Chapter 1 : The 3-Minute Neurological Examination - The MMR

The cranial nerve exam is a type of neurological calendrierdelascience.com is used to identify problems with the cranial nerves by physical calendrierdelascience.com has nine components. Each test is designed to assess the status of one or more of the twelve cranial nerves (I-XII).

The patient is a year-old right-handed woman with a history of chronic headaches who complains of acute onset of double vision and right eyelid droopiness three days ago. History of present illness: When she looked up at the clock on the wall, she had a hard time making out the numbers. At the same time, she also noted a strange sensation in her right eyelid. She went to bed and upon awakening the following morning, she was unable to open her right eye. When she lifted the right eyelid with her fingers, she had double vision with the objects appearing side by side. The double vision was most prominent when she looked to the left, but was also present when she looked straight ahead, up, down, and to the right, and went away when she closed either of her eyes. She also noted that she had pain in both of her eyes that increased if she moved her eyes around, especially on looking to the left. Smith also notes that for the past two to three weeks, she has been having intermittent pounding bifrontal headaches that worsen with straining, such as when coughing or having a bowel movement. The headaches are not positional and are not worse at any particular time of day. She rates the pain as 7 or 8 on a scale of 1 to 10, with 10 being the worst possible headache. The pain lessened somewhat when she took Vicodin that she had lying around. She denies associated nausea, vomiting, photophobia, loss of vision, seeing flashing lights or zigzag lines, numbness, weakness, language difficulties, and gait abnormalities. She has never taken anything for these headaches other than ibuprofen or Vicodin, both of which are partially effective. The last headache of that type was two months ago. Her visual symptoms have not changed since the initial presentation. She denies previous episodes of transient or permanent visual or neurologic changes. She denies head trauma, recent illness, fever, tinnitus or other neurologic symptoms. She is not aware of a change in her appearance, but her husband notes that her right eye seems to protrude; he thinks that this is a change in the last few days. Migraine headaches, as described in HPI. There is no history of diabetes or hypertension. Zoloft 50 mg daily, ibuprofen mg a few times per week, and Vicodin a few times per week. The patient lives with her husband and year-old daughter in a 2-story single-family house and has worked as a medical receptionist for 25 years. She denies tobacco or illicit drug use and rarely drinks a glass of wine. Her mother had migraines and died at the age of 70 after a heart attack. Her maternal grandfather had a stroke at age She states that she had an upper respiratory infection with rhinorrhea, congestion, sore throat, and cough about 6 weeks ago. She denies fever, chills, malaise, weight loss, neck stiffness, chest pain, dyspnea, abdominal pain, diarrhea, constipation, urinary symptoms, joint pain, or back pain. Neurologic complaints as per HPI. The patient is obese but well-appearing. There is no tenderness over the scalp or neck and no bruits over the eyes or at the neck. There is no proptosis, lid swelling, conjunctival injection, or chemosis. Cardiac exam shows a regular rate and no murmur. The patient is alert, attentive, and oriented. Speech is clear and fluent with good repetition, comprehension, and naming. Visual fields are full to confrontation. Fundoscopic exam is normal with sharp discs and no vascular changes. Venous pulsations are present bilaterally. Pupils are 4 mm and briskly reactive to light. At primary gaze, there is no eye deviation. When the patient is looking to the left, the right eye does not adduct. When the patient is looking up, the right eye does not move up as well as the left. She develops horizontal diplopia in all directions of gaze especially when looking to the left. There is ptosis of the right eye. Facial sensation is intact to pinprick in all 3 divisions bilaterally. Corneal responses are intact. Face is symmetric with normal eye closure and smile. Tongue is midline with normal movements and no atrophy. There is no pronator drift of out-stretched arms. Muscle bulk and tone are normal. Strength is full bilaterally.

Chapter 2 : The Precise Neurological Exam

The cranial nerve examination involves a number of steps as you are testing all 12 of the nerves in one station. Be certain to know which nerve is being tested next and what tests you must perform for each specific nerve.

Oculomotor nerve , Trochlear nerve , Abducens nerve Ocular and extraocular movements, pupillary response Ocular movements are tested by standing one meter in front of the patient and asking the patient to follow a target with eyes only, and not the head. The target is moved in an "H" shape and the patient is asked to report any diplopia. Nystagmus is tested for. One or two beats is a normal finding. As the eyes converge, the pupils should constrict. The optokinetic nystagmus test is optional and involves asking the patient to look at a moving strip of horizontal lines. Nystagmus is normally observed. Extraocular movements is tested by inspecting for ptosis , eye position and nystagmus. The pupil size is measured, its shape and any asymmetry is tested. A commonly used abbreviation to describe normal pupils is PERRLA pupils equal, round and reactive to light and accommodation. Pupillary light reflex is tested by having the patient stare into the distance as the examiner shines the penlight obliquely into each pupil. Pupillary constriction is tested for on the eye examined direct response and on the opposite eye consensual response. The swinging flashlight test involves moving the light between the two pupils. Normally both direct and consensual responses are ellicited when the light shines on an eye, and some dilation will occur during the swing between. Trigeminal nerve Facial sensation Light touch is tested in each of the three divisions of the trigeminal nerve and on each side of the face using a cotton wisp or tissue paper. The ophthalmic division is tested by touching the forehead, the maxillary division is tested by touching the cheeks, and the mandibular division is tested by touching the chin. Be careful not to test the mandibular division too laterally, as the mandible is innervated by the great auricular nerve C2 and C3. A common mistake is to use a stroking motion, which will trigger pain and temperature nerves. Instead, a point stimulus should be applied. For pain and temperature repeat the same steps as light touch but use a sharp object and a cold tuning fork respectively. Corneal reflex is conducted along with the facial nerve section of the test. Note the sensory innervation of the cornea is provided by the trigeminal nerve while the motor innervation for blinking the eye is provided by the facial nerve. Palpate the temporalis and masseter as the patient clenches the jaw. The pterygoids can be tested by asking the patient to keep the mouth open against resistance, and move from side to side against resistance. Normally the jaw moves minimally.

Chapter 3 : Examination of Facial Nerve (7th Cranial Nerve) | Epomedicine

The next part of the extraoral examination includes a cranial nerve examination. We perform this examination if there is a patient that has a significant facial pain component.

You can expand or shrink each area as you need to include relevant data for your client. Patient states she has a history of dry macular degeneration and it is being well controlled. Patient uses special eye drops for her eye disease. Admits to blurred vision occasionally. Patient denies any ear problems Last eye infection was over 10 years ago. No discharge noted from ears Nose, Mouth, and Throat discharge, sores or lesions, pain, nosebleeds, bleeding gums, sore throat, allergies, surgeries, usual dental care, medications: Lips pink, mucous membranes pink and moist No discharge or foul odor noted No history of nosebleeds, bleeding gums, allergies or surgeries on nose, mouth or throat. Lymph nodes barely palpable Patient had 4 wisdom teeth removed at age 20 Patient states she flosses daily, and brushes her teeth twice a day with sensodine. No dentures noted No missing teeth. Patient denies any skin disease, any changes in skin, excessive dryness, moisture, itching, any bruising, rashes Patient has thinning hair. Patient uses protective clothing and hats in the sun Patient has an ingrown nail on her left big toe that causes discomfort and goes to a podiatrist to get it looked. Patient does her pedicure every two weeks to have the nail trimmed by professionals. Breasts and Axilla pain or tenderness, lumps, nipple discharge, rash, swelling, trauma or injury to breast, mammography, breast self-exam, medications: Cardiovascular System chest pain or tightness, SOB, cough, swelling of feet or hands, family history of cardiac disease, tire easily, self-history of heart disease, medications: No cardiovascular problems noted Patient states she takes a baby aspirin daily as prescribed by her doctor Patient walks daily for a minimum of 30 minutes. No cough, SOB or pain while breathing No history of lung disease Past smoker of 10 years, Quit when she was 40 years old. Joint and back pain noted after long days working at the Church and in the garden, particularly when in one position for long periods. Limited range of motion in rotator cuff , performs exercises as instructed by physical therapist No accident or trauma to bones Performs activities of daily living independently, just slower. Gastrointestinal System change in appetite â€" increase or loss; difficulty swallowing; foods not tolerated; abdominal pain; nausea or vomiting; frequency of BM; history of GI disease, ulcers, medications: Patient has regular daily bowel movements, takes fiber to help No change in appetite No difficulty swallowing foods History of diverticulosis, avoids nuts and seeds Occasional antacid after too much Italian food Genitourinary System recent change, frequency, urgency, nocturia, dysuria, polyuria, oliguria, hesitancy or straining, urine color, narrowed stream, incontinence, history of urinary disease, pain in flank, groin, suprapubic region or low back: Neurological System exam of all 12 cranial nerves, motor and sensory assessments: Limited range of motion in right arm Detects cold and warm touches as well as sensations Able to distinguish smells EOM intact, pupils reactive to light 6 cardinal fields of vision Lifts eye brows, demonstrated facial movements Able to distinguish taste Able to hear sounds Able to swallow, gag reflex in place, moves tongue without difficulty Able to shrug shoulders and ROM of neck Head and Neck palpate the skull, inspect the neck, inspect the face, palpate the lymph nodes, palpate the trachea, palpate and auscultate the thyroid gland: Lymph node palpable, non tender or swollen Trachea midline Eyes test visual acuity, visual fields, extraocular muscle function, inspect external eye structures, inspect anterior eyeball structures, inspect ocular fundus: EOM intact Sclera clear 6 cardinal fields of gaze Known history of dry macular degeneration, unable to detect abnormality of visual assessment. Ears inspect external structure, otoscopic examination, inspect tympanic membrane, test hearing acuity: No drainage noted in ears, minimal wax seen in ears Tympanic membrane seen, pink non ruptured Sounds able to be detected Nose, Mouth, and Throat Inspect and palpate the nose, palpate the sinus area, inspect the mouth, inspect the throat: Skin smooth, non elastic Cool to touch Mucus membranes moist No lesions on scalp Thinning hair Nails short and clean, in growing toenail left toe Breasts and Axilla deferred for purpose of class assignment Peripheral Vascular and Lymphatic System inspect arms, symmetry, pulses; inspect legs, venous pattern, varicosities, pulses, color, swelling, lumps: Carotid arteries palpable JVD â€" Apical pulses heard, S1 S2 noted HR 80, NSR Thorax and Lungs inspect thoracic cage, symmetry, tactile fremitus, trachea; palpate symmetrical expansion;, percussion

of anterior, lateral and posterior, abnormal breathing sounds: No swelling, mass or deformity noted on spine. Rotator cuff pain when rotated Limited range of motion in right hand related to that Shoulders even, squared No deformity noted in hands, feet, hips Active ROM in legs. Gastrointestinal System contour of abdomen, general symmetry, skin color and condition, pulsation and movement, umbilicus, hair distribution; auscultate bowel sound;, percuss all four quadrants; percuss border of liver; light palpation in all four quadrantsâ€" muscle wall, tenderness, enlarged organs, masses, rebound tenderness, CVA tenderness: Abdomen soft, flat, symmetric Umbilicus shallow and clean Bowel sounds active x4 No tenderness No enlarged organs palpated Negative rebound tenderness Genitourinary System deferred for purpose of this class FHP Assessment Cognitive-Perceptual Pattern: Patient needs corrective lenses Has dry macular degeneration Occasionally back, neck and joint pain from prolonged standing Joint pain related to rotor cuff injury, patient was instructed on exercises to help by physical therapist. Patient east small meals a day, cuts out heavy meals at night One glass of wine at dinner, nights a week Fiber is increased No seeds and nuts related to diverticulosis Sexuality-Reproductive Pattern: Daily bowel movements Has stress incontinence when coughing or laughing Urinary frequency No diseases Pattern of Activity and Exercise: Participates in church activities 6 days a week Drives but has limited vision at night Pattern of Sleep and Rest: Sleeps hours a night Takes 1 hour nap in afternoon Feels rested when she wakes up in the morning. Pattern of Self-Perception and Self-Concept: Loves herself as she is, uses minimal make up. Has beautiful skin Is very religious and views herself as living a humble life She is grateful for life and all she has been blessed with Summarize Your Findings Use format that provides logical progression of assessment. Situation reason for seeking care, patient statements: August 27th, Situation: Health History and Screening Interview Background health and family history, recent observations: The patient is an 80 year old female who has a history of diverticulosis, dry macular degeneration, injured rotator cuff and frequent stress incontinence. The patient has had yearly physical checkups. Assessment assessment of health state or problems, nursing diagnosis: Patient lives an active lifestyle. She controls her macular degeneration with medication, frequent checkups and surgery. Patient has stress incontinence and seeks further education and instruction. Pain in joints restricts patient from church activities sometimes. She sees a physical therapist. When asked to demonstrate exercise techniques given to do daily by physical therapist she is unable to demonstrate all. Effective Therapeutic Regimen Management as evidenced by thorough management of macular degeneration. Altered Urinary Elimination; Stress Incontinence as evidenced by frequent involuntary urinary elimination when coughing and laughing. Recommendation diagnostic evaluation, follow-up care, patient education teaching including health promotion education: Hygienic recommendations were also given in regards to keeping dry and avoiding infections. Patient demonstrated complete understanding of eye care. Next eye appointment is in 2 months. Physician phone number easily accessible in case of emergency.

Chapter 4 : A Practical Guide to Clinical Medicine

Examination of Facial Nerve (7th Cranial Nerve) The anatomy of facial nerve has already been discussed in detail earlier. It is essential to have proper knowledge of anatomy to understand this section of clinical examination of facial nerve.

List of foramina of the human body After emerging from the brain, the cranial nerves travel within the skull , and some must leave this bony compartment in order to reach their destinations. Often the nerves pass through holes in the skull, called foramina , as they travel to their destinations. Other nerves pass through bony canals, longer pathways enclosed by bone. These foramina and canals may contain more than one cranial nerve and may also contain blood vessels. The olfactory nerve I , actually composed of many small separate nerve fibers, passes through perforations in the cribiform plate part of the ethmoid bone. These fibers terminate in the upper part of the nasal cavity and function to convey impulses containing information about odors to the brain. The optic nerve II passes through the optic foramen in the sphenoid bone as it travels to the eye. It conveys visual information to the brain. The oculomotor nerve III , trochlear nerve IV , abducens nerve VI and the ophthalmic branch of the trigeminal nerve V1 travel through the cavernous sinus into the superior orbital fissure , passing out of the skull into the orbit. These nerves control the small muscles that move the eye and also provide sensory innervation to the eye and orbit. The maxillary division of the trigeminal nerve V2 passes through foramen rotundum in the sphenoid bone to supply the skin of the middle of the face. The mandibular division of the trigeminal nerve V3 passes through foramen ovale of the sphenoid bone to supply the lower face with sensory innervation. This nerve also sends branches to almost all of the muscles that control chewing. The facial nerve then reaches the side of the face by using the stylomastoid foramen, also in the temporal bone. Its fibers then spread out to reach and control all of the muscles of facial expression. The vestibulocochlear nerve reaches the organs that control balance and hearing in the temporal bone, and therefore does not reach the external surface of the skull. The glossopharyngeal IX , vagus X and accessory nerve XI all leave the skull via the jugular foramen to enter the neck. The glossopharyngeal nerve provides innervation to the upper throat and the back of the tongue, the vagus provides innervation to the muscles in the voicebox, and continues downward to supply parasympathetic innervation to the chest and abdomen. The accessory nerve controls the trapezius and sternocleidomastoid muscles in the neck and shoulder. The hypoglossal nerve XII exits the skull using the hypoglossal canal in the occipital bone and reaches the tongue to control almost all of the muscles involved in movements of this organ. The sensory innervation includes both "general" sensation such as temperature and touch, and "special" innervation such as taste , vision , smell , balance and hearing [1] [11] The vagus nerve X provides sensory and autonomic parasympathetic motor innervation to structures in the neck and also to most of the organs in the chest and abdomen. Damage to the olfactory nerve I can cause an inability to smell anosmia , a distortion in the sense of smell parosmia , or a distortion or lack of taste. If there is suspicion of a change in the sense of smell, each nostril is tested with substances of known odors such as coffee or soap. Intensely smelling substances, for example ammonia, may lead to the activation of pain receptors nociceptors of the trigeminal nerve that are located in the nasal cavity and this can confound olfactory testing. A person may not be able to see objects on their left or right sides homonymous hemianopsia , or may have difficulty seeing objects on their outer visual fields bitemporal hemianopsia if the optic chiasm is involved. Visual field testing may be used to pin-point structural lesions in the optic nerve, or further along the visual pathways. Both or one eye may be affected; in either case double vision diplopia will likely occur because the movements of the eyes are no longer synchronized. This object may be a finger or a pin, and may be moved at different directions to test for pursuit velocity. Individuals suffering from a lesion to the oculomotor nerve may compensate by tilting their heads to alleviate symptoms due to paralysis of one or more of the eye muscles it controls. This is due to impairment in the superior oblique muscle , which is innervated by the trochlear nerve. Combined, these nerves provide sensation to the skin of the face and also controls the muscles of mastication chewing. This is where a person is unable to move the muscles on one or both sides of their face. In blunt trauma , the facial nerve is the most commonly injured cranial nerve. Strokes typically also

affect the seventh cranial nerve by cutting off blood supply to nerves in the brain that signal this nerve and so can present with similar symptoms. The vestibular part is responsible for innervating the vestibules and semicircular canal of the inner ear ; this structure transmits information about balance , and is an important component of the vestibuloocular reflex , which keeps the head stable and allows the eyes to track moving objects. The cochlear nerve transmits information from the cochlea , allowing sound to be heard. Function of the vestibular nerve may be tested by putting cold and warm water in the ears and watching eye movements caloric stimulation. Major effects of damage to the vagus nerve may include a rise in blood pressure and heart rate. Isolated dysfunction of only the vagus nerve is rare, but - if the conflict or lesion is located above the point at which the vagus first branches off - can be diagnosed by a hoarse voice, due to dysfunction of one of its branches, the recurrent laryngeal nerve. Damage to the accessory nerve XI will lead to ipsilateral weakness in the trapezius muscle. This can be tested by asking the subject to raise their shoulders or shrug, upon which the shoulder blade scapula will protrude into a winged position. A case with unilateral hypoglossal nerve injury in branchial cyst surgery. Damage to the nerve at lower motor neuron level may lead to fasciculations or atrophy of the muscles of the tongue. The fasciculations of the tongue are sometimes said to look like a "bag of worms". Upper motor neuron damage will not lead to atrophy or fasciculations, but only weakness of the innervated muscles. When damaged and extended, the tongue will move towards the weaker or damaged side, as shown in the image.

Chapter 5 : Cranial Nerves - Physiopedia

Many practitioners incorporate cranial nerve testing with their complete examination of the head and neck (see the Head and Neck section of this web site for details). A detailed description of the CN assessment is provided below.

Gait Sample Basic Normal Exam Documentation Documentation of a basic, normal neuro exam should look something along the lines of the following: The patient is alert and oriented to person, place, and time with normal speech. Sensation is intact bilaterally. Cranial nerves are intact. Cerebellar function is intact. Memory is normal and thought process is intact. No gait abnormalities are appreciated. Sample Detailed Normal Exam Documentation If you are documenting a more in-depth neurological exam, your corresponding documentation for a normal exam should look something along the lines of the following: Visual fields normal in all quadrants. Pupils are round, reactive to light and accommodation. Extraocular movements are intact without ptosis. V Facial sensation is intact to bilaterally to dull, sharp, and light touch stimuli. VII Facial muscle strength is normal and equal bilaterally. VIII Hearing is normal bilaterally. IX, X Palate and uvula elevate symmetrically, with intact gag reflex. XI Shoulder shrug strong, and equal bilaterally. XII Tongue protrudes midline and moves symmetrically. Plantar reflex is downward bilaterally. Sensation is intact bilaterally to pain and light touch. Two-point discrimination is intact. Finger-to-nose and heel-to-shin test normal bilaterally. Balances with eyes closed Romberg. Rapid alternating movements normal. Gait is steady with a normal base. Coordination is intact as measured by heel walk and toe walk. Abnormals on a neurological exam may include:

Chapter 6 : Cranial nerve, visual and hearing dysfunction in disorders of the CNS â€“ PM&R KnowledgeNo

See the written guide alongside the video here calendrierdelascience.com Download the app here: calendrierdelascience.com ðŸ‘¾.

Cranial nerve, visual and hearing dysfunction in disorders of the CNS Author s: Definition There are 12 pairs of cranial nerves emerging from the brain and radiating from its surface. Etiology Cranial nerves are injured before, during or after their passage through the skull as a result of compression from increased intracranial pressure, traction or transection, ischemic event from an infarct, or vascular occlusion. Epidemiology including risk factors and primary prevention The exact incidence of cranial nerve injuries is unknown. The following is the reported order of frequency after cranial nerve injury: Fracture of the sphenoid bone or compression of the optic nerve can result in unilateral blindness. Optic chiasm insults can lead to bitemporal hemianopsia. Basilar skull fracture, uncal herniation or compression can result in ptosis, inferolateral displacement of the ipsilateral eye and mydriasis. Fracture of the sphenoid wing, can result in injury which presents as extorsion of the ipsilateral eye with diplopia with attempted downward gaze, but improved diplopia with head tilted to contralateral side. Lesion in cavernous sinus can lead to asymmetry of jaw on opening or weakness with mastication. Injury caused to the cavernous sinus or fractures of the skull base can result in an extraocular palsy resulting by medial deviation of ipsilateral eye and diplopia that improves when the contralateral eye is abducted. Temporal bone fractures can cause immediate facial nerve injury resulting in paralysis of facial expression. A central lesion results in sparing of upper and frontal orbicularis occuli due to crossed innervation. Spared ability to raise eyebrows, wrinkle forehead helps differentiate a peripheral palsy from a central process. Temporal bone fractures can result in hearing loss, vertigo and nystagmus immediately after the injury. Injury to the posterior fossa or jugular foramen can cause numbness of ipsilateral pharynx and dysphagia with absent gag reflex. Injury can lead to ipsilateral drooping of soft-palate with uvular deviation towards the affected side and may present with dysphagia, dysphonia and decreased gag reflex. Damage can result in ipsilateral paralysis of sternocleidomastoid and trapezius. Injury can lead to tongue protrusion away from side of lesion. Disease progression including natural history, disease phases or stages, disease trajectory clinical features and presentation over time Most cranial nerve lesions are present at initial presentation in TBI. However, peripheral CN VII lesions may present as immediate paralysis usually due to a complete laceration of the nerve via skull fracture , or delayed, where a bone fragment compresses the nerve. Incomplete cranial nerve lesions often improve with time, while complete lesions are much less likely to recover. CN VII lesions may result in mobility impairments. A cognitively impaired patient may not describe loss of the ability to smell in a CN I lesion, but may develop anorexia. Inquiring about appetite and weight changes may raise suspicion for cranial nerve lesions that a cognitively impaired patient may otherwise have difficulty recognizing. Familiar, non-noxious smells CN II: Tympanic membrane, hearing, postural responses, nystagmus CN X: Gag reflex, palate elevation CN XI: Tongue deviation with protrusion, fasiculations, atrophy See Pathophysiology section for additional information regarding clinical presentation of cranial nerve dysfunction. Balance testing must also be emphasized. Additionally, one should examine swallowing function, in terms of safety and efficacy for nutritional purposes. Functional communication is important to consider, as well. Laboratory studies A suspected infectious etiology may warrant investigation with a complete blood count, cultures, erythrocyte sedimentation rate, serologies, cerebrospinal fluid CSF evaluation, or polymerase chain reaction evaluation of DNA. Analysis for autoimmune disease may be indicated, including detection of circulating antibodies in serum or CSF. Additionally, one may employ laboratory investigation for endocrine and electrolytic disorders. Imaging Utilization of imaging studies should be employed as directed by the history and physical examination. Computed Tomography CT has great utility in the acute setting after significant trauma, and can assist in assessment for temporal fractures, which are associated with Facial and Vestibulocochlear nerve injuries. If suspicion is raised for olfactory dysfunction, magnetic resonance imaging MRI often provides visual confirmation of abnormalities in the olfactory bulbs and tracts, as well as in the inferior frontal lobes. Supplemental assessment tools Evoked potentials, such as olfactory event-related

potentials, visual evoked responses, and brainstem auditory evoked potentials, provide further diagnostic information in the setting of cranial nerve associated dysfunction in smell, vision, and hearing, as well is in lower neurologically functioning individuals. Significant impending neurological compromise may be heralded acutely by pupillary fixation, and axonal injury to the optic nerve has a poor prognosis for recovery, given inability of those axons to regenerate. Vision following oculomotor nerve injury may begin to improve within two months of injury, and abnormal vision following isolated abducens palsy may resolve spontaneously. Visual, vestibular, or hearing dysfunction necessitates investigation into the structural environment of the home and workplace, with regard to safety and function. Furthermore, reduced upper extremity muscle strength may impair performance in the workplace or function in activities of daily living. Professional Issues Particular care and sensitivity must be undertaken to maximize quality of life following cranial nerve dysfunction, given propensity for significant functional impairment and newfound disability resulting from visual, auditory, vestibular, alimentary, or communicative deficits. Concomitantly, safety must be prioritized concerning impacted functional mobility, self-care, and nutrition, with paramount attention to prevention of further disability. However, thorough examination of cranial nerves is essential and re-examination as the patient recovers may reveal previously undiagnosed deficits. Proper swallowing evaluation prior to allowing oral intake is essential. In cases of a delayed CN VII palsy, treatment with high dose steroids with a slow taper minimizes the risk of a rebound paralysis. At different disease stages There are no established effective treatments for traumatic olfactory dysfunction, but anosmic patients showed slight recovery of olfaction with intranasal steroids. Visual deficits may result from optic nerve or extraocular muscle dysfunction. Depending on etiology, treatment may include patching, prisms, or ultimately neurolysis or surgical intervention. Lubrication and patching can protect the cornea when sensation and eye closure is impaired. Tarsorrhaphy may be performed if conservative measures are ineffective. Visual training may improve visual spatial disorders, balance, dizziness and posture. Specifically for cranial nerve lesions, vision specialists, audiologists and speech therapists can assist in evaluation and treatment. Occasional surgical referral may be indicated. Inability to detect smoke, gas, harmful chemicals or spoiled foods may cause harm. Reminders and fixed schedules can improve hygiene, child or pet care when olfaction is impaired. Dietary changes may be required to optimize nutrition in patients with dysgeusia, including use of spices and textures to improve the appeal of foods. Explaining the rationale for specific diets to families of patients with dysphagia is important, as this is often a source of confusion. Most cranial nerve deficits have been found to improve over the course of a few months to a year. Persistent or unexpected deficits may be evaluated with electrodiagnostic, vestibular and neuro-ophthalmologic studies described above. Visual, hearing, vestibular and swallowing difficulties may be related to cortical or cranial nerve deficits and impede progress in the rehabilitation setting. Recognizing and addressing these deficits may expedite functional gains in a broad range of modalities. Providing reassurance and compensatory strategies may reduce the psychological effects of the deficits. Also, in Phase 2 clinical trial is the use of transcranial alternating current stimulation to improve visual field size in patients with optic nerve damage. On physical exam of patients with brain injury, patients with altered cognition or consciousness it is difficult to elicit a full and accurate CN exam. Currently there are numerous treatment options for CN injures but no standardized treatment guidelines exist. A Textbook of Neuroanatomy. Manual of Traumatic Brain Injury Management. Demos Medical Pub; Accessed February 10, Clinical treatment of traumatic brain injury complicated by cranial nerve injury. Brain Injury Medicine Principles and Practice. Cranial nerve injury after minor head trauma. Neurovascular compression in cranial nerve and systemic disease. Medical Rehabilitation of Traumatic Brain Injury. MR and clinical evaluation. Traumatic brain injury assessed with olfactory event-related brain potentials. Anosmia following head trauma: Tohoku J Exp Med. Impact of olfactory impairment on quality of life and disability. Arch Otolaryngol Head Neck Surg. Clinical Measurement of Taste and Smell. Rehabilitation of visuospatial cognition and visual exploration in neglect: Evaluation and Treatment of Swallowing Disorders. Use of anticonvulsants for treatment of neuropathic pain.

Chapter 7 : Cranial nerve examination - OSCE Guide | Geeky Medics

There are 12 pairs of cranial nerves although the optic nerve is really an extension of the brain rather than a peripheral nerve. The ability to test them swiftly, efficiently and to interpret the findings should be a core competency for general practice.

Testing of motor and sensory function requires a basic understanding of normal anatomy and physiology. Voluntary movement begins with an impulse generated by cell bodies located in the brain. Signals travel from these cells down their respective axons, forming the Cortiospinal a. At the level of the brain stem, this motor pathway crosses over to the opposite side of the body and continue downward on that side of the spinal cord. A discussion of these tracts can be found in other Neurology reference texts. For more information about motor pathways, see the following link: The precise location of the synapse depends upon where the lower motor neuron is destined to travel. If, for example, the LMN terminates in the hand, the synapse occurs in the cervical spine i. The axons of the PNS travel to and from the periphery, connecting the organs of action e. Nerves which carry impulses away from the CNS are referred to Efferents i. Axons that exit and enter the spine at any given level generally connect to the same distal anatomic area. These bundles of axons, referred to as spinal nerve roots, contain both afferent and efferent nerves. For more information about spinal cord anatomy, see the following link: Review of Spinal Anatomy As the efferent neurons travels peripherally, components from different roots commingle and branch, following a highly programmed pattern. Ultimately, contributions from several roots may combine to form a named peripheral nerve, which then follows a precise anatomic route on its way to innervating a specific muscle. The Radial Nerve, for example, travels around the Humerus bone of the upper arm , contains contributions from Cervical Nerve Roots 6, 7 and 8 and innervates muscles that extend the wrist and supinate the forearm. It may help to think of a nerve root as an electrical cable composed of many different colored wires, each wire representing an axon. As the cable moves away from the spinal cord, wires split off and head to different destinations. Prior to reaching their targets, they combine with wires originating from other cables. The group of wires that ultimately ends at a target muscle group may therefore have contributions from several different roots. For more information about radial nerve anatomy and function, see below. Afferents carry impulses in the opposite direction of the motor nerves. That is, they bring information from the periphery to the spinal cord and brain. Sensory nerves begin in the periphery, receiving input from specialized receptor organs. The axons then move proximally, joining in a precise fashion with other axons to form the afferent component of a named peripheral nerve. The Radial Nerve, for example, not only has a motor function described previously but also carries sensory information from discrete parts of the hand and forearm. As the sensory neurons approach the spinal cord, they join specific spinal nerve roots. Each root carries sensory information from a discrete area of the body. The area of skin innervated by a particular nerve root is referred to as a dermatome. Dermatome maps describe the precise areas of the body innervated by each nerve root. These distributions are more or less the same for all people, which is clinically important. In the setting of nerve root dysfunction, the specific area supplied by that root will be affected. This can be mapped out during a careful exam see below , identifying which root s is dysfunctional. To view a dermatomal map, see the following link: Dermatome Map University of Scranton Sensory input travels up through the spinal cord along specific paths, with the precise route defined by the type of sensation being transmitted. Nerves carrying pain impulses, for example, cross to the opposite side of the spinal cord soon after entering, and travel up to the brain on that side of the cord. Vibratory sensations, on the other hand, enter the cord and travel up the same side, crossing over only when they reach the brain stem see following sections for detailed descriptions. For more information about sensory pathways, see the following link: University of Washington Review of Sensory Pathways Ultimately, the sensory nerves terminate in the brain, where the impulses are integrated and perception occurs. Understanding the above neruo-anatomic relationships and patterns of innervation has important clinical implications when trying to determine the precise site of neurological dysfunction. Injury at the spinal nerve root level, for example, will produce a characteristic loss of sensory and motor function. This will differ from that caused by a problem at

the level of the peripheral nerve. An approach to localizing lesions on the basis of motor and sensory findings is described in the sections which follow. Realize that there is a fair amount of inter-individual variation with regards to the specifics of innervation. Also, recognize that often only parts of nerves may become dysfunctional, leading to partial motor or sensory deficits. As such, the patterns of loss are rarely as "pure" as might be suggested by the precise descriptions of nerves and their innervations. Sensory Testing Sensory testing of the face is discussed in the section on Cranial Nerves. Testing of the extremities focuses on the two main afferent pathways: Spinothalamics and Dorsal Columns. These nerves detect pain, temperature and crude touch. They travel from the periphery, enter the spinal cord and then cross to the other side of the cord within one or two vertebral levels of their entry point They then continue up that side to the brain, terminating in the cerebral hemisphere on the opposite side of the body from where they began. These nerves detect position a. They travel from the periphery, entering the spinal cord and then moving up to the base of the brain on the same side of the cord as where they started. Upon reaching the brain stem they cross to the opposite side, terminating in the cerebral hemisphere on the opposite side of the body from where they began. A screening evaluation of these pathways can be performed as follows: To do this, break a Q-tip or tongue depressor in half, such that you create a sharp, pointy end. Alternatively, you can use a disposable needle or the sharp and blunt ends of a safety pin. I would discourage the use of the pointy, metal spikes that accompany some reflex hammers. Better to use a disposable implement. Ask the patient to close their eyes so that they are not able to get visual clues. Start at the top of the foot. Orient the patient by informing them that you are going to first touch them with the sharp implement. Then do the same with a non-sharp object e. This clarifies for the patient what you are defining as sharp and dull. Now, touch the lateral aspect of the foot with either the sharp or dull tool, asking them to report their response. If they give accurate responses, do the same on the other foot. The same test can be repeated for the upper extremities i. As such, it contributes to balance. Similar to the Spinothalamic tracts, disorders which affect this system tend to first occur at the most distal aspects of the body. Thus, proprioception is checked first in the feet and then, if abnormal, more proximally e. Ask the patient to close their eyes so that they do not receive any visual cues. With one hand, grasp either side of great toe at the interphalangeal IP joint. Place your other hand on the lateral and medial aspects of the great toe distal to the IP. Orient patient to up and down as follows: Testing Proprioception Alternately deflect the toe up or down without telling the patient in which direction you are moving it. They should be able to correctly identify the movement and direction. Both great toes should be checked in the same fashion. If normal, no further testing need be done in the screening exam. Similar testing can be done on the fingers. Vibratory sensation travels to the brain via the dorsal columns. Thus, the findings generated from testing this system should corroborate those of proprioception see above. Start at the toes with the patient seated. You will need a hz tuning fork. Grasp the tuning fork by the stem and strike the forked ends against the floor, causing it to vibrate. Place the stem on top of the interphalangeal joint of the great toe. Put a few fingers of your other hand on the bottom-side of this joint. Testing vibratory sensation Ask the patient if they can feel the vibration. You should be able to feel the same sensation with your fingers on the bottom side of the joint. The patient should be able to determine when the vibration stops, which will correlate with when you are no longer able to feel it transmitted through the joint. It sometimes takes a while before the fork stops vibrating. If you want to move things along, rub the index finger of the hand holding the fork along the tines, rapidly dampening the vibration. Repeat testing on the other foot. Patients should normally be able to distinguish simultaneous touch with 2 objects which are separated by at least 5mm. These stimuli are carried via the Dorsal Columns. While not checked routinely, it is useful test if a discrete peripheral neruropathy is suspected e. Testing can be done with a paperclip, opened such that the ends are 5mm apart. The patient should be able to correctly identify whether you are touching them with one or both ends simultaneously, along the entire distribution of the specific nerve which is being assessed. Special Testing for Early Diabetic Neuropathy: A careful foot examination should be performed on all patients with symptoms suggestive of sensory neuropathy or at particular risk for this disorder e.

Chapter 8 : Entire Volume - Nervous System: Cranial Nerves and Motor System | Bates' Visual Guide

The cranial nerve examination is often considered one of the most difficult OSCE stations, but with plenty of practice, you'll be fine. The important thing to remember is that in an OSCE you'll not be required to complete an entire cranial nerve exam in one station.

Corneal reflex test Motor Supply To test the motor supply, ask the patient to clench their teeth together, observing and feeling the bulk of the masseter and temporalis muscles. Ask the patient to then open their mouth against resistance. Finally perform the jaw jerk on the patient by placing your left index finger on their chin and striking it with a tendon hammer. This should cause slight protrusion of the jaw. Muscles of the head and neck Feeling the masseter muscles The jaw jerk Step 13 - Abducens Nerve As previously mentioned, the abducens nerve is tested in the same manner as the oculomotor and trochlear nerves, again in eye movements. Step 14 - The Facial Nerve The facial nerve supplies motor branches to the muscles of facial expression. This nerve is therefore tested by asking the patient to crease up their forehead raise their eyebrows , close their eyes and keep them closed against resistance, puff out their cheeks and reveal their teeth. Crease up the forehead Keep eyes closed against resistance Puff out the cheeks Reveal the teeth Step 15 - The Vestibulocochlear Nerve The vestibulocochlear nerve provides innervation to the hearing apparatus of the ear and can be used to differentiate conductive and sensori-neural hearing loss using the Rinne and Weber tests. A normal patient will find the second position louder. Normally it should be heard equally in both ears. Rinne test - place tuning fork on the mastoid process Rinne test - place tuning fork beside the ear Webers test - place the tuning fork base down in the centre of the forehead Step 16 - The Glossopharyngeal Nerve The glossopharyngeal nerve provides sensory supply to the palate. It can be tested with the gag reflex or by touching the arches of the pharynx. Glossopharyngeal nerve examination The vagus nerve provides motor supply to the pharynx. Asking the patient to speak gives a good indication to the efficacy of the muscles. Check that it lies centrally and does not deviate on movement. Step 18 - The Accessory Nerve The accessory nerve gives motor supply to the sternocleidomastoid and trapezius muscles. To test it, ask the patient to shrug their shoulders and turn their head against resistance. Sternocldeiomastoid muscle test against resistance Sternocleidomastoideus Trapezius muscle test against resistance Trapezius The Hypoglossal Nerve The hypoglossal nerve provides motor supply to the muscles of the tongue. Observe the tongue for any signs of wasting or fasciculations. Ask the patient to stick their tongue out. If the tongue deviates to either side, it suggests a weakening of the muscles on that side.

Chapter 9 : Documenting a Neuro Exam, Decoded | MidlevelU

Nervous: Cranial Nerves Exam. Setup; I: Olfactory; II: Optic; III-IV-VI: extraoculars; V: Trigeminal; VII: Facial; VIII: Vestibulocochlear; IX-X: Glossopharyngeal, Vagus.

The following is a summary of the cranial nerves and their respective functioning. Peripheral lesions are lesions of the cranial nerve nuclei, the cranial nerves or the neuromuscular junctions. Central lesions are lesions in the brainstem not involving a cranial nerve nucleus , cerebrum or cerebellum. If there is a lesion in the brainstem involving a cranial nerve nucleus along with other areas of the brain stem, then the lesion is considered both central and peripheral. Cranial Nerve I Evaluate the patency of the nasal passages bilaterally by asking the patient to breath in through their nose while the examiner occludes one nostril at a time. Once patency is established, ask the patient to close their eyes. Occlude one nostril, and place a small bar of soap near the patent nostril and ask the patient to smell the object and report what it is. Switch nostrils and repeat. Furthermore, ask the patient to compare the strength of the smell in each nostril. Very little localizing information can be obtained from testing the sense of smell. This part of the exam is often omitted, unless their is a reported history suggesting head trauma or toxic inhalation. Cranial Nerve II First test visual acuity by using a pocket visual acuity chart. Perform this part of the examination in a well lit room and make certain that if the patient wears glasses, they are wearing them during the exam. Have them repeat the test covering the opposite eye. If the patient has difficulty reading a selected line, ask them to read the one above. Note the visual acuity for each eye. Next evaluate the visual fields via confrontation. Face the patient one foot away, at eye level. Tell the patient to cover their right eye with their right hand and look the examiner in the eyes. Once this is understood, cover your left eye with your left hand the opposite eye of the patient and extend your arm and first 2 fingers out to the side as far as possible. Beginning with your hand and arm fully extended, slowly bring your outstretched fingers centrally, and notice when your fingers enter your field of vision. The patient should say now at the same time you see your own fingers. Repeat this maneuver a total of eight times per eye, once for every 45 degrees out of the degrees of peripheral vision. Repeat the same maneuver with the other eye. Using an ophthalmoscope, observe the optic disc, physiological cup, retinal vessels and fovea. This is followed by blurring of the optic disc margin and possibly retinal hemorrhages. Observe the diameter of the pupils in a dimly lit room. Note the symmetry between the pupils. Next, shine the penlight or opthalmoscope light into one eye at a time and check both the direct and consensual light responses in each pupil. Note the rate of these reflexes. If they are sluggish or absent, test for pupillary constriction via accommodation by asking the patient to focus on the light pen itself while the examiner moves it closer and closer to their nose. Normally, as the eyes accommodate to the near object the pupils will constrict. The test for accomodation should also be completed in a dimly lit room. End the evaluation of cranial nerves II and III by observing the pupils in a well lit room and note their size and possible asymmetry. Anisocoria is a neurological term indicating that one pupil is larger than another. Yet which pupil is abnormal? For example, if the right pupil is of a greater diameter than the left pupil in room light, is their a sympathetic lesion in the left eye or a parasympathetic lesion in the right eye? To determine this, observe and compare the asymmetry of the pupils in both bright and dim light. If the asymmetry is greatest in dim light than the sympathetic system is disrupted in the left eye, not allowing it to dilate in dim light, while the functioning right eye dilates even further in the dim light causing an increase in asymmetry. Conversely, if the asymmetry is greatest in bright light, then there is a parasympathetic lesion in the right eye. If the asymmetry remains the same in dim and bright light, then the anisocoria is physiologic. Ptosis is the lagging of an eyelid. It has 2 distinct etiologies. The III cranial nerve also innervates a much larger muscle that elevates the eye lid: Thus, disruption of either will cause ptosis. The ptosis from a III nerve palsy is of greater severity than the ptosis due to a lesion of the sympathetic pathway, due to the size of the muscles innervated. As an aside, the parasympathetics run with the III cranial nerve and are usually affected with an abnormal III cranial nerve. Anisocoria can only be produced if the efferent pathway of the pupillary light reflex is disrupted. A lesion of the afferent pathway along the II cranial does not yield anisocoria. To test for a lesion of the afferent pathway one must perform a "swinging light test".

To interpret this test one must understand that the level of pupillary constriction is directly related to the total "perceived" illumination the brain appreciates from both eyes. Therefore with an afferent lesion, "swinging" the light back and forth between the eyes rapidly will cause the pupils to change diameter when the light goes from the normal eye brain perceiving increased illumination to the abnormal eye brain perceiving less illumination. If both eyes are normal, no change would occur, because the total perceived illumination remains constant. Move the penlight slowly at eye level, first to the left and then to the right. Note extra-ocular muscle palsies and horizontal or vertical nystagmus. The limitation of movement of both eyes in one direction is called a conjugate lesion or gaze palsy, and is indicative of a central lesion. A gaze palsy can be either supranuclear in cortical gaze centers or nuclear in brain stem gaze centers. If the lesion is cortical, then only voluntary movement is absent and reflex movements are intact. Disconjugate lesions, where the eyes are not restricted in the same direction or if only one eye is restricted, are due to more peripheral disruptions: One exception to this rule is an isolated impairment of adduction of one eye, which is commonly due to an ipsilateral median longitudinal fasciculus MLF lesion. This lesion is also called an internuclear ophthalmoplegia INO. In INO, nystagmus is often present when the opposite eye is abducted. Gaze-evoked nystagmus nystagmus that is apparent only when the patient looks to the side or down may be caused by many drugs, including ethanol, barbiturates, and phenytoin Dilantin. Ethanol and barbiturates recreational or therapuetic are the most common cause of nystagmus. Dilantin may evoke nystagmus at slight overdoses, and opthalmoplegia at massive overdoses. Abnormal patterns of eye movements may help localize lesions in the central nervous system. Ocular bobbing is the rhythmical conjugate deviation of the eyes downward. Ocular bobbing is without the characteristic rapid component of nystagmus. This movement is characteristic of damage to the pons. Downbeat nystagmus including a rapid component may indicate a lesion compressing on the cervicomedullary junction such as a meningioma or chordoma. An electronystagmogram ENG may be ordered to characterize abnormal eye movements. The basis of this test is that the there is an intrinsic dipole in each eyeball the retina is negatively charged compared to the cornea. During an ENG, recording electrodes are placed on the skin around the eyes and the dipole movement is measured and eye movement is accurately characterized. Cranial Nerve V First, palpate the masseter muscles while you instruct the patient to bite down hard. Also note masseter wasting on observation. Next, test gross sensation of the trigeminal nerve. Tell the patient to close their eyes and say "sharp" or "dull" when they feel an object touch their face. Allowing them to see the needle before this examination may alleviate any fear of being hurt. Touch the patient above each temple, next to the nose and on each side of the chin, all bilaterally. Ask the patient to also compare the strength of the sensation of both sides. If the patient has difficulty distinguishing pinprick and light touch, then proceed to check temperature and vibration sensation using the vibration fork. One may warm it or cool it under a running faucet. Finally, test the corneal reflex using a large Q-tip with the cotton extended into a wisp. Ask the patient to look at a distant object and then approaching laterally, touch the cornea not the sclera and look for the eye to blink. Repeat this on the other eye. Some clinicians omit the corneal reflex unless there is sensory loss on the face as per history or examination, or if cranial nerve palsies are present at the pontine level. Cranial Nerve VII Initially, inspect the face during conversation and rest noting any facial asymmetry including drooping, sagging or smoothing of normal facial creases. Next, ask the patient to raise their eyebrows, smile showing their teeth, frown and puff out both cheeks. Note asymmetry and difficulty performing these maneuvers. Ask the patient to close their eyes strongly and not let the examiner pull them open. When the patient closes their eyes, simultaneously attempt to pull them open with your fingertips. Once again, note asymmetry and weakness. When the whole side of the face is paralyzed the lesion is peripheral. When the forehead is spared on the side of the paralysis, the lesion is central e. This is because a portion of the VII cranial nerve nucleus innervating the forehead receives input from both cerebral hemispheres. The portion of the VII cranial nerve nucleus innervating the mid and lower face does not have this dual cortical input. Hyperacusis increased auditory volume in an affected ear may be produced by damage to the seventh cranial nerve. This is because the seventh cranial nerve innervates the stapedius muscle in the middle ear which damps ossicle movements which decreases volume. With seventh cranial nerve damage this muscle is paralyzed and hyperacusis occurs.