experiences [83,102,103]. From this perspective, imagery should perhaps be studied not only in its own right but in many types of cognitive tasks. Beyond visual working memory, we know that imagery plays a role in affective forecasting [104], eye witness memory [105], making certain moral decisions [106], prior expectation templates to aid in predictable visual tasks [107], and facilitating emotion [108]. Mental simulations are now used to detect consciousness in vegetative state patients [109] and can be decoded using brain imaging during the early stages of dreams [110]. One interesting proposition is that all forms of cognition involve modality-specific mental simulations, known as embodied or grounded cognition [111]. Such theories imply that imagery plays a functional role in all cognitive events. It is exciting to begin to see the detailed, ubiquitous, and multifaceted role imagery plays in our everyday lives, both in function and dysfunction.

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PALLIATIVE CARE NETWORK OF WISCONSIN

FAST FACTS AND CONCEPTS #192
PALLIATIVE CARE FOR ADULTS WITH DEVELOPMENTAL DISABILITIES

Neil M Ellison MD and Drew A Rosielle MD

Introduction The life expectancy for adults with developmental disabilities (DD) is now within 5 years of the general population. This has resulted in an increased frequency of death from age related illnesses such as cancer, heart disease, and chronic lung disease. This *Fast Fact* will review special issues in the care of developmentally disabled people with life-limiting diseases. *Fast Fact* #193 will discuss decision making around life-threatening diseases for adults with DD.

Background Adults with DD may be defined as persons with impaired social functioning and decreased ability (mild to profound) to comprehend new or complex information or to learn new skills. These disabilities begin prior to age 18. Examples include people with autism, Down syndrome, and cerebral palsy associated mental retardation. During the past decades there has been a move towards decreased institutionalization with mainstreaming of persons with DD to the community. Home care is often provided by elderly parents. Community housing may be supervised by individuals with little familiarity with the patient.

Barriers to medical and palliative care for adults with DD

- Suboptimal nutrition, limited exercise, decreased utilization of health screening often negatively impact developmentally disabled people's health.
- Communication barriers can lead to more advanced illness presentation.
- Lack of clarity of goals of care and poorly defined decision-makers (see *Fast Fact* #193).
- Developmentally disabled people may not be allowed appropriate bereavement:
    - Deprived of the knowledge of death of their caregivers and loved ones.
    - Excluded from funerals, memorial services, or other bereavement activities.
- Patient lack of comprehension of their illness, its symptoms, or its treatments:
    - May interpret illness or treatments as punishment for wrong-doing.
    - May not be able to understand death and why their family/caregivers are sad around them.
- Symptom assessment may be compromised by an inability to communicate:
    - Individuals demonstrate a wide range of behaviors indicating discomfort, some of which may be subtle and only apparent to people who know them well, if at all.
    - Signs of distress may be apparent even though it is unclear what is causing the distress: pain, other somatic symptoms, anxiety/fear, sadness?

Providing effective palliative and supportive care to adults with DD

- Work closely with caregivers to maximize time in familiar surroundings, with familiar people and objects, in identifying routines and activities enjoyable to the patient, and in ways to offer emotional reassurance.
- Communicate about symptoms understandable to the patient, as opposed to abstract diseases.
- Symptom assessment and management (see also *Fast Fact* #126):
    - Research has supported the finding that symptom assessment needs to be individualized to the patient, based on the experience of a patient's closest caregivers.
    - DisDAT – a distress assessment tool – has been developed to help clinicians and caregivers identify, communicate about, and document an individual's signs of distress and contentment (3,4).
    - Symptom management should focus on careful examination of patterns of distress, a thorough physical examination, judicious use of diagnostic testing, and empiric use of comfort medications based on what is felt to be the most likely cause. Trial and error, with constant monitoring of adverse medication effects, is often necessary.

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Seizure

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Continuing Education Activity

A seizure represents the uncontrolled, abnormal electrical activity of the brain that may cause changes in the level of consciousness, behavior, memory, or feelings. Convulsive concussion, convulsive syncope, movement disorders, rigors, sleep-related events, or psychogenic non-epileptic spells are all in the differential diagnosis of an event. Seizures can classify as partial or generalized. In a partial seizure, the most common seizure type in adults, one area of the cortex activates first and may manifest through simple symptoms such as a motor or sensory phenomena. Generalized seizures result from diffuse cortical activation at seizure onset or generalization of partial seizure activity. This activity discusses the evaluation and management of seizures and explains the interprofessional team's role in improving outcomes for patients with this condition. This activity examines when this condition should be a consideration on differential diagnosis and how to evaluate it properly. This activity highlights the role of the interprofessional team in caring for patients with this condition.

Objectives:

- Describe the pathophysiology of seizures.
- Review appropriate workup of a patient with seizures.
- Summarize drug treatment options for continuing seizures.
- Explain the importance of improving care coordination among the interprofessional team in enhancing the delivery of care for patients with seizures.

Access free multiple choice questions on this topic.

Introduction

Paroxysmal spells might represent events originating from the central nervous system, cardiac disturbances, psychiatric causes, or might be from other etiologies. Syncope, convulsive concussion, convulsive syncope, rigors, movement disorders, sleep-related events, and psychogenic nonepileptic seizures are all in the differential diagnosis of a transient event with movements. Epileptic seizures constitute one type of paroxysmal event.[1]

An epileptic seizure is a transient occurrence with signs or symptoms due to abnormal excessive and synchronous neuronal activity in the brain. There are many different types of seizures. Current classification designates two large categories - partial or generalized. In a partial seizure, one area of the cortex is thought to be activated initially and may show simple symptoms such as a motor or sensory phenomena. Partial seizures may rapidly secondarily generalize and spread to involve all cortical areas. Generalized seizures result from diffuse cortical activation at seizure onset. The most common seizure type in adults is partial-onset seizures with rapid secondary generalization.[2]

Seizures with dyscognitive features, also known as complex partial seizures, are associated with altered awareness or consciousness. These may have minimal motor manifestations such as lip-smacking or small amplitude extremity movements and may present as an isolated confusional state.

Epilepsy, by definition, is a condition of recurrent unprovoked seizures. Determining whether a first seizure or recurrent seizures are provoked or unprovoked is fundamentally essential for diagnosis and treatment.

Epileptic syndromes serve to condense clinical information into useful nomenclature. Localization-related is used in this terminology to indicate seizures that arise from pathology in a localizable brain area. Idiopathic epilepsy is associated with no symptoms other than seizures. In symptomatic epilepsy, seizures reflect underlying identifiable brain disease. Cryptogenic refers to seizure disorders suspected to be symptomatic of underlying brain disease but are without definitive proof of the underlying cause. Specialists usually diagnose an epileptic syndrome.[3]

Status epilepticus is defined as an enduring epileptic condition. There are as many types of status epilepticus are there are types of seizures. Generalized convulsive status epilepticus is a medical emergency. Current definitions define status epileptics as a single generalized convulsion lasting greater than five minutes or a series of generalized seizures without full return of consciousness.[4]

Etiology

Seizures may be either provoked or unprovoked. Provoked seizures, also known as acute symptomatic seizures, may result from electrolyte disorders, toxins, head injury, infectious processes, vascular anomalies, tumors or other mass lesions, and many other causes. A listing of provoked causes of seizures is lengthy and could include complications of almost any disease process. Some common causes are listed below:

- Electrolyte disturbances (hypoglycemia, hyponatremia, hypernatremia, hypocalcemia, others)
- Acute toxic effects (antidepressants, sympathomimetics, others)
- Withdrawal syndromes (ethanol, benzodiazepines, others)
- Irregularity with prescribed antiepileptic medications
- Sepsis
- CNS infections
- Hypoxic brain injury
- Traumatic brain injury
- Stroke ischemic or hemorrhagic
- Neoplasm
- Inflammatory (lupus cerebritis, anti-NMDA receptor encephalitis, others)
- Fever
- Sleep deprivation

Epilepsy occurs because of a predisposition to seizures from genetic susceptibility or a chronic pathologic process. By definition, unprovoked seizures occur in the absence of provocative causes or more than seven days after an acute injury or insult such as stroke or brain hemorrhage. Recurrent unprovoked seizures define epilepsy.

Of patients in United States general hospitals presenting with generalized convulsive status epilepticus, roughly one-fourth are patients with epilepsy with breakthrough seizures, medication irregularity, or new-onset epilepsy; one-fourth

are patients with ethanol-related seizures, and one-half are patients with seizures that are provoked by a variety of medical conditions.[5]

Epidemiology

The age-adjusted incidence of epilepsy in North America ranges between 16 out of 100,000 and 51 out of 100,000 person-years. The age-adjusted prevalence ranges from 2.2 of 1000 to 41 of 1000, depending on the reporting country. Partial epilepsy may constitute up to two-thirds of incident epilepsies. Incidence increases in lower socioeconomic populations.[6]

About 25% to 30% of new-onset seizures are thought to be provoked or secondary to another cause.

Epilepsy incidence is highest in younger and older age groups and increases steadily after 50 years of age. The most common cause of seizures and epilepsy in older people is cerebrovascular disease.[7]

Pathophysiology

Everyone has some propensity to have seizures. The concept of a seizure threshold means that each individual exists on a seizure susceptibility continuum with many factors influencing that susceptibility. Medications, genetic factors, electrolyte abnormalities, sleep state, infections, brain inflammation, or injury from many causes may lead to an individual crossing that threshold with a resulting seizure.

On a cellular level, seizures start with the excitation of susceptible cerebral neurons, which leads to synchronous discharges of progressively larger groups of connected neurons. Neurotransmitters are undoubtedly involved. Glutamate is the most common excitatory neurotransmitter, and gamma-aminobutyric acid (GABA) is an important inhibitory neurotransmitter. An imbalance of excess excitation and decreased inhibition initiates the abnormal electrical activity. These electrical paroxysmal depolarization shifts (PDS) seem to trigger epileptiform activity. Increased activation or decreased inhibition of such discharges could result in seizures. The part of the brain affected often reflects in the clinical signs or symptoms of the seizure.[3]

Generalized convulsive status epilepticus is accompanied by systemic changes of lactic acidosis, increased catecholamine levels, hyperthermia, respiratory compromise, and other systemic alterations.[8][9][10] However, the ongoing excessive electrical activity that occurs with status epilepticus is damaging to the brain.[11] There is an evolution of generalized convulsive status epilepticus from continuous or discrete seizures to a condition of minimal or no motor activity. The electrical activity reflected by EEG evolves as well.[12] The result may be a type of nonconvulsive generalized status epilepticus.

History and Physical

As with many medical conditions, history is key in assessment and will guide further evaluations. The first question posed to the caregiver is whether the event was a seizure or some other type of transient event. A sudden alteration in consciousness with associated motor movements is the common description of a convulsive seizure. For generalized seizures with associated motor movements, the convulsion typically has a stiffening or tonic phase followed by clonic movements - rhythmic phased motor movements. There may be a noise or cry at the onset of the seizure. Some patients will describe a prodrome or aura before the event. Urinary incontinence may or may not be present. Tongue biting, if present, is most frequently lateral. Following a generalized tonic-clonic seizure, patients will have some transient alteration consciousness referred to as the postictal state. There are many types of seizures other than generalized convulsions, and any transient alteration of consciousness or unusual behavior or individualized perception might conceivably represent a type of seizure.

Key historical points include history with attention to the history of seizures, medication use, past medical history, and social history, especially any history of alcohol or illicit drug use. Any history of immunosuppression or malignancy is critical to discover. Frequently there will be a history of unresponsive spells that, in retrospect, might be seizures. Events

leading up to the seizure are quite important, and friends, family, or coworkers may have crucial historical information. For the patient with known epilepsy, an obvious question would be to ask if there has been any irregularity with medication use.

Physical examination should include a general physical examination and a neurologic examination with attention to the detection of any focal deficits. If someone observes the convulsion, they may see open eyes, no response to verbal or painful stimulation during the event, and rhythmic in-phase motor movements are consistent with a generalized tonic-clonic seizure.

For patients with a suspected seizure and persistent alteration in consciousness, the possibility of transformed status epilepticus should merit consideration. Sometimes termed subtle status epilepticus, the motor movements of this type of nonconvulsive status epilepticus may only be nystagmic eye movements, facial twitching, extremity twitches, or in some cases, no motor movement at all.[12][13]

Evaluation

Further clinical evaluations are guided by history and physical examination. If the clinician believes that the event is a seizure, the first question is whether it is provoked or unprovoked. Typically laboratory work, including electrolytes, is obtained. Lumbar puncture should merit consideration in patients with fever, a history of immunosuppression, or other factors suggesting possible central nervous system infection.

Neuroimaging is often obtained and is of higher yield based on historical factors or focal findings on the neurologic examination. Imaging is a recommendation whenever there is suspicion of an acute intracranial process in patients with a history of acute head trauma, history of malignancy, immunocompromise, fever, persistent headache, anticoagulation use, age older than 40 years, or focal seizure onset.[14][15]

For a healthy adult patient who has returned to baseline normal neurologic status who has apparently had a first seizure, determining serum glucose and sodium is recommended. Pregnancy testing is a recommendation in women of childbearing age.[1] Commonly additional labs and neuroimaging are necessary.

Electroencephalography (EEG) is a biomarker for epilepsy. Focal or generalized epileptiform discharges constitute the EEG hallmark of seizure activity. Frequently EEG is obtained as a risk-stratification tool for a patient with a seizure of possibility of seizures. Should the EEG show epileptiform or other abnormalities, management might change.

Persistent alteration of consciousness or continuing seizures will dictate additional testing such as neuroimaging and other serologic tests. If nonconvulsive status epilepticus is a consideration, arrangements for neurologic consultation and EEG are in order.[1]

Treatment / Management

Patients with reversible causes of seizures, such as hypoglycemia, may be discharged after appropriate interventions and with consideration for a safe home environment. For patients with a history of epilepsy who have returned to baseline mental status, adjusting the medication regimen and follow-up with other providers may be necessary. Testing for medication levels may be appropriate if available for the particular antiepileptic drug. If the patient has been noncompliant with an antiepileptic drug regimen, medications should be resumed.

Patients with alcohol withdrawal seizures represent another group of patients who may be discharged after appropriate treatment and a period of observation. Treatment of alcohol withdrawal seizures deserves special mention since episodic treatment with lorazepam has shown to decrease the risk of recurrence.[16]

A first unprovoked seizure in an adult who has returned to a normal neurological baseline often does not require initiation of medical treatment.[1] Caution regarding engaging in potentially hazardous activities involves discussion with the patient until follow-up, additional testing, and reassessment occurs.

If deciding to start drug therapy, many medications are options to treat a chronic seizure disorder or epilepsy as first-line medication or adjunctive medications. Selection may be guided by side effects and in consultation with a neurologist. They can be grouped based on their mechanism of action and include sodium channel blockers (carbamazepine, oxcarbazepine, eslicarbazepine, phenytoin, fosphenytoin, lamotrigine, lacosamide, and zonisamide), and agonists of GABA receptor (benzodiazepine and barbiturates). Other drugs with associated mechanisms include GABA reuptake inhibitors (tiagabine), inhibitors of GABA-transaminase (vigabatrin), glutamate antagonists (topiramate, felbamate, perampanel), medications with binding to synaptic vesicle 2A protein (levetiracetam, brivaracetam), and drugs with multiple mechanisms (gabapentin, pregabalin, valproic acid).

For the patient with generalized convulsive status epilepticus, immediate treatment of the seizures should begin while stabilization and other diagnostic procedures commence. Supportive care with attention to airway, breathing, and circulation issues are vital. Benzodiazepines such as diazepam, midazolam, or lorazepam are acceptable as the first-line medications for continuing seizures. Recommended dosage varies, but accepted regimens for adults are listed below.[17] [18] Respiratory depression is a common side effect, and patients will need careful monitoring. Underdosing of benzodiazepines is common, and the provider should be certain that there has been an adequate dose of a benzodiazepine given before adding additional medications.[19]

- Lorazepam 4 mg IV; repeat once in 5 to 10 minutes if seizures continue
- Midazolam 10 mg IM or IV; repeat once in 5 to 10 minutes if seizures continue
- Diazepam 10 mg IV; repeat once in 10 minutes if seizures continue

The best second-line medication is unclear even after completing a highly anticipated randomized trial of benzodiazepine refractory status epilepticus- the established status epilepticus treatment trial (ESETT). Second-line medications include fosphenytoin, valproate, levetiracetam, and others. Doses used in the ESETT study are listed below, given with an infusion time of ten minutes. The dosing of these medications in this study was higher than doses typically used in clinical practice. Clinicians have noted similar incidences of adverse effects with these medications, and no one drug was superior to the others.[20]

- Fosphenytoin 20PE/kg (up to 1500 phenytoin equivalents)
- Valproate 40mg/kg (up to 300 mg)
- Levetiracetam 60/mg (up to 4500 mg)

Should generalized convulsive status epilepticus continue, often advanced airway management is necessary. Blood pressure support may be necessary. The best treatment for refractory status epilepticus is unknown, but options include propofol, barbiturates (pentobarbital), or continuous benzodiazepine infusions in addition to other anesthetic medications. ICU admission will be necessary with continuous EEG monitoring.[18][21][22][23][24]

Differential Diagnosis

Spells resembling seizures stem from many different processes. One key differentiation is between a syncopal event and a seizure. Both events have an abrupt onset, but a syncopal event often has a provocative cause, the loss of consciousness is brief, and return to full consciousness is prompt without a confusional state. Incontinence may be present with either type of event. At times syncope is associated with motor movements mimicking a seizure.[25]

Another distinction often involves distinguishing seizures from psychogenic nonepileptic seizures. Please see that chapter for further information.

A partial of seizure mimics follows:[26]

- Syncope, convulsive syncope

- Psychogenic nonepileptic seizures
- Convulsive concussion
- Movement disorders
- Sleep-related movements
- Convulsive concussion

Prognosis

The prognosis of patients with seizures depends mostly on any underlying cause. Patients with seizures from remedial medical or toxicologic causes should do well with the management of those issues.

In other patients with acute symptomatic seizures, the prognosis is related to the disease process. Obviously, as a group, patients with neoplastic causes of seizures or hypoxic brain injury will not fare well compared to many patients with metabolic causes of seizures.

The prognosis of a patient with a single unprovoked seizure has been well delineated. Unprovoked seizures, by definition, have no established cause after clinical evaluation. If basic investigations, including appropriate laboratory work, imaging, and perhaps EEG, are unremarkable, estimates of the recurrence rate of another unprovoked seizure within five years are between one-third and one-half. However, if there is a second or third unprovoked seizure, the risk of further seizures increases to about three-quarters.[27][28]

Complications

In addition to using antiepileptic medications, the treatment of seizures aims at correcting any identifiable causative process. Care is needed to prevent any secondary brain injury, which may include advanced respiratory and cardiovascular support. Monitoring is necessary to detect hypotension and hypoxia and steps taken to correct those conditions when recognized.

Common complications may include traumatic injuries such as tongue lacerations or scalp lacerations.[29]

Convulsive status epilepticus leads to brain damage on the cellular level and may itself be epileptogenic. Transformed or subtle generalized convulsive status epilepticus or nonconvulsive seizures detected by EEG monitoring in critically ill patients may also contribute to brain injury.[30][31]

Deterrence and Patient Education

As with any medical condition, prevention is preferable to reactive interventions. Some conditions leading to provoked seizures may permit interventions before the development of a seizure. Clearly, with provoked seizures related to alcohol withdrawal or drug abuse, efforts should be made for appropriate actions pertaining to those disorders.

One study of patients presenting to emergency departments with seizure-related complaints found that roughly two-thirds of those with antiepileptic drug levels obtains had sub-therapeutic levels.[29] Counseling regarding medical compliance in patients with epilepsy and avoiding any triggers such as sleep-deprivation are crucial.

Patents with spells or seizures of unknown etiology should be counseled not to drive or operate dangerous machinery. Reporting requirements vary state-to-state.

Pearls and Other Issues

Most generalized seizures terminate in less than five minutes, and a seizure of longer duration or serial seizures without regaining full consciousness in between defines status epilepticus. Whether status epilepticus is from provoked causes or unprovoked causes, initial treatment is similar.

A benzodiazepine such as diazepam, midazolam, or lorazepam is accepted as first-line medications. Underdosing is common when compared to guideline recommendations. Side effects are primarily respiratory depression and primarily related to the rate of administration. However, underdosing benzodiazepines may contribute to reduced efficacy, potentially resulting in prolongation of status epilepticus.[19]

Outcomes following generalized convulsive status epilepticus depend on any underlying cause of the seizures and the duration of the status epilepticus.

For patients with seizures who require endotracheal intubation for airway management, neuromuscular blockade with paralysis will obscure signs of seizures. Stat EEG, if available, is recommended. If in doubt, medication administration with the presumption that seizures are continuing seems prudent in the short term.

Enhancing Healthcare Team Outcomes

Clear communication between interprofessional team members is essential since patients' clinical status may abruptly change. This interprofessional team includes primary care clinicians (including PAs and NPs), specialists (e.g., neurologists), nurses, mental health specialists, and pharmacists, who must operate collaboratively as a unit and share open communication regarding the patient's condition to achieve optimal outcomes. [Level 5]

Most patients will have a single, brief, uncomplicated event and return to full consciousness. Detection of any underlying cause of the seizure or seizures is important so that appropriate therapy or counseling is available.

There are many causes of seizures, but regardless of the cause, the basic initial treatment is similar. Seizure patients all require supportive care and assessment of airway, breathing, and circulation with appropriate interventions. Other interventions, including medications and critical care interventions, may be necessary for patients with a prolonged seizure or continuing seizures. Communication and coordination with other health care providers are essential to optimize team response.

Review Questions

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Sudden Unexpected Death in Epilepsy

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Continuing Education Activity

Sudden unexpected death in epilepsy (SUDEP), defined as "death in a patient with epilepsy that is not due to trauma, drowning, status epilepticus, or other known causes but for which there is often evidence of an associated seizure," represents a leading cause of death in patients with epilepsy. This activity reviews the definition, epidemiology, physiology, risk factors, and treatment of sudden unexpected death in epilepsy (SUDEP) and highlights the role of the interprofessional team in stratifying and mitigating the risks of SUDEP in patients with epilepsy.

Objectives:

- Describe the definition of sudden unexpected death in epilepsy.
- Identify the modifiable risk factors for sudden unexpected death in epilepsy.
- Summarize the findings leading to a diagnosis of sudden unexpected death in epilepsy.
- Review strategies employed by an interprofessional team to modify sudden unexpected death in epilepsy risk in high-risk patients.

Access free multiple choice questions on this topic.

Introduction

Epilepsy, defined as the tendency for unprovoked seizures, is a common neurologic disease, affecting up to 1 in 26 individuals.[1] While the spectrum of comorbidity varies greatly among patients with epilepsy, there is an overall increased mortality risk in this population. Causes of death in individuals with epilepsy include non-epilepsy-related conditions such as suicide, cancers, and cardiovascular disease as well as epilepsy-related causes, including status epilepticus, anti-seizure drug effects, motor vehicle collisions, and drowning.[2]

Sudden unexpected death in epilepsy (SUDEP), is defined as “death in a patient with epilepsy that is not due to trauma, drowning, status epilepticus, or other known causes but for which there is often evidence of an associated seizure,” and represents a leading cause of death in patients with epilepsy.[3] SUDEP accounts for approximately 38% of deaths in patients with epilepsy and approximately one death per 1000 patients with an epilepsy diagnosis.[4] As SUDEP is often unwitnessed, diagnosis is typically made post-mortem. Efforts to distinguish SUDEP from other causes of mortality in epilepsy have led to the sub-categorization of SUDEP into four major categories: definite, probable, possible, and unlikely SUDEP.[3] These categorizations have allowed for more precise investigation regarding the risks, mechanisms, and preventative strategies. Increasing awareness of SUDEP amongst health professionals and the general public can allow for improved patient counseling and potential modification of risk factors.

Etiology

Several potential mechanisms have been studied with regard to the pathogenesis of sudden unexpected death in epilepsy. Retrospective observational patient data, hospital data from patients with a sudden unexpected death in epilepsy in hospital epilepsy video monitoring units as well as eye witness accounts have identified respiratory abnormalities, cardiac arrhythmia, and attenuation of cerebral function as commonly observed peri-ictal phenomena.[5] Multiple studies have evaluated respiratory factors that may contribute to SUDEP. In a majority of reported SUDEP cases, the patient was discovered in a prone position, likely during sleep, suggesting suffocation.[5]

Cardiac arrhythmias have been identified, as well. However, signs of respiratory abnormalities (apnea, abnormal breathing patterns, hypoxia) are more commonly associated with brain dysfunction and were more commonly observed in witnessed and monitored cases of SUDEP. As the heart rhythm is generated independent of brain function, cardiac arrhythmia present in cases of SUDEP may suggest a secondary mechanism of the disease, such as channelopathy.[6] Furthermore, in cases where video electroencephalography (EEG) monitoring data was available, clinically significant arrhythmias were not identified.[5] Cerebral suppression has also been suggested as the primary event in a cascade terminating in SUDEP. Generalized suppression of brain function includes suppression of respiratory centers in the brainstem, resulting in severe acidosis. This suppression will also result in hypercapnia and acidosis, as well as laryngospasm.[7]

Epidemiology

Sudden unexpected death in epilepsy has been reported in many populations, from young children to the elderly. The highest prevalence of SUDEP is in people between the ages of 20 and 45.[8][9] A higher odds ratio for SUDEP has been reported in patients seen in an epilepsy center, as well as those with refractory epilepsy, those cared for in a residential care facility, and patients referred for epilepsy surgery. There is a higher odds ratio for SUDEP in males (OR = 1.42) and in people with childhood-onset epilepsy.[10]

Large-scale studies from the United States of America (USA) and Europe have identified higher rates of SUDEP in populations with socioeconomic barriers to care, including lack of employment, lack of access to medications and other treatments, and increased distance from appropriate healthcare providers.[9] SUDEP has been described in a variety of epilepsies, including generalized and focal epilepsy types, but based on recent population-based studies, an increased risk has not been observed in patients purely with absence or myoclonic seizures.[11]

Pathophysiology

Although the exact mechanisms of sudden unexpected death in epilepsy are not clear, multiple mechanisms have been proposed, including electroencephalographic suppression (arousal dysfunction) after a generalized seizure, secondary cardiac arrhythmias (bradycardia, and asystole), and postictal apnea.[12] The generally accepted "final common pathway" for SUDEP is profound cardiopulmonary depression. Inpatient peri-ictal monitoring has provided valuable data regarding physiologic changes that occur in the peri-ictal period, including oxyhemoglobin desaturation (as low as 40%) and severe bradycardia.[8] Various hypotheses have been proposed which implicate postictal dysfunction of arousal centers in the brainstem, leading to secondary hypoventilation. The combination of hypercapnia and hypoxia accompanying postictal hypoventilation would then potentially contribute to cardiac dysfunction and then death.[5] Various risk factors, including channelopathies or genetic mutations causing epilepsy, are thought to increase a patient's susceptibility to this process.

Histopathology

Although the proposed mechanism of sudden unexpected death in epilepsy involves cardiorespiratory failure secondary to a seizure, relatively little is known regarding specific histologic changes occurring in patients who died from SUDEP. Identification of specific histochemical markers and other post-mortem findings may help to distinguish SUDEP from

death from alternative causes. Physical examinations of patients with SUDEP have revealed oral trauma (secondary to biting of the lip or tongue), as well as pulmonary edema.[13]

Gross examination of the brain may reveal findings consistent with hypoxia, including mild gyral flattening consistent with brain edema; however, a significant mass effect has not been identified in SUDEP cases.[13] Various immunohistochemical staining methods have been employed to look for markers of inflammation, gliosis, disruption of the blood-brain barrier, and neuronal injury in patients with SUDEP vs. patients with non-epileptic sudden death. Such markers, including CD-163, HIF-(I-alpha), and HLA-DR reactivity in the medulla, hippocampus, and amygdala, have not shown significant differences in staining patterns compared with non-SUDEP controls.[7]

History and Physical

A number of historical factors have been associated with an increased or decreased incidence of sudden unexpected death in epilepsy. For example, the presence of generalized tonic-clonic seizures (GTCS), specifically greater than 3 in a year, are associated with 10 and 15-fold risk increases for SUDEP, respectively.[14] Furthermore, despite evidence that the addition of anti-seizure drugs in refractory epilepsy has a diminishing benefit with regard to seizure control, failure to add an anti-seizure drug increases the odds of SUDEP 6-fold.[14] In addition, a history of comorbid developmental delay and early-onset epilepsy have been identified as additional significant risk factors in the pediatric population.[15] Few factors are strongly associated with a lowered odds ratio for SUDEP. Two such factors include the presence of another individual in the bedroom who could act as an observer, as well as the use of a monitor/listening device in the room of the child or adult at risk for SUDEP.[14] Although most cases of SUDEP are unwitnessed, systematic evaluation of SUDEP cases occurring in epilepsy monitoring units has allowed for increased patient data collection in the peri-ictal period, in turn allowing for easier identification of risk factors.[10]

The American Academy of Neurology (AAN) 2017 Practice Guidelines on SUDEP studied over 20 other risk factors for SUDEP, including sex, age of seizure onset, a specific type, dose and duration of antiepileptic drug usage, type of epilepsy, comorbid medical and psychologic disorders, imaging and EEG characteristics. None of these were strongly associated with an increased or decreased risk for SUDEP.[14]

Additional SUDEP risk factors under investigation include the time of day during which the seizure occurs as well as physiologic changes that occur during different times of the day and body position (prone vs. supine).[16] Other non-modifiable risk factors include severe epilepsy as well as early age of onset of epilepsy and genetic factors, including pathogenic variants of potassium and sodium channels (such as KCNQ1, SCN1A, LQTS, KCNH2, and SCN5A).[17] Identification of KCNH2 and SCNC5 variants, which have been implicated in cases of long QT syndrome, is important due to the potential for ensuring appropriate medication intervention.[18]

Evaluation

Sudden unexpected death in epilepsy, by definition, is a sudden and unexpected death in a person with epilepsy for which no obvious alternative cause has been found. Evaluation for alternative causes of death is, therefore, mandatory in making a definitive SUDEP diagnosis.[19]

The diagnosis of SUDEP is based largely upon history and post-mortem examination findings such as the patient found in a prone position. The multiple potential confounding factors contributing to a patient's death make the diagnosis difficult, and accurate identification of a cause of death has medico-legal and public health implications.[20] A classification system has been proposed, taking into consideration historical points and comorbidities, which may contribute to death in an epileptic patient. Devinsky et al. (2018) suggested an 8 tiered classification system:[3]

1. *Definite SUDEP*: Sudden, unexpected death in a patient with epilepsy. The death may be witnessed or unwitnessed, and there does not need to be evidence of a recent seizure. Postmortem examination does not reveal an alternative cause of death besides epilepsy.

2. *Definite SUDEP plus comorbidity*: Same as definite SUDEP with a comorbid medical condition that could contribute to death (but not a competing cause).
3. *Probable SUDEP*: Unexpected death in a patient with epilepsy for whom an autopsy was not performed.
4. *Probable SUDEP plus comorbidity*: Same as probable SUDEP with comorbidity that could contribute to death (such as a channelopathy).
5. *Possible SUDEP*: Sudden death in a patient with epilepsy for whom there is a likely alternative cause of death (such as patient found dead in water, but no forensic confirmation of drowning made).
6. *Resuscitated SUDEP*: A patient with epilepsy who is resuscitated and survives greater than 1 hour after cardiopulmonary arrest. No alternative cause of the arrest is discovered after evaluation.
7. *Not SUDEP*: A clear alternative cause of death was identified.
8. *Unclassified*: There is insufficient information on which to make a diagnosis.

Examination findings supporting a diagnosis of SUDEP include prone position, as well as signs of recent seizures including oral trauma, ecchymoses, conjunctival hemorrhage, or lacerations to protruding areas of the head or limbs. These findings are non-specific but may support the diagnosis of a recent seizure.[21]

Treatment / Management

As sudden unexpected death in epilepsy is one of the most feared and catastrophic complications of epilepsy, options for mitigating, or modifying the risk are important. Several interventions for decreasing the risk of SUDEP have been explored, including optimizing epilepsy control, detecting seizures through observation and devices, positioning to decrease the risk of airway compromise, and pharmacologic interventions to reduce the possibility of hypoventilation.[22] Reduction of risk factors thought to contribute to SUDEP (including reducing seizure burden, increasing compliance with medication, referring appropriate patients for more advanced treatments such as surgery) are the most common interventions. In recent years, seizure-monitoring devices (alarms, heart rate monitors, bed sensors) have been a source of hope for families of epileptic patients, and evidence is increasing regarding their use.[23]

A 2012 Cochrane Review, however, has shown that while generalized tonic-clonic seizures (GTCS) may be accurately detected with such methods, there has not been enough data to suggest that they would prevent SUDEP. Similarly, the efficacy of interventions such as “safety pillows” or pharmacologic intervention with selective serotonin reuptake inhibitors to reduce central hypoventilation is not supported by data.[22]

Differential Diagnosis

The differential diagnosis for sudden unexpected death in epilepsy includes a number of alternative causes of death and typically includes entities that can be identified with an autopsy. Common alternative causes of death in patients with epilepsy include:[21]

- Drowning
- Cardiomyopathy
- Cardiac arrhythmia
- Drug overdose
- Pulmonary disease
- Trauma

- Intracranial hemorrhage

Prognosis

Although sudden unexpected death in epilepsy, by definition, is a fatal condition, the prognosis of patients with epilepsy, in general, can range from poor to excellent. The prognosis with regards to developing SUDEP in a patient with epilepsy can be assessed using a variety of tools, including the SUDEP-7 inventory. This determines a patient's risk based on multiple factors including:[24]

- 3 or more tonic-clonic seizures in the past year
- 1 or more tonic-clonic seizures in the past year
- 1 or more seizures of any type in the past year
- More than 50 seizures per month in the past year
- Duration of epilepsy greater than or equal to 30 years
- Treatment with 3 or greater anticonvulsants
- Intellectual disability with measured IQ less than 70

The presence of any of the above risk factors increases the risk score (with a maximum score of 12). However, the individual risk score has not been validated as a means to predict SUDEP. In general, the chance of dying from SUDEP is 1 in 1000, and patients with poorly controlled epilepsy have a greater risk.

Complications

SUDEP is the most catastrophic complication of epilepsy. Other complications include injuries related to seizures, aspiration pneumonitis, drowning, status epilepticus, and anti-seizure medication toxicity. Patients with epilepsy are also at risk for neuropsychiatric disorders, including learning disabilities and mood disorders, including depression. Furthermore, patients are at risk of decreased physical activity and social stigmatization.[25]

Deterrence and Patient Education

Although sudden unexpected death in epilepsy is a feared complication of epilepsy, identification, and modification of risk factors are vital for prevention. Discussion with families of patients at risk for SUDEP is likely to alter patient behavior to lower SUDEP risk. Although discussion may be a source of anxiety for patients and caregivers, studies support the notion that patients prefer information about SUDEP.[26] Professional societies such as the United Kingdom National Institute for Clinical Excellence (NICE) and the Scottish Intercollegiate Guideline Network (SIGN) recommend a discussion about SUDEP with patients after initial diagnosis.[27] Several tools exist to help stratify patients' risk of SUDEP, including The SUDEP-7 Risk Inventory.[24] The use of these tools can aid in patient discussions and help improve outcomes.

Enhancing Healthcare Team Outcomes

The diagnosis of sudden unexpected death in epilepsy is not always straightforward, and definitive determination requires collaboration between neurologists and pathologists. While the neurologist is primarily responsible for the treatment of epilepsy, the pathologist is critical in ruling out alternative diagnoses. The accurate diagnosis of SUDEP is vital for medicolegal reasons and can provide closure to families of patients.

Collaboration between physicians and patient advocacy groups is also important to ensure accurate and accessible information is available to the public. There is ample data to inform patients of their overall risk of SUDEP as well as

evidence to suggest that ongoing seizure management in patients with uncontrolled epilepsy can mitigate SUDEP risk.[Level 2] Furthermore, some evidence exists to support the use of nocturnal monitoring for seizure detection and SUDEP prevention.[Level 4] Lastly, several studies have identified modifiable risk factors in SUDEP.[Level 1] Collaborative efforts between the epilepsy treatment team (including neurologists, neurology nurses, and social workers) along with patient advocacy groups (such as the Danny Did Foundation, Epilepsy Foundation) can help to properly disseminate information amongst the public and create behavioral changes in patients to mitigate risk.[14]

Review Questions

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SUDEP

What Is SUDEP?

Sudden Unexpected Death in Epilepsy (SUDEP) is said to occur when a person with epilepsy dies unexpectedly and was previously in their usual state of health. The death is not known to be related to an accident or seizure emergency such as status epilepticus. When an autopsy is done, no other of cause of death can be found.

How Common Is SUDEP?

- Each year, more than 1 in 1,000 people with epilepsy die from SUDEP.
- People with poorly controlled epilepsy are at greatest risk of dying from SUDEP.
- SUDEP takes more lives annually in the United States than sudden infant death syndrome (SIDS).
- People with only absence or myoclonic seizures are not known to have increased risk for sudden death.

What Causes SUDEP?

No one knows what causes SUDEP, but many areas are being looked at. SUDEP occurs most often at night or during sleep when the death is not witnessed, leaving many questions unanswered. There may be evidence that a person had a seizure before dying, but this isn't always the case.

Current research into the possible causes of SUDEP focuses on problems with breathing, heart rhythm and brain function that occur with a seizure.

- *Breathing:* A seizure typically may cause a person to briefly stop breathing (apnea). If these breathing pauses last too long, they can reduce the amount of oxygen that gets to the heart and the brain. A lack of oxygen can be life threatening if not treated immediately. Also, a person's airway may sometimes get blocked during a convulsive seizure, leading to suffocation (inability to breathe).
- *Heart Rhythm*: Rarely, a seizure may cause a dangerous heart rhythm or cardiac arrest.
- *Brain Function* Seizures may suppress or interfere with the function of vital areas in the brainstem. These areas are responsible for breathing and heart rate as well as other important body functions. As a result, changes in brain function could cause dangerous breathing and heart rate changes.
- *Others:* SUDEP may result from more than one cause, or from a combination of breathing difficulty, abnormal heart rhythm and changes in brain function. Or, it may result from factors researchers have yet to discover.

Who Is at Risk For SUDEP?

The greatest risk factor for SUDEP is having tonic clonic seizures (grand mal).

People with nighttime seizures may also be at higher risk.

Missing medications or not taking seizure medicines as prescribed, because it can lead to more seizures, may also put people at higher risk for SUDEP.

Is My Child at Risk For SUDEP?

The answer depends on how severe the epilepsy is and the type of seizures she is having. While some studies found that rates of SUDEP are lower in children, others found rates similar to those seen in adults.

How Can I Reduce My Risk?

The best way to prevent SUDEP is to have as few seizures as possible.

- Get the best seizure control possible. This may involve actions such as:
    - Taking medication regularly and at the right dose.
    - Visiting your health care team regularly, especially if seizures are not controlled.
    - If medicines do not work, consider other treatment options such as epilepsy surgery, or dietary therapy.
- Take good care of yourself or your loved one. Eat well, get enough rest and regular exercise, avoid drinking too much alcohol or using recreational drugs, and minimize stress when possible.
- Be aware of and avoid any potential seizure triggers whenever possible. Keep a record of things that occurred before a seizure (such as illness, tiredness, stress, missing medications, and where and when the seizure occurred).
- Talk to your doctor about having your heart checked (cardiac evaluation) to rule out any heart problems. This is especially important if the diagnosis of epilepsy is not certain, or the seizures are not controlled.
- Be seizure safe. Make sure family and co-workers know what to do for seizure first-aid, take extra precautions around water, including swimming and bathing.

Is It SUDEP If There Was No Evidence of a Seizure?

Often there are signs that a person had a seizure before dying, but this isn't always the case. While a seizure is not a requirement for SUDEP to be diagnosed, recent studies suggest most SUDEP are likely seizure related.

Is SUDEP Genetic?

There are some studies that suggest genetic factors may play a role, but no definite information is available at this time. Several research efforts are looking into genetics and SUDEP. Read more about the

Should I Talk to My Doctor About SUDEP?

Yes! If your doctor has not spoken to you about the health risks associated with epilepsy, you should ask him or her about SUDEP.

Questions to ask may include:

- What risks do I or my family member have for SUDEP?
- What can we do to lessen the risk of SUDEP?

Can Anti-Suffocation Pillows Prevent SUDEP?

There is no data to support the use of these pillows to prevent SUDEP. They are made to help people who are at risk for suffocation. Talk to your health care provider about any possible benefits of these for you or your loved one.

Do Audio and Video Monitoring Devices or Sleeping with Someone Else In The Room Help Prevent SUDEP?

Having someone available at night who is able to provide help during and after a seizure may be one way to limit SUDEP. For example, a person could help provide first aid, keep the person on their side if they had a generalized seizure, and reposition them after the seizure so their breathing isn't blocked. However, this is often not practical or desired, and more scientific evidence is needed to prove that it is effective in preventing SUDEP.

Several devices are being developed to detect seizures and alert caregivers when a seizure occurs. However, the devices may not alert you that your loved one has stopped breathing. Whether these devices can prevent SUDEP remains unknown.

If I Have Lost a Loved One To SUDEP, Can I Participate in Research?

If you have recently lost a loved one to SUDEP, contact the *North American SUDEP Registry (NASR)* and participate in their study to help find the causes of SUDEP. The multicenter NASR provides clinical data, DNA and brain tissue for the scientific community to study. For more information call *855-432-8555* or email info@sudep-registry.org; or contact Dr. Devinsky at 646-558-0801 or email od4@nyu.edu.

The *Ion Channels in Epilepsy* study at Baylor College of Medicine is also accepting participants. This study is trying to identify genetic risk factors that may make a person with epilepsy more likely to die suddenly. For more information, contact Dr. Goldman at 713-798-0980 or email agoldman@bcm.edu.

Where Can I Get Support If I Have Lost a Loved One To SUDEP?

Contact your local Epilepsy Foundation or any of the organizations with websites listed below.

Where Can I Get More Information?

- American Epilepsy Society
- Centers for Disease Control and Prevention
- Chelsea Hutchison Foundation
- Citizens United for Research in Epilepsy
- Danny Did Foundation &
- Finding A Cure for Epilepsy and Seizures
- Making Sense of SUDEP
- North American SUDEP Registry
- Partners Against Mortality in Epilepsy
- SUDEP Action
- SUDEP Aware

https://www.epilepsy.com/learn/early-death-and-sudep/sudep/sudep-faq