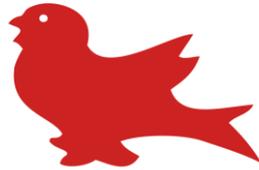


Occupational therapy

Acute care and stroke
A quick guide to assessment



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Types of strokes

Hemorrhagic

3 membranes (meninges) surrounding the brain: dura mater, arachnoid and pia mater

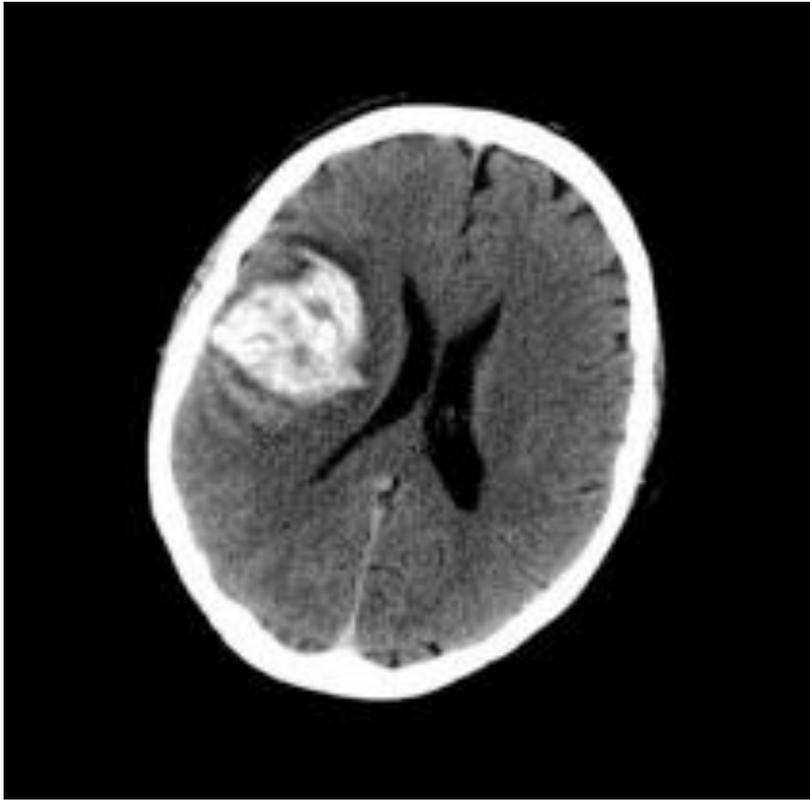
Dura mater - bleeding between dura mater and arachnoid is called subdural hemorrhage

Arachnoid - bleeding between arachnoid and pia mater is called subarachnoid hemorrhage

- ❑ Usually from trauma, head injury

Pia mater - bleeding below pia mater is called intracerebral hemorrhage

- ❑ Different causes: Hypertension, abnormal bleeding functions, vascular malformations, bleeding from brain tumors and infarcts, head trauma



Intracerebral hemorrhage



Intracerebral hemorrhage
secondary to brain tumor (glioblastoma multiforme)

Treatment of hemorrhagic strokes

Attenuation of hematoma expansion – blood pressure control

Another potential strategy to limit hematoma expansion is to lower BP during the first several hours after intracerebral hemorrhage. Acute hypertension is common and is associated with hematoma expansion and worse outcomes

- ❑ Short acting intravenous agents

Surgery

Hematoma evacuation with or without suboccipital decompressive craniectomy is indicated for patients with cerebellar intracerebral hemorrhage and clinical or radiographic evidence of impending brainstem compression.

Types of strokes

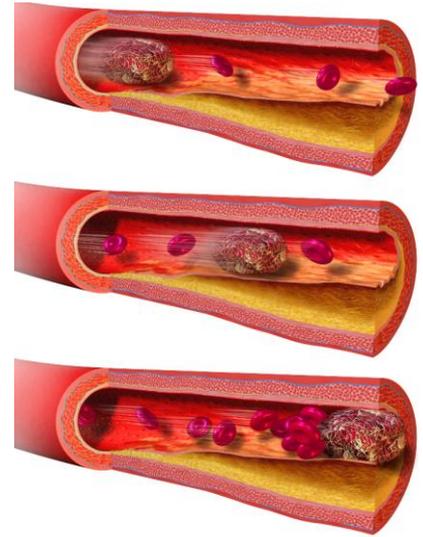
Ischemic

Decrease of blood supply to the brain

If long enough - leads to cell/tissue death and is called ***infarction***

- ❑ Thrombosis - formation of a thrombus inside the blood vessel
 - ❑ Atherosclerosis - stenosis or buildup of plaque within an artery

- ❑ Embolism - particles (clot) breaking loose and blocking a distant artery
 - ❑ Source can be from three locations: heart, aorta, or one of the major arteries in the neck



Treatment of ischemic strokes

Recombinant tissue plasminogen activator (rt-PA)

When administered intravenously or intra arterially rt-PA can lyse emboli or thrombi that obstruct intracranial arteries and are responsible for inciting an ischemic stroke.

Window of time for administration is from 3 hours to 4.5 hours after symptoms onset. Patient's admissibility to rt-PA depends on multiple inclusion/exclusion criteria.

The primary safety concern associated with rt-PA for treatment of acute ischemic stroke is symptomatic hemorrhagic transformation of cerebral infarction (SICH). Following rt-PA infusion patients should be monitored in a critical care or stroke unit setting for at least 24 hours.

Occlusion of left middle cerebral artery before and after r-tPA



Treatment of ischemic strokes

Thrombectomy - patients are sent to the Montreal Neurological Institute from the JGH.

Surgical - The removal of a thrombus from a blood vessel, performed as emergency surgery to restore circulation to the affected part. Anticoagulant therapy may begin before surgery. During surgery a longitudinal incision is made into the blood vessel, and the clot is removed. After surgery the blood pressure is maintained close to its preoperative level because a decrease would predispose to further clotting.

Mechanical - There are now a number of devices on the market that aim to remove thrombus by mechanical rather than pharmacological means, such as thrombus fragmentation devices, to avoid the associated side effects.

Percutaneous thrombectomy - aspiration of thrombus via catheter

Transient ischemic attacks (TIAs)

Transient ischemic attacks

- ❑ Temporary inadequacy of blood flow from narrowing of an artery
- ❑ Temporary blockage of an artery by an embolus that passes

Transient ischemic attacks (TIA) occur in approximately 15% of patients before an eventual stroke, with the highest risk in the first days to weeks following an event.

The diagnosis of a TIA requires absolute resolution of symptoms, whereas a persistent deficit should continue to raise concern for a treatable stroke. If a patient returns completely to their neurological baseline (100%), then the clock starts over and any recurrent deficits may be considered a new event (i.e. reopening the treatment window).

What to expect

Motor weakness/Paralysis

Apraxia

Ataxia

Numbness, hypoesthesia, paresthesia

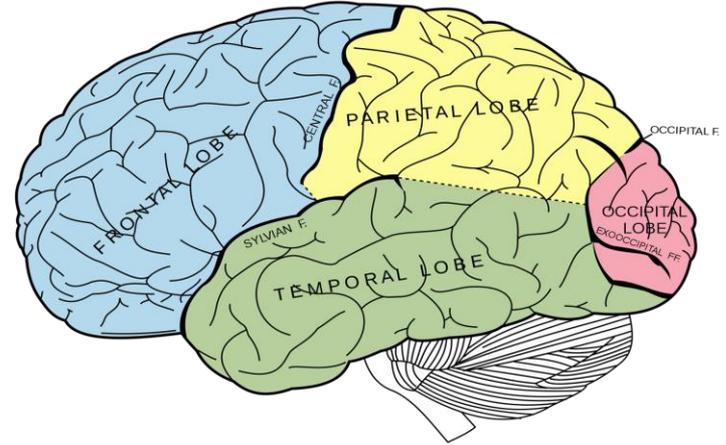
Loss of vision

Dizziness, vertigo, loss of balance and coordination

Altered speech and language, dysarthria

Dysphagia

Abnormalities of memory, thinking and behavior



What to expect

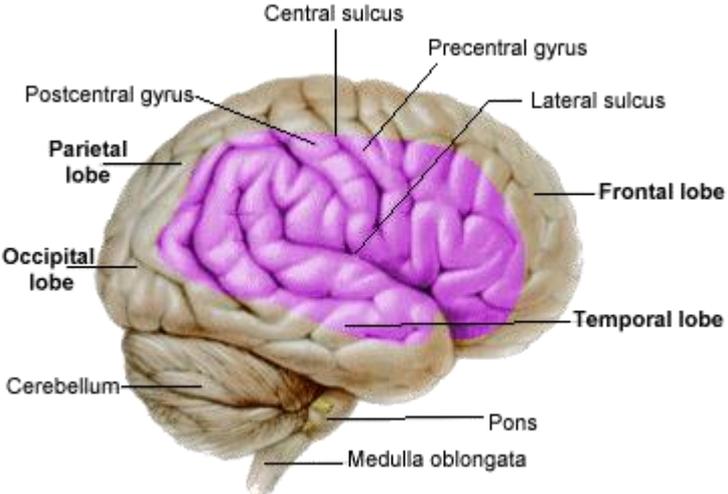
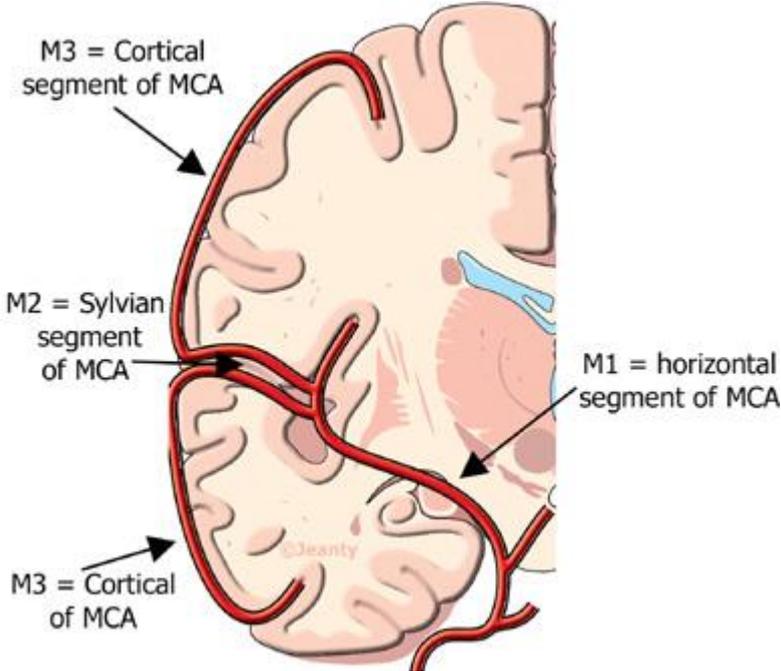
Being pro-active

Research about the anatomical localization of the stroke will give you an extensive amount of information about the anticipated or possible deficits.

PROACTIVE



Middle cerebral artery (MCA) complete occlusion

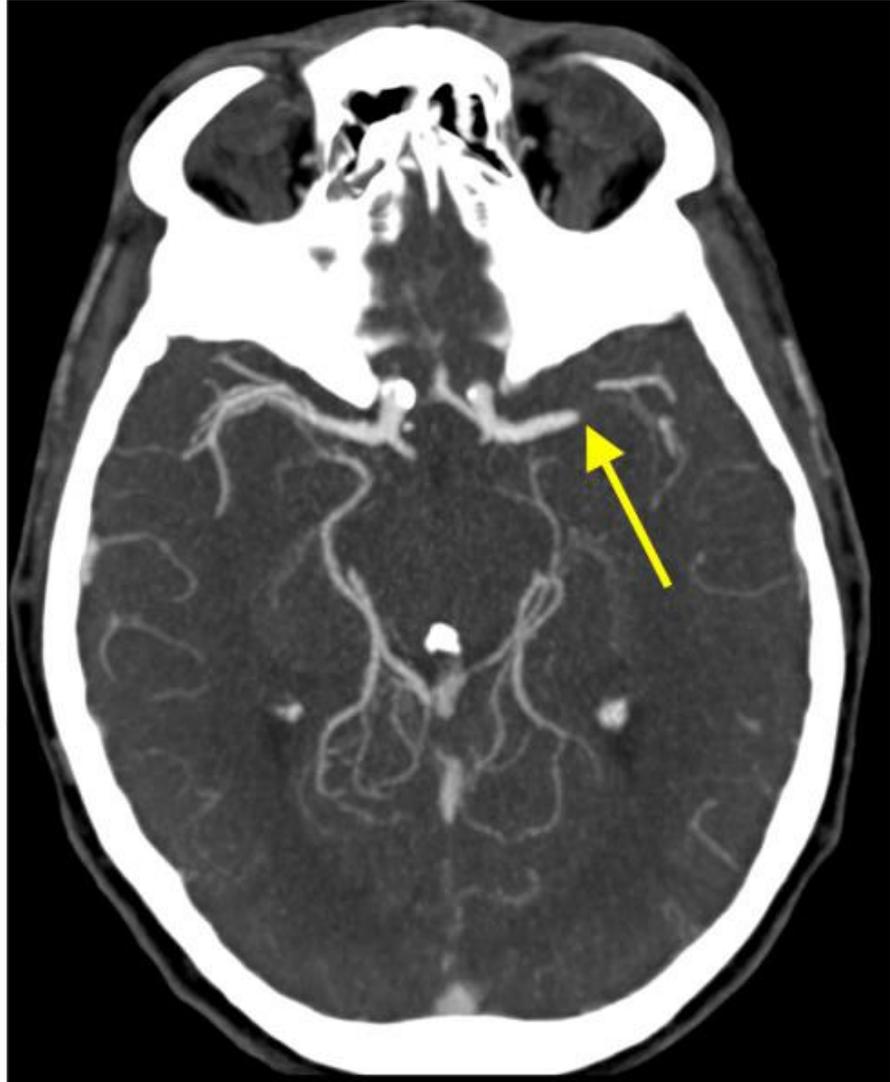
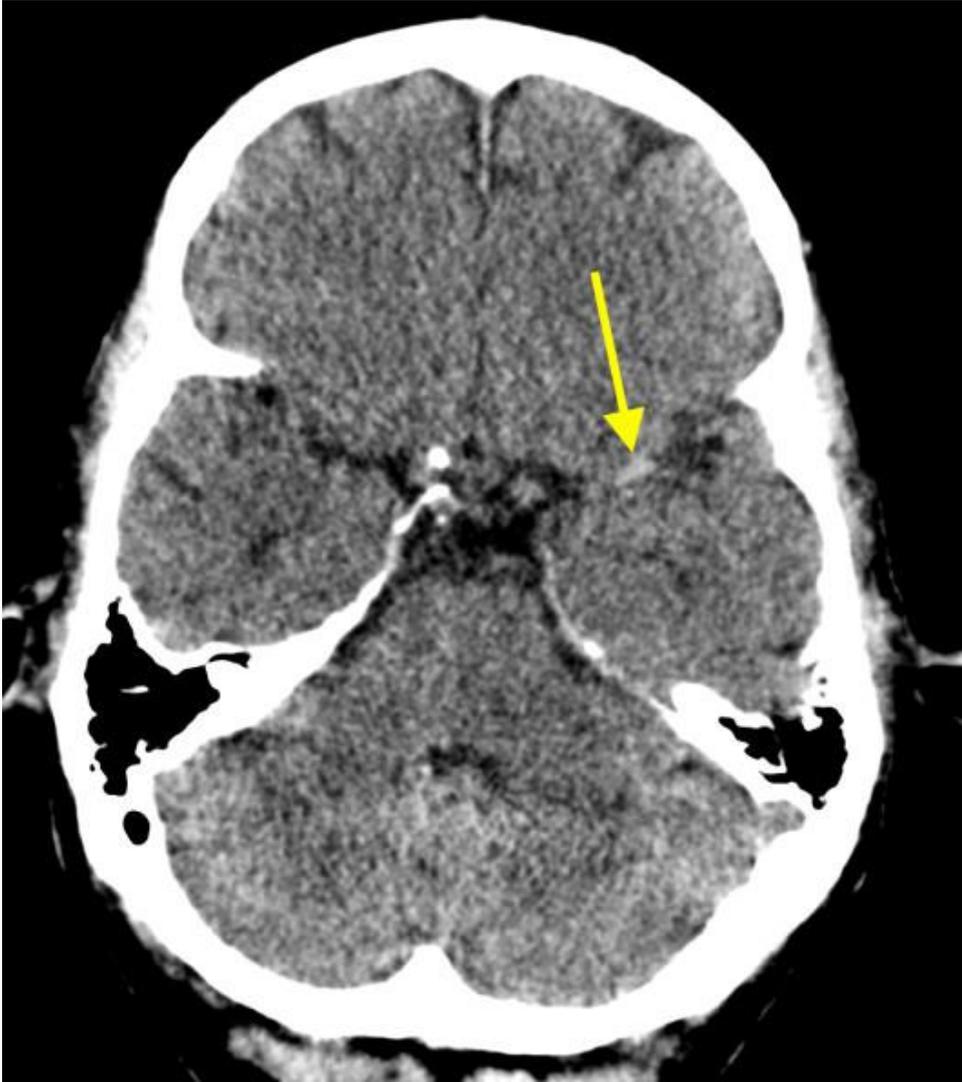


MCA complete occlusion

For example, for a complete (in other words, at its origin) occlusion of the middle cerebral artery, the lateral frontoparietal and superior temporal regions of the brain are affected.

Contralateral hemianesthesia, hemiparesis and hemianopia with gaze preference are to be expected. If the stroke is located in the patient's dominant hemisphere aphasia and apraxia are expected as well.

If located in the non-dominant hemisphere, the patient might demonstrate symptoms of hemineglect and aprosodia (inability of a person to properly convey or interpret emotional [prosody](#)).



Gathering information

Chart review

Type of stroke

Anatomic localization of the stroke

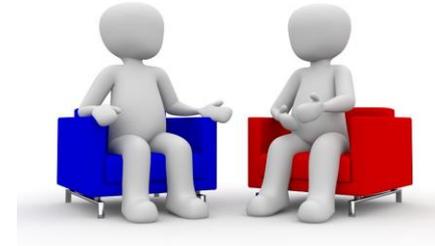
Bullet rounds

Multidisciplinary input

Interview with patient and family



Interview



Activities of daily living

Routines

Instrumental activities of daily living

Social environment; what type of support do they have? At what frequency?

Physical environment

How are they doing on a psychosocial level?

Cognition - Are they oriented, can they recall what happened to them?

→ ***Going beyond the OT evaluation template. We are often the only professionals who are interested in patient's life and how they are coping with their condition. Be an active listener.***

Interview

Patient's and their families **are your best resource for information.**

- Did they notice any memory difficulties?
- Is their speech different from baseline?
- Can they qualify their pain/numbness?
- Have symptoms improved or worsened since their admission to the hospital?



The aphasias

	<i>Fluency</i>	<i>Comprehension</i>	<i>Repetition</i>
Motor/expressive (Broca)	Impaired	Normal	Impaired
Sensory/receptive (Wernicke)	Normal	Impaired	Impaired
Conduction	Normal	Normal	Impaired
Transcortical motor	Impaired	Normal	Normal
Transcortical sensory	Normal	Impaired	Normal
Mixed	Variable	Variable	Variable
Global	Impaired	Impaired	Impaired

Assessments

- ❑ Vision and visual perception
- ❑ Memory and cognition
- ❑ Sensory and motor function
- ❑ Task simulations
- ❑ Tailoring your assessment to the patient's lifestyle, work and difficulties

→ **Participation**

Vision and visual perception

Blink to threat

In comatose or uncooperative patients, visual fields can be tested roughly using *blink-to-threat*, in which the examiner's fingers are moved rapidly towards the patient's eyes from each quadrant to see if a blink occurs.

Testing visual fields

Confrontation - With the examiner seated directly across from the patient, the patient should direct their gaze to the corresponding eye of the examiner. A moving target should start outside the usual 180 ° visual field, then move slowly to a more central position until the patient confirms visualization of the target.

Pupillary response VS Blink-to-Threat Reflex

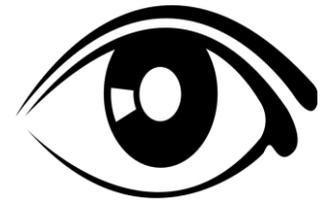
What is different from the usual testing done with a light?

Pupillary response to light requires an intact optic nerve and connection to the pretectum (midbrain) but not to the cortex. It is present in infants at 25 weeks of gestation. It is used in emergency to assess brainstem function.

Whereas blink-to-threat is a learned response. It does not appear before 2-4 months of age and therefore requires higher order cortical processing of the stimulus.

→ ***No blink to threat is a sign of cortical blindness.***

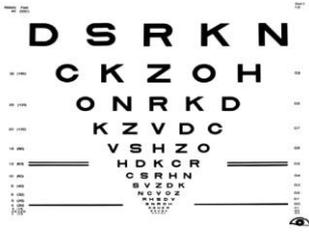
Vision and visual perception



Visual pursuit - Smooth pursuit or “following” eye movements is an ability that is modulated at different levels in the brain. It is assessed by asking the patients to follow your fingers clockwise/counter-clockwise.

Why is it important to assess? Disruption of slow eye movements (known as ‘smooth pursuit’) results in an inability to follow slow moving objects accurately. Compensatory fast jerky movements may replace slow eye movements. This may make it difficult to keep visual attention on one object of interest.

Disturbance of rapid eye movements (known as ‘saccades’), which normally allows a person to look from one object of interest to another may lead to difficulties with activities such as reading, sewing, playing cards etc.



Vision and visual perception

Visual acuity - clarity of vision. Usually assessed by asking the person if they can see appropriately. Also tested by asking the patient to read something on the wall, to tell the color of your eyes or to count the number of fingers at a distance.

Visual perception - gathering visual information from the environment to integrate it into other senses. From example, simply having the patient grab an object in space gives information about their visuo-spatial skills.

Cognition and memory



What to expect?

It is difficult to capture all possible cognitive deficits as there are so many.

Executive function difficulties: information generation, divided attention and task coordination, working memory, sustained attention, planning and problem solving, etc.

Behavioral changes: abulia, apathy, aspontaneity, distractibility, impulsivity, disinhibition, perseveration, disturbances of emotion and social behavior, etc.

Cognition and memory

*Patients with stroke are often elderly and the effects of stroke need not be the same across the lifespan, nor can the effects of aging or undiagnosed degenerative diseases of aging always be eliminated. Whatever their age, patients with a clinical stroke often have accumulated years of subclinical vascular injury that can never be entirely discounted by imaging. **This is why it is important to establish a complete baseline of the patient.***

What to assess?

Clinical observations will give you a good base of information to begin with.

Are they oriented to time, place and person? Can they remember why they are in the hospital? Are they aware of their difficulties? Do they have insight into how these difficulties are affecting their performance in ADLs/IADLs? Can they follow a conversation? Do they remember meeting you before?

Unilateral spatial neglect (USN)

Inability to orient to stimuli on the contralateral side of the brain lesion. Usually present following a lesion in the right hemisphere.

3 types:

- Personal* - neglect of the affected hemibody
- Near extrapersonal* - neglect of environment within reaching distance on contralateral side of brain lesion
- Far extrapersonal* - neglect of the environment beyond reaching distance on contralateral side of brain lesion

Can be assessed through clinical observations. Handing a comb or cloth to the patient to see if they will attend to the affected hemibody. Asking them to reach for their affected arm and observing if they can cross the midline of their body.

Unilateral spatial neglect (USN)

Neglect is also possible following a lesion in the left hemisphere of the brain, however it is rare and less severe.

Why is neglect important to assess? Neglect was found to be the best single predictor of poor functional outcome at 3, 6 and 12 month follow-up amongst other deficits (hemiparesis, hemianopia, age, visuoconstructional ability and memory). However, as neglect is usually compensated for very early after stroke, the authors found that anosognosia, would in fact be the best single predictor as it is almost always present in combination with visual neglect.

Anosognosia - A deficit in which the patient is unaware of his disability. Severe form of neglect to the extent that the patient fails to recognize the presence or severity of his or her paralysis.

Apraxia

Apraxia is usually found in stroke patients with a left-hemispheric lesion.

Apraxia and aphasia frequently coexist and the association between the two deficits is likely due to the involvement of contiguous structures. Many different forms and classifications of apraxia are described in the literature, but there is not one accepted taxonomy.

Ideational and ideomotor apraxia have been the object of most studies in recent years and are sometimes labelled as the two classic forms of apraxia.

A patient with ideational apraxia does not know what to do because the idea or concept of the motor act is lacking, while a patient with ideomotor apraxia does know what to do, but not how to do it.

Apraxia

Ideational apraxia: The types of error one can observe in ideational apraxia are omissions, mislocation or misuse of objects and sequence errors.

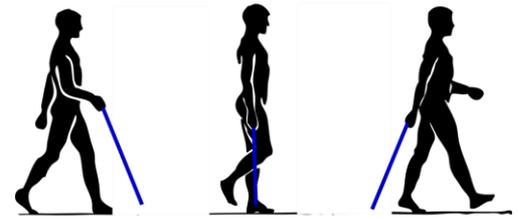
Ideomotor apraxia: The most frequent errors in ideomotor apraxia are the use of body-parts as objects, spatial orientation problems, inappropriate hand postures, perseverations and content errors. Patients with ideomotor apraxia may not be able to perform on command, while exactly the same activity may be executed perfectly in a natural setting.

There are no standardized methods to assess apraxia.

So how do we assess?

Clinical observations and activities of daily living. - imitation of gestures, pantomime and the use of objects.

Ataxia



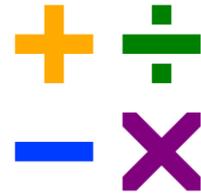
Ataxia means without coordination. It is most common after a cerebellar stroke.

Neurological examination shows dysmetria of limbs (inaccuracy of targeted movements), as shown by abnormal finger-chasing using patient fingers or toes. There is kinetic tremor of limbs and an uncontrolled oscillation of limbs during relatively slow but targeted movements. Early stance and gait problems include the inability to do a tandem stance or stand with feet together; stance becomes broad-based and displays increased sway of the body.

Eye movements show gaze-evoked or other types of nystagmus, abnormal pursuit of visually presented objects (jerky appearance due to intrusion of saccades into pursuit), and inaccurate saccades when the person is asked to move the eyes quickly towards a target (hypometric or hypermetric saccades).

Sensory ataxia is characterised by lower-limb and upper-limb incoordination associated with lack of proprioception. Clinical signs include impaired vibration sense, as well as impaired position and kinaesthetic sense.

Cognition and memory



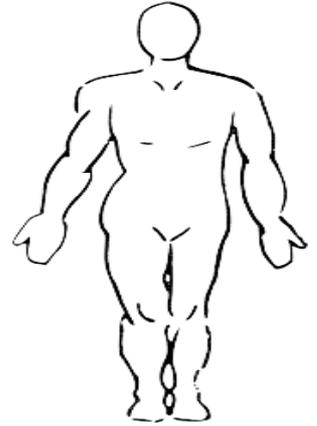
If the expected discharge plan for the patient is **anywhere but rehabilitation** the MoCA should be completed at all times to establish a baseline in the case that the patient is readmitted to the hospital in the future.

If the expected discharge plan for the patient is **inpatient rehabilitation**, you can record observations but cognitive difficulties will be assessed more in depth over there.

If the expected discharge plan for the patient is **home with outpatient cognitive rehabilitation**, you should do every assessment pertinent to the patient's difficulties to establish a strong report. You can use the MoCA and the Execute Function Performance Test (EFPT) if it applies. Otherwise, unstandardized task simulations can give you plenty of information about the patient's cognitive functioning difficulties.

Sensory and motor function

- Range of motion
- Manual muscle testing
- Spasticity - Modified ashworth scale
- Coordination
- Tone
- Sensation to light touch



Reflection



Putting it all together

- Identifying the person's needs
- Are they safe to return home? If not, what limits their safety?
- What could they benefit from or improve before being discharged?
- Input from other members of the multidisciplinary teams
- Is something missing?

Advocacy

One of our major roles as Occupational Therapist is **being an advocate** for our patients.

We are one of the only rehabilitation therapists who look at the patient holistically and functionally. This is why it is important to share our professional opinion during interdisciplinary meetings.

Other professionals might not agree and push for a faster discharge. However, our role is to make sure the patient is safe, that all of their needs are or will be met, and that proper follow-up is planned before they leave the hospital.

ADVOCACY



What will you be for this patient in hospital?

Will you only consult?

- Recommendations for discharge

Or treat?

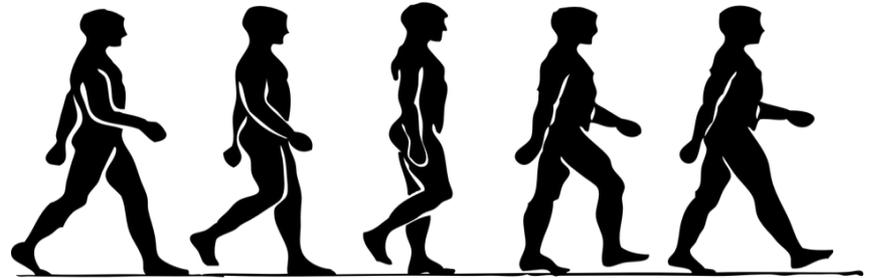
- Establishing a treatment plan before discharge
- Recommendations for discharge

You can facilitate this decision by thinking about the needs of the patient, and if those need can be met in hospital. Do we have the resources to meet this need?

Treatments

Soft tissue

- Preventing musculoskeletal injuries from immobility and bedrest
- Reduce risks of shoulder subluxation
- Passive or active mobilization of affected limbs
- Reducing spasticity
- Stretching
- Static or dynamic splinting
- Edema
- Positioning**

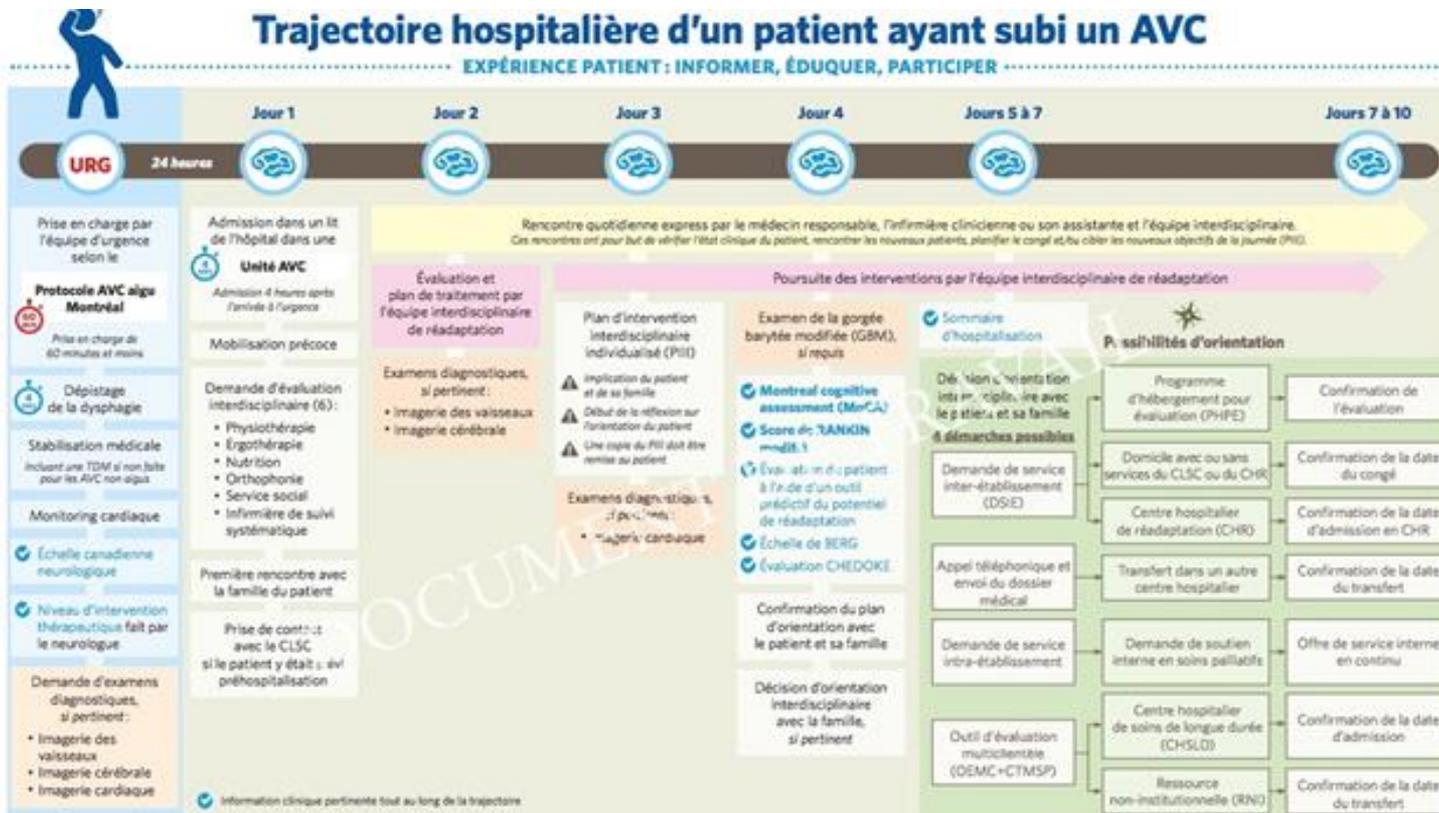


Treatments

Improving mobility, endurance and strengthening

- Trunk control exercises at edge of the bed
- Core strengthening from sitting in Broda chair or regular chair for a minimum amount of time per day
- Integrating the use of the affected arm in activities
- Encouraging patient to participate as much as possible to routine in hospital
- Repetition of active movements
- Practicing transfers
- Getting out of bed!!!!

Trajectory of stroke patients in hospital



Discharge planning

Home

- with CLSC? Transport adapte?
- With out patient-rehabilitation (neuro SARCA, tertiary rehab, visual rehab)
- Groups (heart and stroke foundation)

Rehabilitation

- IRGLM, IUGM, Villa Medica, Julius Richardson, Jewish Rehab, CMR

Relocation

- CHSLD
- Long-term care
- Family, ressources intermédiaire, foster home



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