National Imaging Associates, Inc.	
Clinical guidelines **BRAIN (HEAD) MRI** **BRAIN (HEAD) MRI with IAC (Internal Auditory Canal)**	**Original Date: September 1997**
CPT Codes: **70551, 70552, 70553, +0698T – Brain MRI** ~~**70540, 70542, 70543, +0698T – IAC**~~	**Last Revised Date: ~~May 2022~~ May 2023**
Guideline Number: NIA_CG_001	**Implementation Date: January 202~~43~~**

GENERAL INFORMATION

- *It is an expectation that all patients receive care/services from a licensed clinician. All appropriate supporting documentation, including recent pertinent office visit notes, laboratory data, and results of any special testing must be provided. If applicable: All prior relevant imaging results and the reason that alternative imaging cannot be performed must be included in the documentation submitted.*
- *Where a specific clinical indication is not directly addressed in this guideline, medical necessity determination will be made based on widely accepted standard of care criteria. These criteria are supported by evidence-based or peer-reviewed sources such as medical literature, societal guidelines and state/national recommendations*

INDICATIONS FOR BRAIN MRI

Brain MR/MRA are not approvable simultaneously unless they meet the criteria described below in the Indications for Brain MR/Brain MRA combination studies section. If there is a combination request* for an overlapping body part, either requested at the same time or sequentially (within the past 3 months) the results of the prior study should be:

- Inconclusive or show a need for additional or follow up imaging evaluation **OR**
- The office notes should clearly document an indication why overlapping imaging is needed and how it will change management for the patient.

(*Unless approvable in the combination section as noted in the guidelines)

For evaluation of headache[1-5]

- Chronic headache with a change in character/pattern (e.g., more frequent, increased severity, or duration)

~~* National Imaging Associates, Inc. (NIA) is a subsidiary of Magellan Healthcare, Inc.~~

- Cluster headaches or other trigeminal-autonomic cephalgias, i.e., paroxysmal hemicrania, hemicrania continua, short-lasting unilateral neuralgiform headache attacks (SUNCT/SUNA) imaging is indicated once to eliminate secondary causes[6]
- Acute headache, sudden onset:
    - With a personal or family history (brother, sister, parent, or child) of brain aneurysm or AVM (arteriovenous malformation) **OR**
    - < 48 hours of "worst headache in my life" or "thunderclap" headache.
        - Note: The duration of a thunderclap type headache lasts more than 5 minutes. Sudden onset new headache reaching maximum intensity within 2-3 minutes.
    - Prior history of stroke or intracranial bleed
    - Known coagulopathy or on anticoagulation
- New onset of headache with any of the following[1, 7, 8]:
    - Acute, new, or fluctuating neurologic deficits, such as sensory deficits, limb weakness, abnormal reflexes (pathological, asymmetric, hyperreflexia), speech difficulties, visual loss, lack of coordination, or mental status changes or with signs of increased intracranial pressure (papilledema). (See background)
    - History of cancer or significantly immunocompromised
    - Fever
    - Subacute head trauma
    - Pregnancy or puerperium[9, 10]
    - Age ≥ 50[1, 7, 11-13]
    - Severe unilateral headache with radiation to or from the neck, associated with suspicion of carotid or vertebral artery dissection
    - Related to activity or event (sexual activity, exertion, Valsalva, -position), new or progressively worsening[14]
    - Persistent or progressively worsening during a course of physician-directed treatment[1, 15, 16]

  Note: Neuroimaging warranted for atypical/complex migraine aura, but not for a typical migraine aura (see background)

- Special considerations in the pediatric population with persistent headache[17-19]:
    - Occipital location~~We~~
    - Age < 6 years
    - Symptoms indicative of increased intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting
    - Documented absence of family history of headache
    - Severe headache in a child with an underlying disease that predisposes to intracranial pathology (e.g., immune deficiency, sickle cell disease, neurofibromatosis, history of neoplasm, coagulopathy, hypertension, congenital heart disease)

For evaluation of neurologic symptoms or deficits[20]

- Acute, new, or fluctuating neurologic symptoms or deficits such as, sensory deficits, limb weakness, abnormal reflexes (pathological, asymmetric, hyperreflexia), speech difficulties, visual loss, lack of coordination, or mental status changes (see background)

For evaluation of known or suspected stroke or vascular disease[21-23]

- Known or suspected stroke with any acute, new, or fluctuating symptoms or deficits such as sensory deficits, limb weakness, speech difficulties, visual loss, lack of coordination, or mental status changes (see background)
- Suspected stroke with a personal or first-degree family history (brother, sister, parent, or child) of aneurysm or known coagulopathy or on anticoagulation
- Symptoms of transient ischemic attack (TIA) (episodic neurologic symptoms such as sensory deficits, limb weakness, speech difficulties, visual loss, lack of coordination, or mental status changes)
- Evaluation of suspected acute subarachnoid hemorrhage (SAH)
- Follow-up for known hemorrhage, hematoma, or vascular abnormalities

  Note: MRI is the study of choice for detecting cavernous malformations (CCM) and other low flow vascular malformations (see background). Follow-up imaging of known CCM should be done only to guide treatment decisions or to investigate new symptoms. First-degree relatives of patients with more than one family member with a CCM should have a screening MRI as well as genetic counseling[24-26]

- Suspected central venous thrombosis - see background[21, 27]
- ~~1 time s~~Screening for silent cerebral infarcts in early school age children and adults with HbSS sickle cell disease or HbSβ0 thalassemia[28]
- Evaluation of neurological signs or symptoms in sickle cell disease[29, 30]
- High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity >200[31, 32]

For evaluation of known or suspected trauma[33-35]

- Known or suspected trauma or injury to the head with documentation of one or more of the following acute, new, or fluctuating:
  - Focal neurologic findings
  - Motor changes
  - Mental status changes
  - Amnesia
  - Vomiting
  - Seizures
  - Headache
  - Signs of increased intracranial pressure
- Known coagulopathy or on anticoagulation
- Known or suspected skull fracture by physical exam and/or prior imaging

- Post concussive syndrome if persistent or disabling symptoms and ~~and imaging~~MRI has not been performed[36]
- Subacute or chronic traumatic brain injury with new cognitive and/or neurologic deficit

For evaluation of suspected brain tumor, mass, or metastasis[37, 38]

- Suspected brain tumor with any acute, new, or fluctuating neurologic symptoms or deficits such as sensory deficits, limb weakness, abnormal reflexes (pathological, asymmetric, hyperreflexia), speech difficulties, visual loss, lack of coordination, or mental status changes (see background)
- Suspected brain metastasis or intracranial involvement in patients with a history of cancer based on neurological symptoms or examination findings (may include new or changing lymph nodes)
- Lesion with atypical features for further evaluation or follow up
- ~~Histiocytic Neoplasms for screening and/or with neurological signs or symptoms~~[39, 40]
  - ~~Erdheim-Chester Disease~~
  - ~~Langerhans Cell Histiocytosis~~
  - ~~Rosai-Dorfman Disease)~~
- ~~Midline dermoid cysts/sinuses with concern for intracranial extension~~[34-37]
- Suspected Pituitary Tumors[39-42]
  - Neurologic findings (e.g., visual field deficit suggesting compression of the optic chiasm, diplopia, gaze palsy)
  - Suspected hypofunctioning pituitary gland based on hormonal testing
    - Hypopituitarism
    - Growth hormone deficiency
    - Hypogonadotropic hypogonadism [low sex hormones and gonadotropins (FSH/LH)][43]
      - Total testosterone persistently < 150 with low or normal LH/FSH i.e., severe secondary hypogonadism **OR**
      - Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; **AND**
        - Neurological signs or symptoms; **OR**
        - Other pituitary hormonal abnormalities; **OR**
        - Low free testosterone and consideration and ~~addressing~~ addressment of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use, or comorbid illness)
  - Suspected hyperfunctioning pituitary gland based on hormonal testing
    - Central hyperthyroidism (high TSH)
    - Cushing ~~disease~~ syndrome suspected (high ACTH (>5) with cortisol suppression on low or high dose dexamethasone suppression test)[44-47]~~)~~
    - Acromegaly/gigantism (high GH/IGF-1)
    - Elevated prolactin[48-50]

- ≥ 250 ng/mL **OR**
- ~~In the absence of another cause, e.g., stress, pregnancy, hypothyroidism, renal insufficiency, medication~~After evaluation for another cause (e.g., pregnancy, hypothyroidism, renal insufficiency, medication- see background)
    - ≥ 100 ng/mL **OR**
    - Persistently elevated **OR**
    - Neuroendocrine signs or symptoms (i.e., headache, galactorrhea, abnormal menses, infertility, or bitemporal hemianopsia) **OR**
    - Abnormal pituitary hormones (low testosterone/estrogen/ progesterone **AND** low or normal LH/FSH)
  - Central Diabetes Insipidus (low ADH)
  - Precocious puberty in a child (male < 9; female < 8), with hormonal studies suggesting a central cause [51]
  - Pituitary apoplexy with sudden onset of neurological and hormonal symptoms
- For screening for known non-CNS Cancer[52-61] - see background
  - Default screening for
    - Kidney cancer
    - Lung cancer
    - Merkel cell carcinoma
    - Mucosal melanoma of the head and neck, especially of the oral cavity
    - Poorly differential neuroendocrine cancer (Large or Small cell/Unknown primary of neuroendocrine origin)
  - Screening with preconditions
    - AML…… Suspicion of leukemic meningitis
    - Cutaneous melanoma…… Stage IIIC or higher
    - Testicular cancer-Seminoma…… High risk
    - Gestational Trophoblastic Neoplasia……~~…~~…… Pulmonary metastasis
    - Bladder cancer…… High risk, i.e., small cell
  - All other cancer if CNS symptoms present
- Histiocytic Neoplasms for screening and/or with neurological signs or symptoms[62, 63]
  - Erdheim-Chester Disease
  - Langerhans Cell Histiocytosis
  - Rosai-Dorfman Disease
- For screening of Hereditary Cancer Syndromes - see background
  - Li Fraumeni syndrome- Annually[64]
  - Von Hippel Lindau – Every 2 years, starting at age of 8 years[65]
  - Tuberous Sclerosis – Every 1-3 years, until the age of 25 years[66]
  - MEN1 – Every 3-5 years, starting at the age of 5 years[67]
  - NF-2- Brain IAC: Annually starting at the age of 10 years[68]
  - ~~o~~ Sturge Weber Syndrome: Once, after age 1 to rule out intracranial involvement; in patients <1 year, only if symptomatic[69]

-

For evaluation of known brain tumor, mass, or metastasis

- Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) ~~as per professional NCCN[31] society recommendations~~undergoing active treatment within the past year or as per surveillance imaging guidance for that cancer[38]
- Suspected recurrence with prior history of CNS cancer based on neurological symptoms or examination findings
- Follow-up of known low grade tumor (WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma)
    - For surveillance as per professional society recommendations~~as per NCCN~~[38]
    - If symptomatic, new/changing signs or symptoms or complicating factors
- Follow-up of known pituitary adenoma
    - New neuroendocrine signs or symptoms
    - Functioning adenoma - to assess response to treatment and 1-year follow-up after drug holiday[70-73]
    - Asymptomatic Macroadenoma (≥ 10mm) follow-up every 6-18 months, post-surgical follow-up every 1-2 years after surgery[74]
    - Asymptomatic, non-functioning Microadenoma < 10mm repeat in one year; if stable, repeat every 2-3 years[75]
- Follow-up of known pineal cyst (≥ 5mm) if there are atypical features or symptoms (e.g., headaches, gaze paresis, ataxia, papilledema, nausea/vomiting)[76, 77]
- Follow up of known Rathke cleft cyst[78, 79]
    - If no symptoms, MRI at 1/3/5 years to stability
    - With new neurological symptoms or atypical imaging features
    - Post treatment, yearly for 5 years
- Follow-up of known arachnoid cyst[80-83]
    - In patients < 4 years old, serial imaging is warranted
    - In patients > 4 years old, repeat imaging only if newly symptomatic, i.e., headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction
- Midline dermoid cysts/sinuses with concern for intracranial extension[41-43, 48]~~34-37~~
    - ~~~~
- Tumor monitoring in neurocutaneous syndromes as per tumor type
- Histiocytic Neoplasms to assess treatment response and surveillance of known brain lesions[62, 63, 84]
    - Erdheim-Chester Disease
    - Langerhans Cell Histiocytosis
    - Rosai-Dorfman Disease

Indications for combination studies for the initial pre-therapy staging of cancer, OR active monitoring for recurrence as clinically indicated, OR evaluation of suspected metastases[38]

- ≤ 5 concurrent studies to include CT or MRI of any of the following areas as appropriate depending on the cancer: Neck, Abdomen, Pelvis, Chest, Brain, Cervical Spine, Thoracic Spine or Lumbar Spine

For evaluation of known or suspected seizure disorder[85-90]

- New onset of an unprovoked seizure ~~in adults~~
- Newly identified change in seizure activity/pattern
- Known seizure disorder without previous imaging
- Medically refractory epilepsy

- ~~Imaging indications for new onset seizures in the pediatric population~~[74-77]
  - ~~Abnormal neurological exam, especially a postictal focal deficit~~
  - ~~Significant developmental delay~~
  - ~~Focal onset~~
  - ~~EEG shows focal or suspected structural abnormalities~~
  - ~~<1 year of age~~

Note: In the pediatric population, ~~I~~ **imaging is not indicated in simple febrile seizures** or in idiopathic focal or generalized epilepsy with typical features [BECTS, childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), and juvenile myoclonic epilepsy (JME)][87, 91-93]

For evaluation of suspected multiple sclerosis (MS)[94-97]

- For evaluation of patient with neurologic symptoms or deficits suspicious for MS with
  - A clinically isolated syndrome (optic neuritis, transverse myelitis, or brain stem syndrome); **OR**
  - Recurrent episodes of variable neurological signs or symptoms not attributable to another cause
- To demonstrate dissemination in time for diagnosis (every 6-12 months)

For evaluation of known multiple sclerosis (MS)[94, 97, 98]

- To establish a new baseline (no recent imaging, postpartum, or 3-6 months after switching disease modifying therapy)
- Prior to starting or switching disease-modifying therapy
- 6-month repeat scan in patients with MRI disease activity that is not associated with new cl~~h~~ ~~cl~~inical ~~activity~~ symptoms on a routine follow-up scan (~~ie~~i.e., Radiographically isolated syndrome)[99]
- Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years
- New signs or symptoms suggested of an exacerbation or unexpected clinical worsening

- Progressive Multifocal Leukoencephalopathy (PML) surveillance for patients on natalizumab (~~Tsyabri~~Tysabri)[100]
    - 12 months after the start of treatment in all patients
    - Further surveillance MRI scanning timing is based on risk
        - Annually, if anti-JCV antibody negative,
        - Every 3-4 months, if high risk of PML occurrence:
            - seropositive for JC virus and have been treated with natalizumab for ≥18 months **OR**
            - high anti-JC virus antibody index values (>0.9) **OR**
            - previously treated with immunosuppressive therapies
    - Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics

Note: In the pediatric population, use a similar scan frequency for disease and therapeutic monitoring. Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management.

For evaluation of known or suspected infectious or inflammatory disease (e.g., meningitis or abscess)[101, 102]

- Suspected intracranial abscess or brain infection with acute altered mental status or with positive lab findings (such as elevated WBCs) OR follow-up assessment during or after treatment completed
- Meningitis with positive signs and symptoms (such as fever, headache, mental status changes, stiff neck) **OR** with positive lab findings (such as elevated white blood cells or abnormal lumbar puncture fluid exam)
- Suspected encephalitis with headache and altered mental status or follow-up as clinically warranted
- Endocarditis with suspected septic emboli
- Suspected temporal arteritis in a patient ≥ 50 with temporal headache, abrupt visual changes, jaw claudication, temporal artery tenderness, constitutional symptoms or elevated ESR;[103-107] **AND**
    - Negative initial work-up (color Doppler ultrasonography or biopsy); **OR**
    - Atypical features, failure to response to treatment or concern for intracranial involvement

Note: Protocol should include high-resolution contrast-enhanced imaging the temporal artery

- Central Nervous System (CNS) involvement in patients with known or suspected vasculitis or autoimmune disease with abnormal inflammatory markers or autoimmune antibodies
- Suspected primary CNS vasculitis based on neurological signs and symptoms with completed infectious/inflammatory lab work-up[108, 109]

- Immunocompromised patient (e.g., transplant recipients, HIV with CD4<200, primary immunodeficiency syndromes, hematologic malignancies) with focal neurologic symptoms, headaches, behavioral, cognitive or personality changes
- Neurosarcoidosis[110-112]
    - Initial Evaluation:
        - Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) **OR**
        - Known history of sarcoidosis with neurological signs or symptoms
    - Follow-up of known neurosarcoidosis:
        - To assess treatment response
        - Worsening signs or symptoms

For evaluation of clinical assessment documenting cognitive impairment of unclear cause[113-115]

- Mental status score of either MMSE or MoCA of less than 26 or other similar mental status instruments*/formal neuropsychological testing showing at least mild cognitive impairment **AND** a completed basic metabolic workup (such as thyroid function testing, liver function testing, complete blood count, electrolytes, and B12)

*Other examples ~~include:~~include Mini-Cog, Memory Impairment Screen, Saint Louis University Mental Status Examination (SLUMS), Brief Alzheimer's Screen (BAS), Blessed Dementia Scale (BDS), Clinical Dementia Rating (CDR)[116, 117]

FDA labeling for the drug Aduhelm (for Alzheimer's disease) requires baseline imaging and monitoring with Brain MRI.[118, 119] Criteria for coverage includes the following:

- Baseline study within 1 year of initiating treatment unless the patient has a more recent exacerbation, traumatic event [e.g., falls, etc.], or co-morbidity necessitating an evaluation within one-month preceding initiation
- Prior to the 7th and 12th infusions
- Monitoring if radiographic severe Amyloid Related Imaging Abnormalities (ARIA) is suspected or observed

NOTE: Enhanced clinical vigilance for ARIA is recommended during the first 8 doses of treatment with Aduhelm, particularly during titration. If a patient experiences symptoms which could be suggestive of ARIA, clinical evaluation should be performed, including MRI testing if indicated.

For evaluation of movement disorders[120-125]

- For evaluation of suspected Parkinson's with atypical feature or unresponsive to levodopa
- For evaluation of new non-Parkinson neurological symptoms in known Parkinson's disease complicating the evaluation of the current condition
- For the evaluation of other movement disorder to exclude a structural lesion (i.e., suspected Huntington disease, chorea, atypical parkinsonian syndromes, hemiballismus, atypical dystonia)

Note: MRI not indicated in essential tremor, Tourette' syndrome, or isolated focal dystonia (e.g., blepharospasm, cervical dystonia, laryngeal dystonia, oromandibular dystonia, writer's dystonia)[121, 125, 126]

For evaluation of cranial nerve and visual abnormalities

- ~~Anosmia (loss of smell) or dysosmia documented by objective testing that is persistent and of unknown origin[105-107]~~
- Optic neuritis
- Abnormal eye findings on physical or neurologic examination (papilledema, pathologic nystagmus, optic atrophy, ocular nerve palsies, new onset anisocoria, visual field deficit, etc.)[127]

  Note: See background

- Binocular diplopia with concern for intracranial pathology[128] after comprehensive eye evaluation[129]
- Childhood strabismus with development delay or abnormal fundoscopic exam to rule out intracranial abnormalities[130, 131]
- Horner's syndrome with symptoms localizing the lesion to the central nervous system[132]
- Trigeminal neuralgia or neuropathy~~notably with an atypical presentation~~[5, 133, 134]
- Occipital Neuralgia to exclude a structural lesion, notably in atypical cases[135-137]
- Bell's Palsy- if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset[138]
- Hemifacial spasm[139]
- Other objective cranial nerve palsy (CN IX-XII)[140, 141]
- Bulbar symptoms, i.e., difficulty in chewing, weakness of the facial muscles, dysarthria, palatal weakness, dysphagia, and dysphonia and/or signs, i.e., atrophy and fasciculations of the tongue and absent gag reflex[142]
- Pseudobulbar symptoms, i.e., dysphagia, dysarthria, facial weakness, sudden, stereotyped emotional outbursts that are not reflective of mood and/or signs, i.e., spastic tongue and exaggerated gag/jaw jerk[143]

For evaluation of known or suspected congenital abnormality (such as craniosynostosis, neural tube defects)[144, 145]

- Known or suspected congenital abnormality with any acute, new, or fluctuating neurologic, motor, or mental status changes
- Evaluation of macrocephaly in an infant/child <18 with previously abnormal US, abnormal neurodevelopmental examination, signs of increased ICP or closed anterior fontanelle[146]
- Evaluation of microcephaly in an infant/child < 18
- Evaluation of craniosynostosis and other skull deformities. CT is preferred imaging to assess bony structures; MRI imaging is preferred to assess intracranial soft tissue
- Evaluation of the corticomedullary junction in Achondroplasia[147, 148]

- Cerebral palsy if etiology has not been established in the neonatal period, there is change in the expected clinical or developmental profile or concern for progressive neurological disorder[149, 150]
- X-linked Adrenoleukodystrophy[151]
    - Baseline MRI between 12 and 18 months old
    - Second MRI 1 year after baseline
    - MRI every 6 months between 3 and 12 years old
    - Annual MRI after 12 years old
- Prior treatment **OR** treatment planned for congenital abnormality

Note: For evaluation of known or suspected hydrocephalus please see section on CSF abnormalities.

Cerebral Spinal Fluid (CSF) Abnormalities

- Evaluation of suspected hydrocephalus with any acute, new, or fluctuating neurologic, motor, or mental status changes
- Known hydrocephalus†
- For initial evaluation of a suspected Arnold Chiari malformation†
- Follow-up imaging of a known type II or type III Arnold Chiari malformation. For Arnold Chiari type I, follow-up imaging only if new or changing signs/symptoms[152]
- Initial evaluation for a known syrinx or syringomyelia†
- Known or suspected normal pressure hydrocephalus (NPH)[153]
    - With symptoms of gait difficulty, cognitive disturbance, and urinary incontinence
- Follow-up shunt evaluation[154-157]
    - Post operativity if indicated based on underlying disease or pre-operative radiographic findings and/or
    - 6-12 months after placement and/or
    - With neurologic symptoms that suggest shunt malfunction
- Evaluation of known or suspected cerebrospinal fluid (CSF) leakage[158]
- Cisternography for intermittent and complex CSF rhinorrhea/otorrhea. CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay)[159, 160]
- Suspected spontaneous intra-cranial hypotension with distinct postural headache (other symptoms include nausea, vomiting, dizziness, tinnitus, diplopia neck pain or imbalance)[161, 162]
- CSF flow study for evaluation and management of CSF flow disorders[163, 164]

†Often congenital, but can present later in life; or less commonly acquired secondary to tumor, stroke, trauma, infection, etc.[165]

Pre-operative/procedural evaluation for brain/skull surgery

- Pre-operative evaluation for a planned surgery or procedure

Post-operative/procedural evaluation

- A follow-up study may be needed to help evaluate a patient's progress after treatment, procedure, intervention, or surgery. Documentation requires a medical reason that clearly indicates why additional imaging is needed for the type and area(s) requested.

Further evaluation of indeterminate findings on prior imaging (unless follow up is otherwise specified within the guideline):

- For initial evaluation of an inconclusive finding on a prior imaging report that requires further clarification.
- One follow-up exam of a prior indeterminate MR/CT finding to ensure no suspicious interval change has occurred. (No further surveillance unless specified as highly suspicious or change was found on last follow-up exam)

Other Indications for a Brain MRI

- Vertigo associated with any of the following[166-168]
  - Signs or symptoms suggestive of a CNS lesion (ataxia, visual loss, double vision, weakness, or a change in sensation)
  - Progressive unilateral hearing loss
  - Risk factors for cerebrovascular disease with concern for stroke
  - After full neurologic examination and vestibular testing with concern for central vertigo (i.e., skew deviation, vertical nystagmus, head thrust test, videonystagmography (VNG)/ electronystagmography (ENG))
- Diagnosis of central sleep apnea on polysomnogram
  - Children > 1 year[169]
  - Adults in the absence of heart failure, chronic opioid use, high altitude, or treatment emergent central sleep apnea **AND** concern for a central neurological cause (Chiari malformation, tumor, infectious/inflammatory disease) **OR** with an abnormal neurological exam[170]
- Syncope with clinical concern for seizure or associated neurological signs or symptoms[171, 172]
- Cyclical vomiting syndrome or abdominal migraine with any localizing neurological symptoms[173-175]
- Soft tissue mass of the head with nondiagnostic initial evaluation (ultrasound and/or radiograph)[176-178]
- Psychological changes with neurological deficits on exam or after completion of a full neurological assessment that suggests a possible neurologic cause[179]
- Global developmental delay or developmental delay with abnormal neurological examination in a child < 18 years[180, 181]
- Unexplained event (BRUE) formerly apparent life-threatening event (ALTE) in infants < 1 year with concern for neurological cause based on history and exam[182]

  Note: Imaging is not indicated in low-risk patients

- Bone Marrow Transplant (BMT)

- For initial workup of BMT (along with CT Chest[183], CT Sinus and CT Abdomen and Pelvis)[184]).

Indications for a Brain MRI with Internal Auditory Canal (IAC) (If only images of the IACs is needed w/o Brain imaging see Guideline Number: NIA_CG_014)

- Unilateral non-pulsatile tinnitus
- Pulsatile tinnitus
- Suspected acoustic neuroma (Schwannoma) or cerebellar pontine angle tumor with any of the following signs and symptoms: unilateral hearing loss by audiometry, headache, disturbed balance or gait, unilateral tinnitus, facial weakness, or altered sense of taste
- Suspected cholesteatoma
- Suspected glomus tumor
- Asymmetric sensorineural hearing loss on audiogram
- Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality[185-187] (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner ear.
- CSF otorrhea (MRI/Nuclear Cisternography -for intermittent leaks, CT for active leaks)[188]; there ~~CSF fluid~~ should be a high suspicion or ~~always be~~ confirmatory~~ed~~ CSF fluid~~with~~ laboratory testing (Beta-2 transferrin assay)
- Clinical suspicion of acute mastoiditis as a complication of acute otitis media with intracranial complications (i.e., meningeal signs, cranial nerve deficits, focal neurological findings, altered mental status)[189, 190]
- Bell's Palsy for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset[138]

Indications for MR Perfusion Imaging[191]

- Neurovascular disease
  - Assessment of ischemic penumbra in acute stroke
  - Assessment of cerebrovascular reserve
  - Further evaluation of known vascular abnormality (stenosis, malformation, vasospasm, vasculitis, Moya-Moya)
- Mass lesions
  - Differentiating tumor from tumor mimic
  - Differentiating glioblastoma from brain metastasis[192]
  - Discriminating low- from high-grade gliomas[193]
  - Differentiating recurrent brain tumors from radiation/chemo necrosis[194, 195]
  - Surgical planning

Indications for Combination Studies[21, 22]

Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging.

Exception: For approved indications as noted above and being performed in a child under 8 years of age who will need anesthesia for the procedure and there is a suspicion of concurrent intracranial pathology[196]

- **Brain MRI/Neck MRA***
  - Recent ischemic stroke or transient ischemic attack
  - Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits
- **Brain MRI/Brain MRA***
  - Recent ischemic stroke or transient ischemic attack
  - Thunderclap headache with continued concern for underlying vascular abnormality after initial negative brain imaging > 6 hours after onset[197-199]

    Note: Negative brain CT < 6 hours after headache onset excludes subarachnoid hemorrhage in neurologically intact patients[200]
  - Acute, sudden onset of headache with personal history of a vascular abnormality or first-degree family history of aneurysm
  - Headache associated with exercise, exertion, Valsalva -or sexual activity[6, 14]
  - Suspected venous thrombosis (dural sinus thrombosis) – Brain MRV see background
  - Neurological signs or symptoms in sickle cell patients
  - High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200 [30]
- **Brain MRI/Brain MRA/Neck MRA***
  - Recent stroke or transient ischemic attack (TIA)
  - Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits
- **Brain MRI with IAC/ Brain MRA/Neck MRA (any ~~combination)*~~combination)***
  - Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology[201, 202]

***Note:** MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA.

- **Brain MRI/Cervical MRI/Thoracic MRI (any combination)**
  - Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course, and where in the CNS the likely localization(s) is/are), and other available information, including prior imaging.

- For evaluation of neuromyelitis optica spectrum disorders (recurrent or bilateral optic neuritis; recurrent transverse myelitis)[203]
- For known MS, prior to the initiation or change of disease modification treatments and assess disease burden (to establish a new baseline)[204]
- Follow-up scans, including brain and spine imaging, if patients have known spine disease:
  - 6-12 months after starting/changing treatment
  - Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years

- **Brain MRI/Cervical MRI/Thoracic MRI/Lumbar MRI (any combination)**
  - For initial evaluation of a suspected Arnold Chiari malformation
  - Follow-up imaging of a known type II or type III Arnold Chiari malformation. For Arnold Chiari type I, follow-up imaging only if new or changing signs/symptoms[152, 205]
  - Oncological Applications (e.g., primary nervous system, metastatic)
    - Drop metastasis from brain or spine (see background)
    - Suspected leptomeningeal carcinomatosis (see background)[206]
    - Tumor evaluation and monitoring in neurocutaneous syndromes - See background
  - CSF leak highly suspected and supported by patient history and/or physical exam findings (known or suspected spontaneous (idiopathic) intracranial hypotension (SIH), post lumbar puncture headache, post spinal surgery headache, orthostatic headache, rhinorrhea or otorrhea, or cerebrospinal-venous fistula)
- **Brain MRI/Orbit MRI**
  - Optic neuropathy or unilateral optic disk swelling of unclear etiology to distinguish between a compressive lesion of the optic nerve, optic neuritis, ischemic optic neuropathy (arteritic or non-arteritic), central retinal vein occlusion or optic nerve ~~infitrative~~infiltrative disorders[207]
  - Bilateral optic disk swelling (papilledema) with visual loss[208]
  - Optic Neuritis
    - If atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery or recurrence)[209, 210]
    - If needed to confirm optic neuritis and rule out compressive lesions
  - Known or suspected neuromyelitis optica spectrum disorder with severe, recurrent, or bilateral optic neuritis[203]
  - Suspected retinoblastoma[211, 212]
- **Brain MRI/FACE/SINUS/NECK MRI** ~~Anosmia or dysosmia on objective testing that is persistent and of unknown origin[140, 211, 212]~~
  - Granulomatosis with polyangiitis (Wegener's granulomatosis) disease[213]
  - Trigeminal neuralgia or neuropathy with an atypical presentation (for evaluation of the extracranial nerve course) [140, 214] See background

- Bell's Palsy/hemifacial spasm for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset[138]
- Objective cranial nerve palsy (CN IX-XII) (for evaluation of the extracranial nerve course)[140, 141]

BACKGROUND

Brain (head) MRI is the procedure of choice for most brain disorders. It provides clear images of the brainstem and posterior brain, which are difficult to view on a CT scan. It is also useful for the diagnosis of demyelinating disorders (such as multiple sclerosis (MS) that cause destruction of the myelin sheath of the nerve). The evaluation of blood flow and the flow of cerebrospinal fluid (CSF) is possible with this non-invasive procedure.

MRI for Headache – Generally, magnetic resonance imaging is the preferred imaging technique for evaluating the brain parenchyma, and CT is preferable for evaluating subarachnoid hemorrhage. CT is faster and more readily available than MRI and is often used in urgent clinical situations. Neurologic imaging is warranted in patients with headache disorders along with abnormal neurologic examination results or predisposing factors for brain pathology. Contrast-enhanced MRI is performed for evaluation of inflammatory, infectious, neoplastic, and demyelinating conditions.

Headache timeframes and other characteristics – Generally, acute headaches are present from hours to days, subacute from days to weeks and chronic headaches for more than 3 months. Acute severe headaches are more likely to be pathological (e.g., SAH, cerebral venous thrombosis) than non-acute (e.g., migraine, tension-type). Headaches can also be categorized as new onset or chronic/recurrent. Non-acute new onset headaches do not require imaging unless there is a red flag as delineated above. Incidental findings lead to additional medical procedures and expense that do not improve patient well-being. Primary headache syndromes, such as migraine and tension headaches, are often episodic with persistent or progressive headache not responding to treatment requiring further investigation (e.g., new daily persistent headache). Imaging is indicated in chronic headaches if there is a change in the headache frequency (number of headaches episodes/month), duration of each episode, severity of the headaches or new characteristics, such as changing aura or associated symptoms.[1, 6, 215-217]

Migraine with aura[6, 7, 218] – The headache phase of a migraine is preceded and/or accompanied by transient neurological symptoms referred to as aura in at least a third of migraine attacks. The most common aura consists of positive and/or negative visual phenomena, present in up to 99% of the individuals. Somatosensory is the secondary most common type of aura (mostly paresthesia's in an upper limb and/or hemiface). Language/speech (mainly paraphasia and anomic aphasia) can also be affected. These neurological symptoms typically evolve over a period of minutes and may last up to 20 minutes or more. The gradual evolution of symptoms is thought to reflect spreading of a neurological event across the visual and somatosensory cortices. Characteristically, the aura usually precedes and

terminates prior to headache, usually within 60 minutes. In others, it may persist or begin during the headache phase. ICHD-3 definition of the aura of migraine with typical aura consists of visual and/or sensory and/or speech/language symptoms, but no motor, brainstem or retinal symptoms and is characterized by gradual development, duration of each symptom no longer than one hour, a mix of positive and negative features and complete reversibility. Atypical or complex aura includes motor, brainstem, monocular visual disturbances, or ocular cranial nerve involvement (hemiplegic migraine, basilar migraine/brainstem aura, retinal migraine, ophthalmoplegic migraine) and secondary causes need to be excluded. Additional features of an aura that raise concern for an underlying vascular etiology include late age of onset, short duration, evolution of the focal symptoms, negative rather than positive visual phenomenon, and history of vascular risk factors.

Neurological Deficits – Examples of abnormal reflexes related to upper motor neuron lesion/central pathology include hyperreflexia, clonus, Hoffman sign and Babinski, snout, palmar grasp, and rooting reflexes.

Visual loss has many possible etiologies, and MRI is only indicated in suspected neurological causes of visual loss based on history and exam. Visual field defects, such as bitemporal hemianopsia, homonymous hemianopsia, or quadranopsia, require imaging as well as does suspected optic nerve pathology. Subjective symptoms such as blurred vision or double vision with no clear correlate on neurological examination requires a comprehensive eye evaluation to exclude more common causes, such as cataracts, refractive errors, retinopathy, glaucoma, or macular degeneration. Transient visual loss with history consistent with TIA but normal exam at time of examination also should be imaged. Positive visual phenomena, such as photopsias or scintillations that march across the visual field, suggest migraine whereas negative phenomenon, such as shaded or blurred, is more characteristic of ischemia.

Table 1: Gait and brain imaging[219-224]

Gait	Characteristic	Work up/Imaging
Hemiparetic	**Spastic unilateral, circumduction**	**Brain and/or, Cervical spine imaging based on associated symptoms**
Diplegic	**Spastic bilateral, circumduction**	**Brain, Cervical and Thoracic Spine imaging**
Myelopathic	**Wide based, stiff, unsteady**	**Cervical and/or Thoracic spine MRI based on associated symptoms**
Ataxic	**Broad based, clumsy, staggering, lack of coordination, usually also with limb ataxia**	**Brain imaging**

Apraxic	**Magnetic, shuffling, difficulty initiating**	**Brain imaging**
Parkinsonian	**Stooped, small steps, rigid, turning en bloc, decreased arm swing**	**Brain Imaging**
Choreiform	**Irregular, jerky, involuntary movements**	**Medication review, consider brain imaging as per movement disorder Brain MR guidelines**
Sensory ataxic	**Cautious, stomping, worsening without visual input (ie + Romberg)**	**EMG, blood work, consider spinal (cervical or thoracic cord imaging) imaging based on EMG**
Neurogenic	**Steppage, dragging of toes**	**EMG, if there is foot drop, Lumbar spine MRI Pelvis MR appropriate evidence of plexopathy**
Vestibular	**Insecure, veer to one side, worse when eyes closed, vertigo**	**Consider Brain/IAC MRI as per GL**

Non-neurological causes of gait dysfunction include pain (antalgic), side effects of drugs (analgesic, antihistamines, benzos, psych meds, antihypertensives), visual loss, hearing impairment, orthopedic disorders, rheumatologic disorders, psychogenic, and cardiorespiratory problems (orthostasis).[220, 222-224]

MRI and recent stroke or transient ischemic attack – A stroke or central nervous system infarction is defined as "brain, spinal cord, or retinal cell death attributable to ischemia, based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury. … Ischemic stroke specifically refers to central nervous system infarction accompanied by overt symptoms, whereas silent infarction causes no known symptoms."[225] If imaging or pathology is not available, a clinical stroke is diagnosed by symptoms persisting for more than 24 hours. Ischemic stroke can be further classified by the type and location of ischemia and the presumed etiology of the brain injury. These include large-artery atherosclerotic occlusion (extracranial or intracranial), cardiac embolism, small-vessel disease and less commonly dissection, hypercoagulable states, sickle cell disease and undetermined causes.[226] TIAs in contrast, "are a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction on imaging."[227] On average, the annual risk of future ischemic stroke after a TIA or initial ischemic stroke is 3–4%, with an incidence as high as 11% over the next 7 days and 24–29% over the following 5 years. This has significantly decreased in the last half century due to advances in secondary prevention.[228]

Therefore, when revascularization therapy is not indicated or available in individuals with an ischemic stroke or TIA, the focus of the work-up is on secondary prevention. This includes noninvasive vascular imaging to identify the underlying etiology, assess immediate complications and risk of future stroke. The majority of stroke evaluations take place in the inpatient setting. Admitting TIA patients is

reasonable if they present within 72 hours and have an ABCD(2) score ≥3, indicating high risk of early recurrence, or the evaluation cannot be rapidly completed on an outpatient basis.[227] Minimally, both stroke and TIA should have an evaluation for high-risk modifiable factors, such as carotid stenosis atrial fibrillation as the cause of ischemic symptoms.[226] Diagnostic recommendations include neuroimaging evaluation as soon as possible, preferably with magnetic resonance imaging, including DWI; noninvasive imaging of the extracranial vessels should be performed, and noninvasive imaging of intracranial vessels is reasonable.[229]

Individuals with a history of stroke and recent work-up with new signs or symptoms indicating progression or complications of the initial CVA should have repeat brain imaging as an initial study. Individuals with remote or silent strokes discovered on imaging should be evaluated for high-risk modifiable risk factors based on the location and type of the presumed etiology of the brain injury.

Non-aneurysmal vascular malformations – Non-aneurysmal vascular malformations can be divided in low flow vascular malformations and high flow vascular malformations. Low flow vascular malformations include dural venous anomalies (DVA), cavernomas, and capillary telangiectasias. High flow vascular malformations include AVM and dural arteriovenous fistulas (dAVF). For low flow malformations, MRI is the study of choice. Limited medical literature is available to support vascular imagining (CTA or MRA). CTA plays a limited role in the assessment of cavernoma but may be used to demonstrate a DVA. MRA is not usually helpful in the assessment of cavernoma, capillary telangiectasia, and DVA. Vascular imaging is indicated in high flow vascular malformations.[230-232]

MRI and Central Venous Thrombosis – a MR Venogram is indicated for the definite evaluation of a central venous thrombosis/dural sinus thrombosis. The most frequent presentations are isolated headache, intracranial hypertension syndrome (headache, nausea/vomiting, transient visual obscurations, pulsatile tinnitus, CN VI palsy, papilledema),[233] seizures, focal neurological deficits, and encephalopathy. Risk factors are hypercoagulable states inducing genetic prothrombotic conditions, antiphospholipid syndrome and other acquired prothrombotic diseases (such as cancer), oral contraceptives, pregnancy, puerperium (6-weeks postpartum), infections, and trauma. COVID-19 infection is associated with hypercoagulability, a thromboinflammatory response, and an increased incidence of venous thromboembolic events (VTE).[234, 235] Since venous thrombosis can cause SAH, infarctions, and hemorrhage, parenchymal imaging with MRI/CT is also appropriate.[27, 236, 237]

Galactorrhea and MRI – Isolated galactorrhea without elevated prolactin (normoprolactinemic) is usually due to breast pathology, i.e., breast feeding, trauma, ill-fitting undergarments. Consider mammogram, breast ultrasound, and serial dilution of the individual's prolactin sample to correct for possible hook effect.[238, 239]

Chart 1: Causes of Hyperprolactinemia[240]

Physiological	1) Coitus 2) Exercise 3) Lactation 4) Pregnancy 5) Sleep 6) Stress
Pathological	*1) Hypothalamic-pituitary stalk damage* a) Granulomas b) Infiltrations c) Irradiation d) Rathke's cyst e) Trauma: pituitary stalk section, suprasellar surgery f) Tumors: craniopharyngioma, germinoma, hypothalamic metastases, meningioma, suprasellar pituitary mass extension *2) Pituitary* a) Acromegaly b) Idiopathic c) Lymphocytic hypophysitis or parasellar mass d) Macroadenoma (compressive) e) Macroprolactinemia f) Plurihormonal adenoma g) Prolactinoma h) Surgery i) Trauma *3) Systematic Disorders* a) Chest – neurogenic chest wall trauma, surgery, herpes zoster b) Chronic renal failure c) Cirrhosis d) Cranial radiation e) Epileptic seizures f) Polycystic ovarian disease g) Pseudocyesis
Pharmacological	1) Anesthetics 2) Anticonvulsant 3) Antihistamines (H_2) 4) Antihypertensives 5) Cholinergic agonist 6) Drug-induced hypersecretion 7) Catecholamine depletory

	8) Dopamine receptor blockers 9) Dopamine synthesis inhibitor 10) Estrogens: oral contraceptives, oral contraceptive withdrawal 11) Neuroleptics/antipsychotics

Table 2: MRI and staging screening in Non-CNS Cancers[53, 54, 56, 58]

(NON-BRAIN/CNS) CANCER	PRECONDITION
Cutaneous melanoma	Stage IIIC or higher, default staging screening ≥ stage IIIC, surveillance with periodic brain MRI up to 3 years even if asymptomatic without prior brain mets; and if prior brain mets, surveillance every 3-6 months up to 3 years
Testicular cancer-Seminoma	If high risk, such as beta HCG >5000IU/L, or multiple lung or visceral mets, choriocarcinoma, neurological symptoms, or AFP>10,000ng/ml
Merkel cell carcinoma	Default staging screening, but especially for high risk (≥stage IIIb, immunosuppression)
Lung cancer	Default staging screening brain MRI also for surveillance in small cell every 3 months for 2 years if they have had no prophylactic cranial radiation

Surveillance for trilateral heritable retinoblastoma (Pineoblastoma surveillance)
Brain MRI at the time of retinoblastoma diagnosis; some centers recommend a brain MRI every 6 months until 5 years old[241, 242]

MRI and Neurocutaneous Syndromes

- In NF-1, clinical evaluation appears to be more useful to detect complications than is screening imaging in asymptomatic individuals. Imaging is indicated in evaluation of suspected tumors based on clinical evaluation and for follow-up of known intracranial tumors.[243]
- Conversely in NF-2, routine MR imaging screening is always indicated, given the high prevalence of CNS tumors, especially vestibular schwannomas. In individuals with NF-2, routine screening brain/IAC imaging is indicated annually starting from age 10 if asymptomatic or earlier with clinical signs/symptoms. Most individuals with NF2 eventually develop a spinal tumor, most commonly schwannomas, but meningioma and ependymomas are also seen. Spinal imaging at baseline and every 2 to 3 years is also advised with more frequent imaging, if warranted, based on sites of tumor involvement.[68]
- In individuals with Tuberous Sclerosis, Brain MRI should be obtained every 1-3 years up until age 25 for surveillance for CNS abnormalities.[66]
- In Von Hippel Lindau Syndrome, imaging of the brain and spinal cord for hemangioblastomas is recommended every 2 years.[65]
- In Sturge Weber Syndrome, Brain MRI can rule out intracranial involvement only after age 1 and is recommended in individuals <1 year only if symptomatic.[69]

~~**MRI and Positron Emission Tomography (PET) for Chronic Seizures –** When MRI is performed in the evaluation of individuals for epilepsy surgery, almost a third of those with electrographic evidence of~~

~~temporal lobe epilepsy have normal MRI scans. Interictal positron emission tomography (PET) may be used to differentiate individuals with MRI-negative temporal lobe epilepsy.~~

Multiple Sclerosis[95, 244, 245] – The diagnosis of MS requires demonstration of lesions in the CNS disseminated in time and space and the absence of fever, infection, or other more likely etiologies. An expanding amount of available disease-modifying treatments are effective in slowing down disease progression, especially in the early stages. These treatments can have serious side effects and can be costly; therefore, the accurate and expeditious diagnosis of MS is critical.

The diagnosis of MS can be made on clinical presentation alone with 2 clinical attacks and objective clinical evidence of more than 2 lesions. Attacks may be individual-reported or objectively observed and must last for a minimum of 24 hours and be 30 days apart. However, corroborating magnetic resonance imaging (MRI) is the diagnostic standard and is used, as well, to rule out other disorders. Additionally, MRI findings can replace certain clinical criteria in a substantial number of individuals. In the revised McDonald Criteria, MRI findings can be used to establish dissemination in both time and space.

Table 3: Variable Symptoms and Signs of MS

Symptoms	***Signs***
Depressed mood	Ataxia
Memory loss/cognitive changes	Dysmetria
Dizziness or vertigo	Decreased sensation (pain, vibration, position)
Fatigue	Decreased strength
Hearing loss and tinnitus	Hyperreflexia, spasticity
Heat sensitivity (Uhthoff Phenomenon)	Nystagmus
Incoordination and gait disturbances	Lhermitte's sign
Sensory disturbances (dysesthesias, numbness, ~~paresthesias~~paresthesia's)	Visual defects (internuclear ophthalmoplegia, optic disc pallor, red color desaturation, reduced visual acuity)
Pain	
Urinary symptoms	
Visual disturbances (diplopia,	

oscillopsia)

Weakness

In the presence of a clear, clinically isolated syndrome such as optic neuritis, transverse myelitis, or brain stem syndrome, brain MRI is the next diagnostic step. MS can also have variable and often subjective symptoms that come and go (see Table 3). If there are recurrent episodes of variable neurological signs or symptoms not attributable to another cause with clinical concern for MS, imaging is warranted as well.

MRI and Neuromyelitis optica spectrum disorders (NMOSD)[203] – NMOSD are inflammatory disorders of the central nervous system characterized by severe, immune-mediated demyelination and axonal damage predominantly affecting the optic nerves and spinal cord, but also the brain and brainstem. NMOSD can be distinguished from multiple sclerosis and other inflammatory disorders by the presence of the aquaporin-4 (AQP4) antibody. Features of NMOSD include attacks of bilateral or sequential optic neuritis acute transverse myelitis and the area postrema syndrome (with intractable hiccups or nausea and vomiting). The evaluation of suspected NMOSD entails brain and spinal cord neuroimaging. In contrast to MS (in which spinal cord involvement tends to be incomplete and asymmetric), NMOSD have a longer extent of spinal cord demyelination generally involving three or more vertebral segments.

Temporal Arteritis – Giant cell arteritis (GCA) is an inflammatory disorder that should be considered in individuals over the age of 50 with the following signs or symptoms: new headaches, acute onset of visual disturbances (especially transient monocular visual loss), jaw claudication, constitutional symptoms, tenderness over the temporal artery, and elevated ESR and/or CRP. A diagnosis of polymyalgia rheumatica (PMR) is highly associated. Large vessel GCA denotes involvement of the aorta and its first-order branches, especially the subclavian arteries, and is common. Extra- and intracranial cerebral vasculitis can also be seen but is ~~more rare~~rarer, and strokes are related to vasculitis of extracranial cerebral arteries causing vertebral or internal carotid arteries stenosis. Gold standard for diagnosis of GCA is temporal artery biopsy. Color Doppler ultrasound (CDUS) can be used as a surrogate for temporal artery biopsy in some cases. High-resolution magnetic resonance imaging (MRI) can visualize the temporal arteries when used with contrast. The presence of clinical manifestations unusual in GCA should prompt consideration of alternative diagnoses. Examples of such include adenopathy, pulmonary infiltrates, digital cyanosis, ulceration or gangrene, mononeuritis multiplex, stroke in the distribution of the middle cerebral artery, glomerulitis, and/or rapidly rising creatinine.[103-107, 246]

MMSE – The Mini Mental State Examination (MMSE) is a tool that can be used to ~~systematically and thoroughly assess mental status~~assess mental status systematically and thoroughly. It is an 11-question measure that tests five areas of cognitive function: orientation, registration, attention and calculation, recall, and language. The MMSE has been the most commonly used measure of cognitive function in dementia research, but researchers have recognized that it is relatively insensitive and variable in mildly impaired individuals. The maximum score is 30. A score of 23 or lower is indicative of cognitive

impairment. The MMSE takes only 5-10 minutes to administer and is, therefore, practical to use repeatedly and routinely.

MoCA – The Montreal Cognitive Assessment (MoCA) was designed as a rapid screening instrument for mild cognitive dysfunction. It assesses different cognitive domains: attention and concentration, executive functions, memory, language, visuoconstructional skills, conceptual thinking, calculations, and orientation. MoCA differs from the MMSE mainly by including tests of executive function and abstraction, and by putting less weight on orientation to time and place. Ten of the MMSE's 30 points are scored solely on the time-place orientation test, whereas the MoCA assigns it a maximum of six points. The MoCA also puts more weight on recall and attention-calculation performance, while de-emphasizing language skill. Time to administer the MoCA is approximately 10 minutes. The total possible score is 30 points; a score of 26 or above is considered normal.

MRI and Movement disorders – Atypical parkinsonian syndromes include progressive supranuclear palsy (PSP), multiple system atrophy (MSA), corticobasal degeneration (CBD), and dementia with Lewy bodies.

Anosmia – Nonstructural causes of anosmia include post-viral symptoms, medications (Amitriptyline, Enalapril, Nifedipine, Propranolol, Penicillamine, Sumatriptan, Cisplatin, Trifluoperazine, Propylthiouracil). These should be considered prior to advanced imaging to look for a structural cause.

Anosmia and dysgeusia have been reported as common early symptoms in individuals with COVID-19, occurring in greater than 80 percent of individuals. For isolated anosmia, imaging is typically not needed once the diagnosis of COVID has been made given the high association. As such, COVID testing should be done prior to imaging.[247-249]

MRI Orbits, Face, and Neck MRI rather than MRI Brain is the mainstay for directly imaging the olfactory apparatus and sinonasal or anterior cranial fossa tumors that may impair or directly involve the olfactory apparatus.

~~Evaluation of olfactory function is essential to determine the degree of chemosensory loss and confirm the individual's complaint. It also allows monitoring of olfactory function over time, helps to detect malingerers, and to establish compensation for disability. The two general types of olfactory testing include psychophysical and electrophysiologic testing. Psychophysical tests are used for clinical evaluation of olfactory loss; whereas, electrophysiologic tests, such as electro-olfactogram (EOG) or odor event–related potentials (OERPs) are used for research purposes only.~~

~~Olfactory threshold tests rely on measuring detection thresholds of a specific odorant, such as phenyl ethyl alcohol (PEA) or butyl alcohol. Odor identification tests are quantitative tests in which individuals are asked to identify the odorants at the suprathreshold level. Examples include *The Connecticut odor identification, The University of Pennsylvania Identification Test (UPSIT) and the Cross-Cultural Smell Identification Test (CC-SIT)*. In Europe, a commonly used test is a threshold- and odorant-identification forced-choice test that uses odorant-impregnated felt-tipped pens (Sniffin' Sticks). A simple olfactory screening test using a 70% isopropyl alcohol pad as a stimulant has also been well described in the literature.[248]~~**Trigeminal Neuralgia (TN)** – According to the International Headache Society, TN is

defined as “a disorder characterized by recurrent unilateral brief electric shock-like pain, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli.”[6] Atypical features include bilateral, hearing loss, dizziness/vertigo, visual changes, sensory loss, numbness, pain > 2min, pain outside trigeminal nerve distribution and progression.[140, 214]

Occipital Neuralgia – According to the International Headache Society, occipital neuralgia is defined “Unilateral or bilateral paroxysmal, shooting or stabbing pain in the posterior part of the scalp, in the distribution(s) of the greater, lesser and/or third occipital nerves, sometimes accompanied by diminished sensation or ~~dysaesthesia~~dysesthesia in the affected area and commonly associated with tenderness over the involved nerve(s). Pain is eased temporarily by local ~~anaesthetic~~anesthetic block of the affected nerve(s). Occipital neuralgia must be distinguished from occipital referral of pain arising from the atlantoaxial or upper zygapophyseal joints or from tender trigger points in neck muscles or their insertions.”[6]

MRI for Macrocephaly – Consider ultrasound in infants with macrocephaly and a normal neurological examination, no evidence of increased ICP and an open anterior fontanelle. If head US is normal, the infant should be monitored closely.[250] The anterior fontanelle generally closes between 10 and 24 months of age, with 3% closing between 5-9 months and 11% after 24 months.[251]

MRI and Normal Pressure Hydrocephalus (NPH) – Although diagnosis can be made based on CT findings alone, MRI is more accurate for disclosing associated pathologies (such as cerebrovascular disease), excluding other potential etiologies and for detecting NPH typical signs of prognostic value. A CT scan can exclude NPH and is appropriate for screening purposes and in individuals who cannot undergo MRI.[153]

MRI and Vertigo – The most common causes of vertigo seen are benign paroxysmal positional vertigo (BPPV), vestibular neuronitis (VN) and Ménière’s disease. These peripheral causes of vertigo are benign, and treatment involves reassurance and management of symptoms. Central causes of vertigo, such as cerebrovascular accidents (CVAs), tumors and multiple sclerosis (MS), need to be considered if the individual presents with associated neurological symptoms, such as weakness, diplopia, sensory changes, ataxia, or confusion. Magnetic resonance imaging is appropriate in the evaluation of individuals with vertigo who have neurologic signs and symptoms, progressive unilateral hearing loss or risk factors for cerebrovascular disease. MRI is more appropriate than CT for diagnosing vertigo due to its superiority in visualizing the posterior portion of the brain, where most central nervous system disease that causes vertigo is found. A full neurologic and otologic evaluation including provocative maneuvers, vestibular function testing and audiogram can help evaluate vertigo of unclear etiology and differentiate between central and peripheral vertigo.

MRI and developmental delay – Significant developmental delay is defined as significant delay (more than two standard deviations below the mean) in one or more developmental domains: gross/fine motor, speech/language, cognition, social/personal, and activities of daily living. Isolated delay in social/language development is characteristic of autism spectrum disorders or hearing loss. Isolated delay in motor development is characteristic of cerebral palsy (a static encephalopathy) or myopathy.

Global developmental delay (GDD) is a subset of developmental delay defined as significant delay (by at least 2 SD's) in two or more developmental categories. Note that the term "GDD" is usually reserved for children <5 years old, whereas in older children >5 years, disability is quantifiable with IQ testing. The yield of magnetic resonance imaging is low in children with autism spectrum disorder and no other neurologic findings; therefore, MRI is not recommended as a part of routine evaluation.[252]

Low risk brief resolved unexplained event (BRUE) formerly apparent life-threatening event (ALTE) requires all the following:

- Age > 60 days
- Gestational age ≥ 32 weeks or older and corrected gestational age ≥ 45 weeks
- First brief event
- Event lasting < 1 minute
- No CPR required by the trained medical provider
- No concerning historical features or physical examination findings.

Combination MRI/MRA of the Brain – This is one of the most misused combination studies and other than what is indicated above these examinations should be ordered in sequence, not together. Vascular abnormalities can be visualized on the brain MRI.

Individuals presenting with a new migraine with aura (especially an atypical or complex aura) can mimic a transient ischemic attack or an acute stroke. If there is a new neurologic deficit, imaging should be guided by concern for cerebrovascular disease, not that the individual has a headache.[11, 197]

Leptomeningeal Carcinomatosis[253-256] – Leptomeningeal metastasis is an uncommon and typically late complication of cancer with poor prognosis and limited treatment options. Diagnosis is often challenging with nonspecific presenting symptoms ranging from headache and confusion to focal neurologic deficits such as cranial nerve palsies. Standard diagnostic evaluation involves a neurologic examination, MRI of the brain and spine with gadolinium, and cytologic evaluation of the cerebral spinal fluid (CSF). Hematologic malignancies (leukemia and lymphoma), primary brain tumors as well as solid malignancies can spread to the leptomeninges. The most common solid tumors giving rise to LM are breast cancer (12 - 35 %), small and non-small cell lung cancer (10-26 %), melanoma (5 -25 %), gastrointestinal malignancies (4-14 %), and cancers of unknown primary (1-7 %).

Drop Metastases – Drop metastases are intradural extramedullary spinal metastases that arise from intracranial lesions. Common examples of intracranial neoplasms that result in drop metastases include pineal tumors, ependymomas, medulloblastomas, germinomas, primitive neuroectodermal tumors (PNET), glioblastomas multiform, anaplastic astrocytomas, oligodendrogliomas and less commonly choroid plexus neoplasms and teratomas.[257]

POLICY HISTORY

Date	Summary

April 2023	Updated and reformatted references
	Updated background section
	Added:
	- Indeterminate imaging section
	- Follow-up of known Rathke cleft cyst
	  - If no symptoms, MRI at 1/3/5 years to stability
	  - With new neurological symptoms or atypical imaging features
	- Post treatment, yearly for 5 years
	Clarified:
	- Abnormal reflexes *(pathological, asymmetric, hyperreflexia)*
	- New onset headache - Related to activity or event (sexual activity, *exertion, Valsalva*, position), new or progressively worsening
	- Post concussive syndrome if persistent or disabling symptoms and *MRI* has not been performed
	- *Screening* for silent cerebral infarcts in early school age children and adults with *HbSS sickle cell disease or HbSβ0 thalassemia*
	- Cushing syndrome *suspected (high ACTH (>5) with cortisol suppression on low or high dose dexamethasone suppression test)*
	- Elevated prolactin after evaluation for another cause – neuroendocrine signs or symptoms (i.e., headache, galactorrhea, abnormal menses, infertility, or bitemporal hemianopsia) and/or *abnormal pituitary hormones (low testosterone /estrogen/ progesterone AND low or normal LH/FSH)*
	- Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; AND Low free testosterone and *consideration and addressment* of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use, or comorbid illness)
	- Tumor surveillance *as per professional society recommendations*
	- Note: In the pediatric population, imaging is not indicated in simple febrile seizures *or in idiopathic focal or generalized epilepsy with typical features [BECTS, childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), and juvenile myoclonic epilepsy (JME)]*
	- 6-month repeat scan in patients with MRI disease activity that is not associated with *new clinical symptoms on a routine follow-up scan (i.e. Radiographically isolated syndrome)*
	- Indications for MR Perfusion Imaging section
	- Brain MRI/Brain MRA – Headache associated with exercise, *exertion, Valsalva* or sexual activity
	Deleted:

	~~Pediatric seizure indications and combined with adult~~ ~~Anosmia (loss of smell) or dysosmia documented by objective testing that is persistent and of unknown origin (also in combo section)~~
~~May 2022~~	~~Updated and reformatted references~~ ~~Updated background section~~ ~~Combo statements added~~ ~~Reorganized indications~~ ~~Changed visual deficits section added to background~~ ~~Reorganized suspected tumor section~~ ~~Clarified:~~ • ~~Acute headache, sudden onset~~ • ~~New onset headache related to activity or event (sexual activity, exertion, position), new or progressively worsening~~ • ~~Visual loss in background/removed note~~ • ~~Low flow vascular malformations~~ • ~~Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) for screening and/or with neurological signs or symptoms~~ • ~~Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH;~~ ○ ~~*Low free testosterone* and consideration of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use or comorbid illness)~~ • ~~Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) as per NCCN~~ • ~~Tumor monitoring in neurocutaneous syndromes as per tumor type~~ • ~~Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) To assess treatment response and surveillance of known brain lesions~~ • ~~To demonstrate dissemination in time for diagnosis (every 6-12 months)~~ • ~~To establish a new baseline (3-6 months after switching disease modifying therapy)~~ • ~~PML surveillance - Every 3-4 months, if high risk of PML occurrence; Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics~~ • ~~Examples of mental status instruments to screen for cognitive impairment~~ • ~~For evaluation of new non-Parkinson neurological symptoms~~ • ~~Binocular diplopia with concern for intracranial pathology after comprehensive eye evaluation~~

	• Trigeminal neuralgia or *neuropathy*, notably with an atypical presentation • **MRI Brain/MRI Orbit Combo –** Optic Neuritis if atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery, or recurrence • **MRI Brain/MRI Face/Sinus/Neck Combo** Trigeminal neuralgia or neuropathy with an atypical presentation (for evaluation of the extracranial nerve course) Added: • Abnormal reflexes to neurologic deficit sections • 1-time screening for silent cerebral infarcts in school age children and adults with sickle cell disease • High stroke risk in sickle cell patients (2 – 16 years of age) with a transcranial doppler velocity > 200 • Midline dermoid cysts/sinuses with concern for intracranial extension • Elevated prolactin in the absence of other cause: ≥ 100, persistently elevated or neuroendocrine signs or symptoms • Follow-up of known low grade tumor (WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma) ○ For surveillance as per NCCN ○ If symptomatic, new/changing signs or symptoms or complicating factors • 6-month repeat scan in patients with MRI disease activity that is not associated with clinical activity on a follow-up scan (MS) • Note about pediatric MS imaging – same as adults except Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management • Neurosarcoid ○ Initial Evaluation: ▪ Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) OR ▪ Known history of sarcoidosis with neurological signs or symptoms ○ Follow-up of known neurosarcoidosis: ▪ To assess treatment response ▪ Worsening signs or symptoms • Tourette syndrome to list of movement disorders in which MRI is not indicated • Occipital Neuralgia

	• ~~X-linked Adrenoleukodystrophy~~
	  ○ ~~Baseline MRI between 12 and 18 months old~~
	  ○ ~~Second MRI 1 year after baseline~~
	  ○ ~~MRI every 6 months between 3 and 12 years old~~
	  ○ ~~Annual MRI after 12 years old~~
	• ~~Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner.~~
	• ~~Pulsatile tinnitus to combo section (MRI Brain with IAC/MRA Head/MRA Neck)~~
	• ~~**General Combo statement**~~
	~~Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging.~~
	• ~~**Combo Brain MRI/MRA**:~~
	  ○ ~~Neurological signs or symptoms in sickle cell patients~~
	  ○ ~~High stroke risk in sickle cell patients (2 – 16 years of age) with a transcranial doppler velocity > 200~~
	• ~~**Brain MRI with IAC/ Brain MRA/Neck MRA (any combination)**~~
	  ○ ~~Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology~~
	  ○ ~~Note: MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA.~~
	• ~~**MRI Brain/MRI Face/Sinus/Neck Combo**~~
	  ○ ~~Bell's Palsy/hemifacial spams for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset~~
	• ~~**MRI Brain/Spine Combo section**~~
	  ○ ~~Drop metastasis from brain or spine~~
	  ○ ~~Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course,~~

	~~and where in the CNS the likely localization(s) is/are), and other available information, including prior imaging~~ ~~Changed:~~ • ~~Thunderclap headache with continued concern for underlying vascular abnormality after initial negative brain imaging > 6 hours after onset (as well as in combo Brain MRI/MRA)~~ ~~Deleted:~~ • ~~Precocious puberty: and evidence of an accelerated bone age on x-y~~ • ~~Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years~~ • ~~Follow-up of known meningioma section/background~~
~~November 2021~~	~~Added +0698T.~~
~~July 2021~~	~~**Reordered Indications**~~ ~~**Updated references**~~ ~~**Updated background section**~~ ~~**Added**~~ • ~~Brain MR/MRA are not approvable simultaneously unless they meet the criteria described below in the Indications for Brain MR/Brain MRA combination studies section.~~ • ~~Cluster headaches or other trigeminal-autonomic cephalgias i.e. paroxysmal hemicrania, hemicrania continua, short-lasting unilateral neuralgiform headache attacks (SUNCT/SUNA) imaging is indicated once to eliminate secondary causes (IHS, 2018)~~ • ~~Note: MRI is the study of choice for detecting cavernous malformations (CCM). Follow-up imaging of known CCM should be done only to guide treatment decisions or to investigate new symptoms. First-degree relatives of patients with more than one family member with a CCM should also have a screening MRI as well as genetic counseling~~ • ~~Langerhans cell histiocytosis with visual, neurological, or endocrine abnormality; polyuria or polydipsia; suspected craniofacial bone lesions, aural discharge, or suspected hearing impairment/mastoid involvement~~ • ~~Langerhans cell histiocytosis- To assess treatment response and surveillance of known brain lesions~~ • ~~Progressive Multifocal Leukoencephalopathy (PML) surveillance for patients on natalizumab (Tsyabri)~~ ○ ~~12 months after the start of treatment in all patients~~ ○ ~~Further surveillance MRI scanning timing is based on anti-JCV antibody status~~

	~~▪ If anti-JCV antibody negative, annually~~ ~~▪ If anti-JCV antibody positive and antibody index < 1.5, every 6 months~~ ~~▪ If anti-JCV antibody positive and antibody index > 1.5, every 3-4 months~~ • ~~Temporal Arteritis: Note: Protocol should include high-resolution contrast-enhanced imaging the temporal artery~~ • ~~similar mental status instruments */formal neuropsychological *Other examples include Ottawa 3DY (O3DY), Brief Alzheimer's Screen (BAS), Blessed Dementia Scale (BDS), caregiver-completed AD8 (cAD8), Brief Cognitive Rating Scale (BCRS), Clinical Dementia Rating (CDR) (Carptenter, 2011; McDougall, 1990)~~ • ~~FDA labeling for the drug Aduhelm (for Alzheimer's disease) requires baseline imaging and monitoring with Brain MRI. Criteria for coverage includes the following:~~ ~~o Baseline study within 1 year of initiating treatment unless the patient has a more recent exacerbation, traumatic event [e.g., falls, etc.], or co-morbidity necessitating an evaluation within one-month preceding initiation~~ ~~o Prior to the 7th and 12th infusions~~ ~~o Monitoring if radiographic severe Amyloid-Related Imaging Abnormalities (ARIA) is suspected or observed~~ ~~NOTE: Enhanced clinical vigilance for ARIA is recommended during the first 8 doses of treatment with Aduhelm, particularly during titration. If a patient experiences symptoms which could be suggestive of ARIA, clinical evaluation should be performed, including MRI testing if indicated.~~ • ~~Optic atrophy as an abnormal eye finding~~ • ~~Childhood strabismus with development delay or abnormal fundoscopic exam to rule out intracranial abnormalities~~ • ~~Bulbar symptoms ie. difficulty in chewing, weakness of the facial muscles, dysarthria, palatal weakness, dysphagia, and dysphonia and/or signs i.e. atrophy and fasciculations of the tongue and absent gag reflex~~ • ~~Pseudobulbar symptoms i.e. dysphagia, dysarthria, facial weakness, sudden, stereotyped emotional outbursts that are not reflective of mood and/or signs i.e. spastic tongue and exaggerated gag/jaw jerk~~ • ~~Evaluation of the corticomedullary junction in Achondroplasia~~ • ~~Evaluation of suspected hydrocephalus with any acute, new, or fluctuating neurologic, motor, or mental status changes (separated this from known hydrocephalus)~~

	• ~~Cisternography for intermittent and complex CSF rhinorrhea/otorrhea. CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay).~~ • ~~Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits to Brain MRI/Brain MRA/Neck MRA combo~~ • ~~Headache associated with exercise or sexual activity (Brain MRI/Brain MRA combo)~~ • ~~Pre-operative evaluation for a planned surgery or procedure~~ **~~Brain MRI/ Cervical MRI/Thoracic MRI (any combination)~~** ○ ~~For evaluation of neuromyelitis optica spectrum disorders (recurrent or bilateral optic neuritis; recurrent transverse myelitis)~~ ○ ~~For known MS, prior to the initiation or change of disease modification treatments and assess disease burden (to establish a new baseline)~~ ○ ~~Follow-up scans for known MS if patients have known spine disease:~~ ▪ ~~6-12 months after starting/changing treatment~~ ▪ ~~Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years~~ **~~Brain MRI/ Cervical MRI/Thoracic MRI/Lumbar (any combination)~~** • ~~Follow up imaging of a known Arnold Chiari malformation (II/III). For Chiari, I follow-up imaging only if new or changing signs/symptoms~~ ○ ~~Suspected Leptomeningeal carcinomatosis (LC)~~ ○ ~~Tumor evaluation and monitoring in neurocutaneous syndromes – See Background~~ ○ ~~CSF leak highly suspected and supported by patient history and/or physical exam findings (known or suspected spontaneous (idiopathic) intracranial hypotension (SIH), post lumbar puncture headache, post spinal surgery headache, orthostatic headache, rhinorrhea or otorrhea, or cerebrospinal-venous fistula)~~ **~~Brain MRI/Orbit MRI~~** ~~Optic Neuritis- If needed to confirm optic neuritis and rule-out compressive lesions~~ **~~Clarified~~** • ~~Symptoms indicative of increased intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting~~

	• ~~Suspected stroke with a personal or first-degree family history (brother, sister, parent, or child) of aneurysm or known coagulopathy or on anticoagulation~~ • ~~Symptoms of transient ischemic attack (TIA) (episodic neurologic symptoms such as sensory deficits, limb weakness, speech difficulties, visual loss, lack of coordination, or mental status changes)~~ • ~~Known or suspected skull fracture by physical exam and/or prior imaging~~ • ~~Neurologic findings (e.g. visual field deficit suggesting compression of the optic chiasm, diplopia, gaze palsy) – Pituitary~~ • ~~Follow-up known of pituitary adenoma - New neuroendocrine signs or symptoms~~ • ~~Follow of known arachnoid cyst (Al-Holou, 2010, 2013; Mustansir, 2018)~~ ○ ~~> 4 years old, repeat imaging only if newly symptomatic i.e. headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction.~~ • ~~Temporal Arteritis - Atypical features, failure to response to treatment or concern for intracranial involvement~~ • ~~Central Nervous System (CNS) involvement in patients with known or suspected vasculitis or autoimmune disease with abnormal inflammatory markers or autoimmune antibodies~~ • ~~Suspected primary CNS vasculitis based on neurological signs and symptoms with completed infectious/inflammatory lab work-up~~ • ~~Anosmia or dysosmia on objective testing that is persistent and of unknown origin (also in combo section)~~ • ~~Trigeminal Neuralgia or other trigeminal autonomic cephalgias, notably in those with atypical presentation (also in combo section)~~ • ~~Clarified age < 18 for imaging of microcephaly and macrocephaly~~ • ~~For initial evaluation of a suspected Arnold Chiari malformation~~ • ~~For follow up imaging of a known Arnold Chiari malformation (II/III). For Chiari I follow-up imaging only if new or changing signs/symptoms~~ • ~~After full neurologic examination and vestibular testing with concern for central vertigo (i.e. skew deviation, vertical nystagmus, head thrust test, videonystagmography (VNG)/electronystagmography (ENG))~~ • ~~Clarified age < 18 for imaging of developmental delay~~ • ~~Brain with IAC – CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay).~~ • ~~Optic neuropathy or unilateral optic disk swelling of unclear etiology (Brain MRI/Orbit MRI)~~

	~~**Deleted**~~ • ~~Approved indications as noted above and being performed in a child under 8 years of age who will need anesthesia for the procedure and there is a suspicion of concurrent vascular and intracranial pathology (redundant)~~ • ~~Brain MRI/Cervical MRI combo section (included in Brain MRI/ Cervical MRI/Thoracic MRI/Lumbar combos)~~
~~May 2020~~	~~Clarified:~~ • ~~New onset headache with (neurologic deficit) or with signs of increased intracranial pressure (papilledema)~~ • ~~Special additional considerations in the pediatric population with persistent headache~~ ○ ~~Documented absence of family history of headache~~ • ~~For evaluation of known or suspected stroke or vascular disease:~~ • ~~Suspected brain tumor~~ • ~~Suspected brain metastasis or intracranial involvement in patients with a history of cancer based on neurological symptoms or examination findings~~ • ~~Follow up of known malignant brain tumor~~ ~~Clarified:~~ • ~~Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years~~ • ~~Follow up of known non-malignant brain tumor/lesion if symptomatic, new/changing signs or symptoms or complicating factors~~ • ~~New onset of an unprovoked seizure in adults~~ • ~~Suspected intracranial abscess or brain infection~~ • ~~Suspected Encephalitis with headache and altered mental status or follow-up as clinically warranted~~ • ~~Mental status score of either MMSE or MoCA of less than 26 or other similar mental status instruments/neuropsychological testing~~ ~~Clarified:~~ • ~~Anosmia (loss of smell) documented by objective testing that is persistent and of unknown origin~~

	• ~~Chiari malformation/syrinx Often congenital, but can present later in life; or less commonly acquired secondary to tumor, stroke, trauma, infection etc.~~
	• ~~Vertigo associated with any of the following~~
	  ○ ~~Risk factors for cerebrovascular disease with concern for stroke~~
	  ○ ~~After full neurologic examination and vestibular testing with concern for central vertigo~~
	• ~~Combo Brain MRI/Orbit MRI~~
	  ○ ~~Reworded: Unilateral optic disk swelling/optic neuropathy of unclear etiology to distinguish between a compressive lesion of the optic nerve, optic neuritis, ischemic optic neuropathy (arteritic or non-arteritic), central retinal vein occlusion or optic nerve infiltrative disorders~~
	  ○ ~~Bilateral optic disk swelling (papilledema) with vision loss~~
	~~Added:~~
	• ~~Visual loss (as a neurological deficit) Not explained by underlying ocular diagnosis, glaucoma or macular degeneration~~
	• ~~Under New acute headache, sudden onset:~~
	  ○ ~~With a personal or family history of brain aneurysm or AVM (arteriovenous malformation)~~
	  ○ ~~Known coagulopathy or on anticoagulation~~
	• ~~Under New onset of headache and any of the following~~
	  ○ ~~Fever~~
	  ○ ~~Subacute head trauma~~
	  ○ ~~Pregnancy or puerperium~~
	  ○ ~~Age ≥ 50~~
	  ○ ~~Neurological deficits – Note: Neuroimaging warranted for atypical/complex migraine aura, but not for a typical migraine aura (see background)~~
	~~Added:~~
	• ~~Special additional considerations in the pediatric population with persistent headache~~
	  ○ ~~Symptoms indicative of intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting~~
	  ○ ~~Severe headache in a child with an underlying disease that predisposes to intracranial pathology (e.g.; immune deficiency, sickle cell disease neurofibromatosis, history of neoplasm, coagulopathy, hypertension, congenital heart disease)~~

	• ~~Suspected stroke with a personal or family history (brother, sister, parent or child) of aneurysm or known coagulopathy/anticoagulation~~
	
	~~Added:~~
	• ~~Suspected Pituitary Tumors:~~
	  ○ ~~With the following:~~
	  ▪ ~~Neurologic findings (e.g. visual field deficit suggesting compression of the optic chiasm)~~
	  ▪ ~~Suspected hypofunctioning pituitary gland based on hormonal testing e.g., hypo pituitarism, growth hormone deficiency, hypogonadotropic hypogonadism [i.e. low gonadotropins (FSH/LH) and sex hormones*]~~
	  ▪ ~~* severe secondary hypogonadism with total testosterone persistently < 150 and low or normal LH/FSH OR~~
	  ▪ ~~* testosterone levels below normal range with low or normal LH/FSH AND~~
	  • ~~neurological sign and symptoms OR~~
	  • ~~other pituitary hormonal abnormalities OR~~
	  • ~~consideration of reversible functional causes of gonadotropin suppression (e.g. obesity, opioid use, or comorbid illness)~~
	~~Added:~~
	• ~~Suspected hyperfunctioning pituitary gland based on hormonal testing i.e., central hyperthyroidism (high TSH), Cushing disease (high ACTH), acromegaly/gigantism (high GH/IGF-1) or elevated prolactin (>250 ng/mL or persistently elevated in the absence of another cause eg. stress, pregnancy, hypothyroidism, medication)~~
	• ~~Note: Galactorrhea without elevated prolactin, imaging is not indicated~~
	• ~~Central Diabetes Insipidus (low ADH)~~
	• ~~Precocious puberty in a child (male < 9; female < 8), with hormonal studies suggesting a central cause and evidence of an accelerated bone age on X-ray~~
	• ~~Pituitary apoplexy with sudden onset of neurological and hormonal symptoms~~
	• ~~Suspected recurrence with prior history of CNS cancer based on neurological symptoms or examination~~
	~~Added:~~
	• ~~Follow up of known meningioma~~
	  ○ ~~If <2cm or heavily calcified at 2 years and 5 years~~

	○ > 2cm annually for 3 years and then scans at 5 years and 10 years. ○ Multiple meningiomas, annually ○ After treatment (surgery or radiotherapy), post-operative if concern for residual tumor, every 6-12 months then annually for 3-5 years based on WHO Grade (see background) • Follow-up known of pituitary adenoma ○ New signs or symptoms ○ Functioning adenoma - to assess response to treatment and 1-year follow-up after drug holiday Added: • Follow of known pineal cyst (≥ 5mm) if there are atypical features or symptoms (e.g., headaches, gaze paresis, ataxia, papilledema, nausea/vomiting) • Follow of known arachnoid cyst ○ < 4 years old, serial imaging is warranted ○ > 4 years old, repeat imaging is approvable if newly symptomatic i.e. headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction • For screening for known Non-CNS Cancer ○ Default screening for ▪ Kidney cancer ▪ Lung cancer ▪ Merkel cell carcinoma Added: • Mucosal melanoma of the head and neck, especially of the oral cavity • Poorly differential neuroendocrine cancer (Large or Small cell/Unknown primary of neuroendocrine origin) • Screening with preconditions ○ AML…….Suspicion of leukemic meningitis ○ Cutaneous melanoma….Stage IIIC or higher ○ Testicular Cancer-Seminoma….. High risk ○ Gestational Trophoblastic Neoplasia…Pulmonary metastasis ○ Bladder cancer……High risk, i.e. small cell • All other cancer if CNS symptoms present Added: • For screening of Hereditary Cancer Syndromes ○ Li Fraumeni syndrome- Annually ○ Von Hippel Lindau – Every 2 years, starting at age of 8 years ○ Tuberous Sclerosis – Every 1-3 years, until the age of 25 years

	○ MEN1 – Every 3-5 years, starting at the age of 5 years ○ NF-2- Brain IAC: Annually starting, from age of 10 years ○ Sturge-Weber Syndrome: Once, after age 1 to rule out intracranial involvement after; in patients <1 year, only if symptomatic • Known seizure disorder without previous imaging Added: • Imaging indications for new-onset seizures in the pediatric population ○ Abnormal neurological exam, especially a postictal focal deficit ○ Significant developmental delay ○ Focal onset ○ EEG shows focal or suspected structural abnormalities ○ <1 year of age Note: Imaging is not indicated in simple febrile seizures • Suspected temporal arteritis in a patient > 50 with temporal headache, abrupt visual changes, jaw claudication, temporal artery tenderness, constitutional symptoms or elevated ESR AND ○ Negative initial work-up (color Doppler ultrasonography or biopsy) OR ○ Atypical features or failure to response to treatment with concern for large vessel involvement Added: • MRI indicted for atypical dystonia. Note: MRI not indicated in essential tremor or isolated focal dystonia (e.g., blepharospasm, cervical dystonia, laryngeal dystonia, oromandibular dystonia, writer's dystonia) • Binocular diplopia with concern for intracranial pathology • Hemifacial spasm • Other objective cranial nerve palsy (CN IX-XII) • Follow-up shunt evaluation (Pople, 2002, Reddy, 2014, Kamenova, 2018) ○ Post operatively if indicated based on underlying disease and pre-operative radiographic findings and/or ○ 6-12 months after placement and/or ○ With neurologic symptoms that suggest shunt malfunction Added: • Suspected spontaneous intra-cranial hypotension with distinct postural headache other symptoms include: nausea, vomiting, dizziness, tinnitus, diplopia neck pain or imbalance • CSF flow study for evaluation and management of CSF flow disorders • Diagnosis of central sleep apnea on polysomnogram ○ Children > 1 year

	○ Adults in the absence of heart failure, chronic opioid use, high altitude, or treatment emergent central sleep apnea AND concern for a central neurological cause (Chiari malformation, tumor, infectious/inflammatory disease) OR with an abnormal neurological exam • Syncope with clinical concern for seizure or associated neurological signs or symptoms • Cyclical vomiting syndrome or abdominal migraine with any localizing neurological symptoms • Soft tissue mass of the head with nondiagnostic initial evaluation (ultrasound and/or radiograph) Added: • Cerebral palsy if etiology has not been established the neonatal period, there is change in the expected clinical or developmental profile or concern for progressive neurological disorder • Unexplained event (BRUE) formerly apparent life-threatening event (ALTE) in infants < 1 year with concern for neurological cause based on history and exam Note: Imaging is not indicated in low risk patients • Under Indications for a Brain MRI with Internal Auditory Canal (IAC): ○ CSF otorrhea (MRI for intermittent leak, CT for active leaks) ○ Clinical Suspicion of acute mastoiditis as a complication of acute otitis media with intracranial complications (i.e. meningeal signs, cranial nerve deficits, focal neurological findings, altered mental status) ○ Bell's Palsy for evaluation of the extracranial nerve course - if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset Added: • Combo Brain MRI/MRA ○ Thunderclap headache with continued concern for underlying vascular abnormality after initial negative work-up ▪ Negative Brain CT; ▪ AND Negative Lumbar Puncture ▪ Acute, sudden onset of headache with personal history of a vascular abnormality or first-degree family history of aneurysm • Combo Brain MRI/Orbit MRI

	~~○ Optic Neuritis if atypical presentation, severe visual impairment or poor recovery following initial onset or treatment onset~~ ~~• Combo Brain MRI/Face/Sinus/Neck MRI~~ ~~○ Bells/hemifacial spasm that meets above criteria~~ ~~○ Objective cranial nerve palsy (CN IX XII) (for evaluation of the extracranial nerve course~~ ~~○ Granulomatosis with polyangiitis (Wegener's granulomatosis) disease~~ ~~Deleted:~~ ~~• Under New onset of headache and any of the following~~ ~~○ Temporal headache in person > 55, with sedimentation rate (ESR) > 55 with tenderness over the temporal artery.~~ ~~• Known or suspected pituitary tumor with corroborating physical exam (i.e., galactorrhea or acromegaly) neurologic findings and/or lab abnormalities.~~ ~~• Known brain tumor and new onset of headache.~~ ~~• Follow up shunt evaluation within six (6) months of placement or one (1) year follow up and/or with neurologic symptoms~~ ~~• From combo Brain MRI/MRA Clinical suspicion of subarachnoid hemorrhage (SAH) ie thunderclap headache~~
~~August 2019~~	~~• For evaluation of patient with neurologic symptoms or deficits suspicious for MS: Added: "clinically isolated syndrome OR recurrent episodes of variable neurological signs or symptoms not attributable to another cause; And Removed time frame of 'within the last 4 weeks'~~ ~~• Removed: Stable condition with no prior imaging within the past ten (10) months or within the past six (6) months if patient has relapsing disease~~ ~~• Removed: Exacerbation of symptoms or change in symptom characteristics such as frequency or type and demonstrated compliance with medical therapy.~~ ~~• For evaluation of MS, added:~~ ~~○ To establish a new baseline (no recent imaging, postpartum, or 6-12 months after switching disease modifying therapy)~~ ~~○ Prior to starting or switching disease-modifying therapy~~ ~~○ Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years~~ ~~○ New signs or symptoms suggested of an exacerbation or unexpected clinical worsening~~

	○ PML surveillance for patients on natalizumab • For evaluation of known or suspected seizure disorder, added: ○ Newly identified change in seizure activity/pattern • Renamed Parkinson's section to: Movement disorders and added: ○ For the evaluation of other movement disorder to exclude a structural lesion (i.e., suspected Huntington disease, chorea, atypical parkinsonian syndromes, hemiballismus, secondary dystonia). ○ * MRI not indicated in essential tremor or primary dystonia ○ For suspected Parkinson's, added 'with atypical feature or unresponsive to levodopa • For evaluation of neurologic symptoms or deficits, added: visual loss • For trauma, added: ○ On anticoagulation ○ Post concussive syndrome if persistent or disabling symptoms and imaging has not been performed ○ Subacute or chronic traumatic brain injury with new cognitive and/or neurologic deficit • For evaluation of headache, added or removed: ○ Prior history of stroke or intracranial bleed with sudden onset of severe headache (moved) ○ New headache and signs of increased intracranial pressure ○ Related to activity or event (sexual activity, exertion, position) (new or progressively worsening) ○ New headache and persistent or progressively worsening during a course of physician directed treatment ○ Special considerations in the pediatric population with persistent headache: ▪ Occipital location ▪ Age < 6 years ▪ No family history of headache • For evaluation of brain tumor: ○ Specified 'malignant' for f/u of known tumor ○ Added: Follow up of known benign tumor if symptomatic, new/changing signs or symptoms or complicating factors; Follow up of known meningioma; and tumor evaluation and monitoring in neurocutaneous syndromes

	~~○ Removed: Known lung cancer or rule out metastasis and/or preoperative evaluation, Metastatic melanoma (not all melanomas)~~ ~~• For evaluation of suspected stroke:~~ ~~○ Moved 'patient with history of a known stroke with new and sudden onset of severe headache'~~ ~~○ Separated: Family history of aneurysm~~ ~~• For evaluation inflammatory disease or infections:~~ ~~○ Changed meningitis with positive signs and symptoms from 'And' positive lab findings to 'OR' positive labs~~ ~~○ For suspected encephalitis removed 'severe' headache~~ ~~• For evaluation of congenital abnormality:~~ ~~○ Modified the age restriction of > 6 months age for eval of macrocephaly to include 'in an infant/child with previously abnormal US, abnormal neurodevelopmental exam, signs of increased ICP or closed anterior fontanelle'~~ ~~• For known or suspected normal pressure hydrocephalus (NPH):~~ ~~○ Added - With symptoms of gait difficulty, cognitive disturbance and urinary incontinence~~ ~~• Other Indications:~~ ~~○ Added detail to Vertigo including:~~ ~~▪ Signs or symptoms suggestive of a CNS lesion (ataxia, visual loss, double vision, weakness or a change in sensation)~~ ~~▪ Progressive unilateral hearing loss~~ ~~▪ Risk factors for cerebrovascular disease~~ ~~▪ After full neurologic examination and ENT work-up with concern for central vertigo~~ ~~○ Modified developmental delay to include: Global developmental delay or developmental delay with abnormal neurological examination~~ ~~○ Added:~~ ~~▪ Horner's syndrome with symptoms localizing the lesion to the central nervous system~~ ~~▪ Trigeminal Neuralgia – if <40 years of age or atypical features (ie bilateral, hearing loss, dizziness/vertigo, visual changes, sensory loss, numbness, pain >2min, pain outside trigeminal nerve distribution, progression)~~

	~~▪ Bell's Palsy- if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset.~~ ~~▪ Psychological changes with neurological deficits on exam or after completion of a full neurological assessment that suggests a possible neurologic cause~~ ~~▪ New-onset anisocoria~~ ~~○ Removed Objective cranial nerve palsy; and Cholesteatoma (duplicated)~~ ~~• For Brain MRI/Neck MRA: deleted 'confirmed carotid occlusion > 60%, surgery or angioplasty candidate' and added 'Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits'~~ ~~• Added Brain MRI/Brain MRA section, including: Clinical suspicion of subarachnoid hemorrhage (SAH) ie thunderclap headache; and Suspected venous thrombosis (dural sinus thrombosis)~~ ~~• Added Brain MRI/Brain MRA/Neck MRA section, including: Recent stroke or transient ischemic attack (TIA); and Approved indications as noted above and being performed in a child under 8 years of age who will need anesthesia for the procedure and there is a suspicion of concurrent vascular and intracranial pathology~~ ~~• For Brain MRI/Cervical MRI, added: Suspected MS with new or changing symptoms consistent with cervical spinal cord disease; and Follow up to the initiation or change in medication for patient with known Multiple Sclerosis~~ ~~• For Brain MRI/Orbit MRI, added: Bilateral papilledema with visual loss; and Known or suspected neuromyelitis optica spectrum disorder with severe, recurrent or bilateral optic neuritis; AND changed age restriction from 3 years to 8 years for children requiring anesthesia for the procedure with suspicion of concurrent orbital and intracranial pathology or tumor~~ ~~• Added section for Brain MRI/Face/Sinus/Neck MRI, including: Anosmia on objective testing; and Trigeminal neuralgia or cranial nerve palsy that meets the above criteria~~ ~~• Updated background information and references~~

REFERENCES

1. American College of Radiology. ACR Appropriateness Criteria® Headache. American College of Radiology. Updated 2022. Accessed January 23, 2023. https://acsearch.acr.org/docs/69482/Narrative/
2. Holle D, Obermann M. The role of neuroimaging in the diagnosis of headache disorders. *Ther Adv Neurol Disord*. Nov 2013;6(6):369-74. doi:10.1177/1756285613489765
3. Quinones-Hinojosa A, Gulati M, Singh V, Lawton MT. Spontaneous intracerebral hemorrhage due to coagulation disorders. *Neurosurg Focus*. Oct 15 2003;15(4):E3. doi:10.3171/foc.2003.15.4.3
4. Schaefer PW, Miller JC, Singhal AB, Thrall JH, Lee SI. Headache: when is neurologic imaging indicated? *J Am Coll Radiol*. Aug 2007;4(8):566-9. doi:10.1016/j.jacr.2006.10.001
5. Wilbrink LA, Ferrari MD, Kruit MC, Haan J. Neuroimaging in trigeminal autonomic cephalgias: when, how, and of what? *Curr Opin Neurol*. Jun 2009;22(3):247-53. doi:10.1097/wco.0b013e32832b4bb3
6. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia*. Jan 2018;38(1):1-211. doi:10.1177/0333102417738202
7. Micieli A, Kingston W. An Approach to Identifying Headache Patients That Require Neuroimaging. *Front Public Health*. 2019;7:52. doi:10.3389/fpubh.2019.00052
8. Mitsikostas DD, Ashina M, Craven A, et al. European Headache Federation consensus on technical investigation for primary headache disorders. *J Headache Pain*. 2015;17:5. doi:10.1186/s10194-016-0596-y
9. Hamilton K. Secondary Headaches During Pregnancy and the Postpartum Period. BMC. Updated May 2020. Accessed January 23, 2023. https://practicalneurology.com/articles/2020-may/secondary-headaches-during-pregnancy-and-the-postpartum-period
10. Shobeiri E, Torabinejad B. Brain magnetic resonance imaging findings in postpartum headache. *Neuroradiol J*. Feb 2019;32(1):4-9. doi:10.1177/1971400918804193
11. Nahas SJ. New Guidelines on Headache Imaging. NEJM Journal Watch. Updated January 8, 2020. Accessed January 23, 2023. https://www.jwatch.org/na50541/2020/01/08/new-guidelines-headache-imaging
12. D.W. D. Clinical clues and clinical rules: Primary vs secondary headache. *Advanced Studies in Medicine*. 2003;3(6C):S550-S555.
13. Togha M, Karimitafti MJ, Ghorbani Z, et al. Characteristics and comorbidities of headache in patients over 50 years of age: a cross-sectional study. *BMC Geriatrics*. 2022/04/10 2022;22(1):313. doi:10.1186/s12877-022-03027-1
14. Cordenier A, De Hertogh W, De Keyser J, Versijpt J. Headache associated with cough: a review. *J Headache Pain*. May 20 2013;14(1):42. doi:10.1186/1129-2377-14-42
15. Kuruvilla DE, Lipton RB. Appropriate use of neuroimaging in headache. *Curr Pain Headache Rep*. Jun 2015;19(6):17. doi:10.1007/s11916-015-0490-3
16. Martin VT. The diagnostic evaluation of secondary headache disorders. *Headache*. Feb 2011;51(2):346-52. doi:10.1111/j.1526-4610.2010.01841.x
17. Gofshteyn JS, Stephenson DJ. Diagnosis and Management of Childhood Headache. *Curr Probl Pediatr Adolesc Health Care*. Feb 2016;46(2):36-51. doi:10.1016/j.cppeds.2015.11.003

18. Dao JM, Qubty W. Headache Diagnosis in Children and Adolescents. *Curr Pain Headache Rep*. Feb 23 2018;22(3):17. doi:10.1007/s11916-018-0675-7
19. Trofimova A, Vey BL, Mullins ME, Wolf DS, Kadom N. Imaging of Children With Nontraumatic Headaches. *AJR Am J Roentgenol*. Jan 2018;210(1):8-17. doi:10.2214/ajr.17.18561
20. Wippold FJ, 2nd. Focal neurologic deficit. *AJNR Am J Neuroradiol*. Nov 2008;29(10):1998-2000.
21. American College of Radiology. ACR Appropriateness Criteria®Cerebrovascular Disease. American College of Radiology (ACR). Updated 2016. Accessed January 22, 2023. https://acsearch.acr.org/docs/69478/Narrative/
22. American College of Radiology. ACR Appropriateness Criteria®Cerebrovascular Disease-Child. American College of Radiology (ACR). Updated 2019. Accessed January 23, 2023. https://acsearch.acr.org/docs/3102253/Narrative/
23. Jauch EC, Saver JL, Adams HP, Jr., et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. Mar 2013;44(3):870-947. doi:10.1161/STR.0b013e318284056a
24. Akers A, Al-Shahi Salman R, I AA, et al. Synopsis of Guidelines for the Clinical Management of Cerebral Cavernous Malformations: Consensus Recommendations Based on Systematic Literature Review by the Angioma Alliance Scientific Advisory Board Clinical Experts Panel. *Neurosurgery*. May 1 2017;80(5):665-680. doi:10.1093/neuros/nyx091
25. Velz J, Stienen MN, Neidert MC, Yang Y, Regli L, Bozinov O. Routinely Performed Serial Follow-Up Imaging in Asymptomatic Patients With Multiple Cerebral Cavernous Malformations Has No Influence on Surgical Decision Making. *Front Neurol*. 2018;9:848. doi:10.3389/fneur.2018.00848
26. Zyck S, Gould GC. Cavernous Venous Malformation. StatPearls Publishing. Updated May 9, 2022. Accessed January 23, 2023. https://www.ncbi.nlm.nih.gov/books/NBK526009/
27. Bushnell C, Saposnik G. Evaluation and management of cerebral venous thrombosis. *Continuum (Minneap Minn)*. Apr 2014;20(2 Cerebrovascular Disease):335-51. doi:10.1212/01.CON.0000446105.67173.a8
28. DeBaun MR, Jordan LC, King AA, et al. American Society of Hematology 2020 guidelines for sickle cell disease: prevention, diagnosis, and treatment of cerebrovascular disease in children and adults. *Blood Adv*. Apr 28 2020;4(8):1554-1588. doi:10.1182/bloodadvances.2019001142
29. Mackin RS, Insel P, Truran D, et al. Neuroimaging abnormalities in adults with sickle cell anemia: associations with cognition. *Neurology*. Mar 11 2014;82(10):835-41. doi:10.1212/wnl.0000000000000188
30. Thust SC, Burke C, Siddiqui A. Neuroimaging findings in sickle cell disease. *Br J Radiol*. Aug 2014;87(1040):20130699. doi:10.1259/bjr.20130699
31. Abboud MR, Cure J, Granger S, et al. Magnetic resonance angiography in children with sickle cell disease and abnormal transcranial Doppler ultrasonography findings enrolled in the STOP study. *Blood*. Apr 1 2004;103(7):2822-6. doi:10.1182/blood-2003-06-1972
32. Sheehan VA, Hansbury EN, Smeltzer MP, Fortner G, McCarville MB, Aygun B. Transcranial Doppler velocity and brain MRI/MRA changes in children with sickle cell anemia on chronic transfusions to prevent primary stroke. *Pediatr Blood Cancer*. Sep 2013;60(9):1499-502. doi:10.1002/pbc.24569

33. American College of Radiology. ACR Appropriateness Criteria® Head Trauma. American College of Radiology (ACR). Updated 2020. Accessed January 23, 2023. https://acsearch.acr.org/docs/69481/Narrative/
34. Jagoda AS, Bazarian JJ, Bruns JJ, Jr., et al. Clinical policy: neuroimaging and decisionmaking in adult mild traumatic brain injury in the acute setting. *Ann Emerg Med*. Dec 2008;52(6):714-48. doi:10.1016/j.annemergmed.2008.08.021
35. Polinder S, Cnossen MC, Real RGL, et al. A Multidimensional Approach to Post-concussion Symptoms in Mild Traumatic Brain Injury. *Front Neurol*. 2018;9:1113. doi:10.3389/fneur.2018.01113
36. Panwar J, Hsu CC, Tator CH, Mikulis D. Magnetic Resonance Imaging Criteria for Post-Concussion Syndrome: A Study of 127 Post-Concussion Syndrome Patients. *J Neurotrauma*. May 15 2020;37(10):1190-1196. doi:10.1089/neu.2019.6809
37. Kernick DP, Ahmed F, Bahra A, et al. Imaging patients with suspected brain tumour: guidance for primary care. *Br J Gen Pract*. Dec 2008;58(557):880-5. doi:10.3399/bjgp08X376203
38. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Central Nervous System Cancers Version 2.2022. National Comprehensive Cancer Network (NCCN). Updated September 29, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/cns.pdf
39. American College of Radiology. ACR Appropriateness Criteria® Neuroendocrine Imaging. American College of Radiology. Updated 2018. Accessed January 23, 2023. https://acsearch.acr.org/docs/69485/Narrative/
40. Consensus guidelines for the diagnosis and treatment of growth hormone (GH) deficiency in childhood and adolescence: summary statement of the GH Research Society. GH Research Society. *J Clin Endocrinol Metab*. Nov 2000;85(11):3990-3. doi:10.1210/jcem.85.11.6984
41. Kannan S, Kennedy L. Diagnosis of acromegaly: state of the art. *Expert Opin Med Diagn*. Sep 2013;7(5):443-53. doi:10.1517/17530059.2013.820181
42. Majumdar A, Mangal NS. Hyperprolactinemia. *J Hum Reprod Sci*. Jul 2013;6(3):168-75. doi:10.4103/0974-1208.121400
43. Bhasin S, Brito JP, Cunningham GR, et al. Testosterone Therapy in Men With Hypogonadism: An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. May 1 2018;103(5):1715-1744. doi:10.1210/jc.2018-00229
44. Bonneville J-F, Potorac I, Petrossians P, Tshibanda L, Beckers A. Pituitary MRI in Cushing's disease - an update. *Journal of Neuroendocrinology*. 2022;34(8):e13123. doi:https://doi.org/10.1111/jne.13123
45. P. D, N.P. V. Dexamethasone Suppression Test. StatPearls Publishing Updated Aug 8 2022. Accessed April 7, 2023. https://www.ncbi.nlm.nih.gov/books/NBK542317/#_NBK542317_pubdet_
46. L.K. N. Recent Updates on the Diagnosis and Management of Cushing's Syndrome. *Endocrinol Metab*. 2018;33(2):139-146.
47. ARUP Consult aALtstfhp. Adrenal Hyperfunction (Cushing Syndrome) Testing Algorithm. ARUP Consult (https://arupconsult.com). Updated May 2021. Accessed April 7, 2023. https://arupconsult.com/algorithm/adrenal-hyperfunction-cushing-syndrome-testing-algorithm
48. Melmed S, Casanueva FF, Hoffman AR, et al. Diagnosis and treatment of hyperprolactinemia: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. Feb 2011;96(2):273-88. doi:10.1210/jc.2010-1692

49. Casanueva FF, Molitch ME, Schlechte JA, et al. Guidelines of the Pituitary Society for the diagnosis and management of prolactinomas. *Clin Endocrinol (Oxf)*. Aug 2006;65(2):265-73. doi:10.1111/j.1365-2265.2006.02562.x
50. Vilar L, Vilar CF, Lyra R, Freitas MDC. Pitfalls in the Diagnostic Evaluation of Hyperprolactinemia. *Neuroendocrinology*. 2019;109(1):7-19. doi:10.1159/000499694
51. Faizah M, Zuhanis A, Rahmah R, et al. Precocious puberty in children: A review of imaging findings. *Biomed Imaging Interv J*. Jan 2012;8(1):e6. doi:10.2349/biij.8.1.e6
52. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Kidney Cancer Version 4.2023. National Comprehensive Cancer Network (NCCN). Updated January 18, 2023. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/kidney.pdf
53. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Merkel Cell Carcinoma Version 2.2022. National Comprehensive Cancer Network (NCCN). Updated March 24, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/mcc.pdf
54. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Melanoma: Cutaneous Version 1.2023. National Comprehensive Cancer Network (NCCN). Updated December 22, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/cutaneous_melanoma.pdf
55. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Melanoma: Uveal Version 2.2022. National Comprehensive Cancer Network (NCCN). Updated April 5, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/uveal.pdf
56. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Non-Small Cell Lung Cancer Version 1.2023. National Comprehensive Cancer Network (NCCN). Updated December 22, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/nscl.pdf
57. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Neuroendocrine and Adrenal Tumors Version 2.2022. National Comprehensive Cancer Network (NCCN). Updated December 21, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/neuroendocrine.pdf
58. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Testicular Cancer Version 2.2022. National Comprehensive Cancer Network (NCCN). Updated January 4, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/testicular.pdf
59. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Bladder Cancer Version 3.2022. National Comprehensive Cancer Network (NCCN). Updated December 21, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/bladder.pdf
60. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Acute Myeloid Leukemia Version 3.2022. National Comprehensive Cancer Network (NCCN). Updated January 13, 2023. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/aml.pdf
61. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines): Gestational Trophoblastic Neoplasia Version 1.2023. National Comprehensive Cancer Network (NCCN). Updated December 20, 2022. Accessed January 23, 2023. https://www.nccn.org/professionals/physician_gls/pdf/gtn.pdf
62. Go RS, Jacobsen E, Baiocchi R, et al. Histiocytic Neoplasms, Version 2.2021, NCCN Clinical Practice Guidelines in Oncology. *J Natl Compr Canc Netw*. Nov 2021;19(11):1277-1303. doi:10.6004/jnccn.2021.0053

63. Goyal G, Young JR, Koster MJ, et al. The Mayo Clinic Histiocytosis Working Group Consensus Statement for the Diagnosis and Evaluation of Adult Patients With Histiocytic Neoplasms: Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease. *Mayo Clin Proc*. Oct 2019;94(10):2054-2071. doi:10.1016/j.mayocp.2019.02.023
64. Kumar P, Gill RM, Phelps A, Tulpule A, Matthay K, Nicolaides T. Surveillance Screening in Li-Fraumeni Syndrome: Raising Awareness of False Positives. *Cureus*. Apr 24 2018;10(4):e2527. doi:10.7759/cureus.2527
65. Rednam SP, Erez A, Druker H, et al. Von Hippel-Lindau and Hereditary Pheochromocytoma/Paraganglioma Syndromes: Clinical Features, Genetics, and Surveillance Recommendations in Childhood. *Clin Cancer Res*. Jun 15 2017;23(12):e68-e75. doi:10.1158/1078-0432.Ccr-17-0547
66. Krueger DA, Northrup H. Tuberous sclerosis complex surveillance and management: recommendations of the 2012 International Tuberous Sclerosis Complex Consensus Conference. *Pediatr Neurol*. Oct 2013;49(4):255-65. doi:10.1016/j.pediatrneurol.2013.08.002
67. Brandi ML, Gagel RF, Angeli A, et al. Guidelines for diagnosis and therapy of MEN type 1 and type 2. *J Clin Endocrinol Metab*. Dec 2001;86(12):5658-71. doi:10.1210/jcem.86.12.8070
68. Evans DGR, Salvador H, Chang VY, et al. Cancer and Central Nervous System Tumor Surveillance in Pediatric Neurofibromatosis 2 and Related Disorders. *Clin Cancer Res*. Jun 15 2017;23(12):e54-e61. doi:10.1158/1078-0432.Ccr-17-0590
69. Comi AM. Presentation, diagnosis, pathophysiology, and treatment of the neurological features of Sturge-Weber syndrome. *Neurologist*. Jul 2011;17(4):179-84. doi:10.1097/NRL.0b013e318220c5b6
70. Stoller JK, Nielsen C, Buccola J. Pituitary Tumor. *Cleveland Clinic Intensive Review of Internal Medicine*. 6th ed. Wolters Kluwer; 2015.
71. Eroukhmanoff J, Tejedor I, Potorac I, et al. MRI follow-up is unnecessary in patients with macroprolactinomas and long-term normal prolactin levels on dopamine agonist treatment. *Eur J Endocrinol*. Mar 2017;176(3):323-328. doi:10.1530/eje-16-0897
72. Kurosaki M, Kambe A, Watanabe T, Fujii S, Ogawa T. Serial 3 T magnetic resonance imaging during cabergoline treatment of macroprolactinomas. *Neurol Res*. Apr 2015;37(4):341-6. doi:10.1179/1743132814y.0000000457
73. Varlamov EV, Hinojosa-Amaya JM, Fleseriu M. Magnetic resonance imaging in the management of prolactinomas; a review of the evidence. *Pituitary*. Feb 2020;23(1):16-26. doi:10.1007/s11102-019-01001-6
74. Dekkers OM, Pereira AM, Romijn JA. Treatment and follow-up of clinically nonfunctioning pituitary macroadenomas. *J Clin Endocrinol Metab*. Oct 2008;93(10):3717-26. doi:10.1210/jc.2008-0643
75. Lake MG, Krook LS, Cruz SV. Pituitary adenomas: an overview. *Am Fam Physician*. Sep 1 2013;88(5):319-27.
76. Cauley KA, Linnell GJ, Braff SP, Filippi CG. Serial follow-up MRI of indeterminate cystic lesions of the pineal region: experience at a rural tertiary care referral center. *AJR Am J Roentgenol*. Aug 2009;193(2):533-7. doi:10.2214/ajr.08.1906
77. Jussila MP, Olsén P, Salokorpi N, Suo-Palosaari M. Follow-up of pineal cysts in children: is it necessary? *Neuroradiology*. Dec 2017;59(12):1265-1273. doi:10.1007/s00234-017-1926-8

78. Trifanescu R, Ansorge O, Wass JAH, Grossman AB, Karavitaki N. Rathke's cleft cysts. *Clinical Endocrinology*. 2012;76(2):151-160. doi:https://doi.org/10.1111/j.1365-2265.2011.04235.x
79. Petersson M, Berinder K, Eden Engström B, et al. Natural history and surgical outcome of Rathke's cleft cysts—A study from the Swedish Pituitary Registry. *Clinical Endocrinology*. 2022;96(1):54-61. doi:https://doi.org/10.1111/cen.14622
80. Al-Holou WN, Yew AY, Boomsaad ZE, Garton HJ, Muraszko KM, Maher CO. Prevalence and natural history of arachnoid cysts in children. *J Neurosurg Pediatr*. Jun 2010;5(6):578-85. doi:10.3171/2010.2.Peds09464
81. Al-Holou WN, Terman S, Kilburg C, Garton HJ, Muraszko KM, Maher CO. Prevalence and natural history of arachnoid cysts in adults. *J Neurosurg*. Feb 2013;118(2):222-31. doi:10.3171/2012.10.Jns12548
82. Jafrani R, Raskin JS, Kaufman A, Lam S. Intracranial arachnoid cysts: Pediatric neurosurgery update. *Surg Neurol Int*. 2019;10:15. doi:10.4103/sni.sni_320_18
83. Mustansir F, Bashir S, Darbar A. Management of Arachnoid Cysts: A Comprehensive Review. *Cureus*. Apr 10 2018;10(4):e2458. doi:10.7759/cureus.2458
84. Haupt R, Minkov M, Astigarraga I, et al. Langerhans cell histiocytosis (LCH): guidelines for diagnosis, clinical work-up, and treatment for patients till the age of 18 years. *Pediatr Blood Cancer*. Feb 2013;60(2):175-84. doi:10.1002/pbc.24367
85. American College of Radiology. ACR Appropriateness Criteria® Seizures and Epilepsy. American College of Radiology. Updated 2019. Accessed January 23, 2023. https://acsearch.acr.org/docs/69479/Narrative/
86. Cendes F, Theodore WH, Brinkmann BH, Sulc V, Cascino GD. Neuroimaging of epilepsy. *Handb Clin Neurol*. 2016;136:985-1014. doi:10.1016/b978-0-444-53486-6.00051-x
87. Gaillard WD, Chiron C, Cross JH, et al. Guidelines for imaging infants and children with recent-onset epilepsy. *Epilepsia*. Sep 2009;50(9):2147-53. doi:10.1111/j.1528-1167.2009.02075.x
88. Ho K, Lawn N, Bynevelt M, Lee J, Dunne J. Neuroimaging of first-ever seizure: Contribution of MRI if CT is normal. *Neurol Clin Pract*. Oct 2013;3(5):398-403. doi:10.1212/CPJ.0b013e3182a78f25
89. Krumholz A, Wiebe S, Gronseth G, et al. Practice Parameter: evaluating an apparent unprovoked first seizure in adults (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. *Neurology*. Nov 20 2007;69(21):1996-2007. doi:10.1212/01.wnl.0000285084.93652.43
90. Ramli N, Rahmat K, Lim KS, Tan CT. Neuroimaging in refractory epilepsy. Current practice and evolving trends. *Eur J Radiol*. Sep 2015;84(9):1791-800. doi:10.1016/j.ejrad.2015.03.024
91. Hourani R, Nasreddine W, Dirani M, et al. When Should a Brain MRI Be Performed in Children with New-Onset Seizures? Results of a Large Prospective Trial. *American Journal of Neuroradiology*. 2021;42(9):1695-1701. doi:10.3174/ajnr.A7193
92. Hirtz D, Ashwal S, Berg A, et al. Practice parameter: evaluating a first nonfebrile seizure in children: report of the quality standards subcommittee of the American Academy of Neurology, The Child Neurology Society, and The American Epilepsy Society. *Neurology*. Sep 12 2000;55(5):616-23. doi:10.1212/wnl.55.5.616
93. Bernasconi A, Cendes F, Theodore WH, et al. Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: A consensus report from the International

League Against Epilepsy Neuroimaging Task Force. *Epilepsia*. Jun 2019;60(6):1054-1068. doi:10.1111/epi.15612
94. Consortium of Multiple Sclerosis Centers. 2018 MRI Protocol and Clinical Guidelines for MS. Consortium of Multiple Sclerosis Centers (CMSC). Updated May 22, 2018. Accessed January 23, 2023. https://www.mscare.org/page/MRI_protocol
95. Thompson AJ, Banwell BL, Barkhof F, et al. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. *Lancet Neurol*. Feb 2018;17(2):162-173. doi:10.1016/s1474-4422(17)30470-2
96. Traboulsee A, Simon JH, Stone L, et al. Revised Recommendations of the Consortium of MS Centers Task Force for a Standardized MRI Protocol and Clinical Guidelines for the Diagnosis and Follow-Up of Multiple Sclerosis. *AJNR Am J Neuroradiol*. Mar 2016;37(3):394-401. doi:10.3174/ajnr.A4539
97. Wattjes MP, Ciccarelli O, Reich DS, et al. 2021 MAGNIMS-CMSC-NAIMS consensus recommendations on the use of MRI in patients with multiple sclerosis. *Lancet Neurol*. Aug 2021;20(8):653-670. doi:10.1016/s1474-4422(21)00095-8
98. Rae-Grant A, Day GS, Marrie RA, et al. Practice guideline recommendations summary: Disease-modifying therapies for adults with multiple sclerosis: Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology. *Neurology*. Apr 24 2018;90(17):777-788. doi:10.1212/wnl.0000000000005347
99. Amato MP, De Stefano N, Inglese M, et al. Secondary Prevention in Radiologically Isolated Syndromes and Prodromal Stages of Multiple Sclerosis. Review. *Frontiers in Neurology*. 2022-March-14 2022;13doi:10.3389/fneur.2022.787160
100. McGuigan C, Craner M, Guadagno J, et al. Stratification and monitoring of natalizumab-associated progressive multifocal leukoencephalopathy risk: recommendations from an expert group. *J Neurol Neurosurg Psychiatry*. Feb 2016;87(2):117-25. doi:10.1136/jnnp-2015-311100
101. Lummel N, Koch M, Klein M, Pfister HW, Brückmann H, Linn J. Spectrum and Prevalence of Pathological Intracranial Magnetic Resonance Imaging Findings in Acute Bacterial Meningitis. *Clin Neuroradiol*. Jun 2016;26(2):159-67. doi:10.1007/s00062-014-0339-x
102. Oliveira CR, Morriss MC, Mistrot JG, Cantey JB, Doern CD, Sánchez PJ. Brain magnetic resonance imaging of infants with bacterial meningitis. *J Pediatr*. Jul 2014;165(1):134-9. doi:10.1016/j.jpeds.2014.02.061
103. Diamantopoulos AP, Haugeberg G, Hetland H, Soldal DM, Bie R, Myklebust G. Diagnostic value of color Doppler ultrasonography of temporal arteries and large vessels in giant cell arteritis: a consecutive case series. *Arthritis Care Res (Hoboken)*. Jan 2014;66(1):113-9. doi:10.1002/acr.22178
104. D'Souza NM, Morgan ML, Almarzouqi SJ, Lee AG. Magnetic resonance imaging findings in giant cell arteritis. *Eye (Lond)*. May 2016;30(5):758-62. doi:10.1038/eye.2016.19
105. Klink T, Geiger J, Both M, et al. Giant cell arteritis: diagnostic accuracy of MR imaging of superficial cranial arteries in initial diagnosis-results from a multicenter trial. *Radiology*. Dec 2014;273(3):844-52. doi:10.1148/radiol.14140056
106. Salehi-Abari I. 2016 ACR revised criteria for early diagnosis of giant cell (temporal) arteritis. *Autoimmune Dis Ther Approaches Open Access*. 2016;3:1-4.
107. Yip A, Jernberg ET, Bardi M, et al. Magnetic resonance imaging compared to ultrasonography in giant cell arteritis: a cross-sectional study. *Arthritis Res Ther*. Oct 19 2020;22(1):247. doi:10.1186/s13075-020-02335-4

108. Zuccoli G, Pipitone N, Haldipur A, Brown RD, Jr., Hunder G, Salvarani C. Imaging findings in primary central nervous system vasculitis. *Clin Exp Rheumatol*. Jan-Feb 2011;29(1 Suppl 64):S104-9.
109. Godasi R, Pang G, Chauhan S, Bollu PC. Primary Central Nervous System Vasculitis. StatPearls Publishing. Updated October 12, 2022. Accessed January 23, 2023. https://www.ncbi.nlm.nih.gov/books/NBK482476/
110. Voortman M, Drent M, Baughman RP. Management of neurosarcoidosis: a clinical challenge. *Curr Opin Neurol*. Jun 2019;32(3):475-483. doi:10.1097/wco.0000000000000684
111. Fritz D, van de Beek D, Brouwer MC. Clinical features, treatment and outcome in neurosarcoidosis: systematic review and meta-analysis. *BMC Neurol*. Nov 15 2016;16(1):220. doi:10.1186/s12883-016-0741-x
112. Shah R, Roberson GH, Curé JK. Correlation of MR imaging findings and clinical manifestations in neurosarcoidosis. *AJNR Am J Neuroradiol*. May 2009;30(5):953-61. doi:10.3174/ajnr.A1470
113. Harvey PD. Clinical applications of neuropsychological assessment. *Dialogues Clin Neurosci*. Mar 2012;14(1):91-9. doi:10.31887/DCNS.2012.14.1/pharvey
114. Health Quality Ontario. The appropriate use of neuroimaging in the diagnostic work-up of dementia: an evidence-based analysis. *Ont Health Technol Assess Ser*. 2014;14(1):1-64.
115. Narayanan L, Murray AD. What can imaging tell us about cognitive impairment and dementia? *World J Radiol*. Mar 28 2016;8(3):240-54. doi:10.4329/wjr.v8.i3.240
116. Carpenter CR, Bassett ER, Fischer GM, Shirshekan J, Galvin JE, Morris JC. Four sensitive screening tools to detect cognitive dysfunction in geriatric emergency department patients: brief Alzheimer's Screen, Short Blessed Test, Ottawa 3DY, and the caregiver-completed AD8. *Acad Emerg Med*. Apr 2011;18(4):374-84. doi:10.1111/j.1553-2712.2011.01040.x
117. McDougall GJ. A review of screening instruments for assessing cognition and mental status in older adults. *Nurse Pract*. Nov 1990;15(11):18-28.
118. U.S. Food & Drug Administration. Reference ID: 4807032 Full Prescribing Information ADUHELM(tm). U.S. Food & Drug Administration. Updated June 2021. Accessed January 23, 2023. https://www.accessdata.fda.gov/drugsatfda_docs/label/2021/761178s000lbl.pdf
119. Inc. B. Aduhelm™ [prescribing information] Biogen Inc. 2023. Updated February 2023. 2023. https://www.biogencdn.com/us/aduhelm-pi.pdf
120. American College of Radiology. ACR Appropriateness Criteria® Movement Disorders and Neurodegenerative Diseases. American College of Radiology. Updated 2019. Accessed January 23, 2023. https://acsearch.acr.org/docs/3111293/Narrative/
121. Albanese A, Asmus F, Bhatia KP, et al. EFNS guidelines on diagnosis and treatment of primary dystonias. *Eur J Neurol*. Jan 2011;18(1):5-18. doi:10.1111/j.1468-1331.2010.03042.x
122. Mascalchi M, Vella A, Ceravolo R. Movement disorders: role of imaging in diagnosis. *J Magn Reson Imaging*. Feb 2012;35(2):239-56. doi:10.1002/jmri.22825
123. McFarland NR. Diagnostic Approach to Atypical Parkinsonian Syndromes. *Continuum (Minneap Minn)*. Aug 2016;22(4 Movement Disorders):1117-42. doi:10.1212/con.0000000000000348
124. Pyatigorskaya N, Gallea C, Garcia-Lorenzo D, Vidailhet M, Lehericy S. A review of the use of magnetic resonance imaging in Parkinson's disease. *Ther Adv Neurol Disord*. Jul 2014;7(4):206-20. doi:10.1177/1756285613511507

125. Sharifi S, Nederveen AJ, Booij J, van Rootselaar AF. Neuroimaging essentials in essential tremor: a systematic review. *Neuroimage Clin*. 2014;5:217-31. doi:10.1016/j.nicl.2014.05.003
126. Comella CL, National Organization for Rare Disorders. Cervical Dystonia. National Organization for Rare Disorders (NORD). Updated 2019. Accessed January 23, 2023. https://rarediseases.org/rare-diseases/cervical-dystonia/
127. Chang VA, Meyer DM, Meyer BC. Isolated Anisocoria as a Presenting Stroke Code Symptom is Unlikely to Result in Alteplase Administration. *J Stroke Cerebrovasc Dis*. Jan 2019;28(1):163-166. doi:10.1016/j.jstrokecerebrovasdis.2018.09.029
128. Iliescu DA, Timaru CM, Alexe N, et al. Management of diplopia. *Rom J Ophthalmol*. Jul-Sep 2017;61(3):166-170. doi:10.22336/rjo.2017.31
129. American Association for Pediatric Ophthalmology and Strabismus. Five things physicians and patients should question: Don't routinely order neuro-imaging for all patients with double vision. Choosing Wisely Initiative ABIM Foundation. Updated May 29, 2019. Accessed January 23, 2023. https://www.choosingwisely.org/clinician-lists/american-association-pediatric-ophthalmology-strabismus-imaging-for-double-vision/
130. Kadom N. Pediatric strabismus imaging. *Curr Opin Ophthalmol*. Sep 2008;19(5):371-8. doi:10.1097/ICU.0b013e328309f165
131. Yoon L, Kim HY, Kwak MJ, et al. Utility of Magnetic Resonance Imaging (MRI) in Children With Strabismus. *J Child Neurol*. Sep 2019;34(10):574-581. doi:10.1177/0883073819846807
132. Lee JH, Lee HK, Lee DH, Choi CG, Kim SJ, Suh DC. Neuroimaging strategies for three types of Horner syndrome with emphasis on anatomic location. *AJR Am J Roentgenol*. Jan 2007;188(1):W74-81. doi:10.2214/ajr.05.1588
133. Bendtsen L, Zakrzewska JM, Abbott J, et al. European Academy of Neurology guideline on trigeminal neuralgia. *Eur J Neurol*. Jun 2019;26(6):831-849. doi:10.1111/ene.13950
134. Cruccu G, Finnerup NB, Jensen TS, et al. Trigeminal neuralgia: New classification and diagnostic grading for practice and research. *Neurology*. Jul 12 2016;87(2):220-8. doi:10.1212/wnl.0000000000002840
135. Garza I. Craniocervical junction schwannoma mimicking occipital neuralgia. *Headache*. Sep 2007;47(8):1204-5. doi:10.1111/j.1526-4610.2007.00887.x
136. Choi I, Jeon SR. Neuralgias of the Head: Occipital Neuralgia. *J Korean Med Sci*. Apr 2016;31(4):479-88. doi:10.3346/jkms.2016.31.4.479
137. Vanelderen P, Lataster A, Levy R, Mekhail N, Van Kleef M, Van Zundert J. 8. Occipital Neuralgia. *Pain Practice*. 2010;10(2):137-144. doi:https://doi.org/10.1111/j.1533-2500.2009.00355.x
138. Quesnel AM, Lindsay RW, Hadlock TA. When the bell tolls on Bell's palsy: finding occult malignancy in acute-onset facial paralysis. *Am J Otolaryngol*. Sep-Oct 2010;31(5):339-42. doi:10.1016/j.amjoto.2009.04.003
139. Hermier M. Imaging of hemifacial spasm. *Neurochirurgie*. May 2018;64(2):117-123. doi:10.1016/j.neuchi.2018.01.005
140. American College of Radiology. ACR Appropriateness Criteria® Cranial Neuropathy. American College of Radiology (ACR). Updated 2022. Accessed January 22, 2023. https://acsearch.acr.org/docs/69509/Narrative/

141. Mumtaz S, Jensen MB. Facial neuropathy with imaging enhancement of the facial nerve: a case report. *Future Neurol*. Nov 1 2014;9(6):571-576. doi:10.2217/fnl.14.55
142. Yedavalli VS, Patil A, Shah P. Amyotrophic Lateral Sclerosis and its Mimics/Variants: A Comprehensive Review. *J Clin Imaging Sci*. 2018;8:53. doi:10.4103/jcis.JCIS_40_18
143. King RR, Reiss JP. The epidemiology and pathophysiology of pseudobulbar affect and its association with neurodegeneration. *Degener Neurol Neuromuscul Dis*. 2013;3:23-31. doi:10.2147/dnnd.S34160
144. Ashwal S, Michelson D, Plawner L, Dobyns WB. Practice parameter: Evaluation of the child with microcephaly (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*. Sep 15 2009;73(11):887-97. doi:10.1212/WNL.0b013e3181b783f7
145. Vinocur DN, Medina LS. Imaging in the evaluation of children with suspected craniosynostosis. *Evidence-based imaging in pediatrics*. Springer; 2010:43-52.
146. Tan AP, Mankad K, Gonçalves FG, Talenti G, Alexia E. Macrocephaly: Solving the Diagnostic Dilemma. *Top Magn Reson Imaging*. Aug 2018;27(4):197-217. doi:10.1097/rmr.0000000000000170
147. Dougherty H, Shaunak M, Irving M, Thompson D, Cheung MS. Identification of Characteristic Neurological Complications in Infants with Achondroplasia by Routine MRI Screening. *ESPE Abstracts*. 2018;89
148. Kubota T, Adachi M, Kitaoka T, et al. Clinical Practice Guidelines for Achondroplasia. *Clin Pediatr Endocrinol*. 2020;29(1):25-42. doi:10.1297/cpe.29.25
149. Ashwal S, Russman BS, Blasco PA, et al. Practice parameter: diagnostic assessment of the child with cerebral palsy: report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology*. Mar 23 2004;62(6):851-63. doi:10.1212/01.wnl.0000117981.35364.1b
150. Cerebral palsy in under 25s: assessment and management National Institute for Health and Care Excellence (NICE). Updated January 25, 2017. Accessed January 23, 2023. https://www.nice.org.uk/guidance/ng62/resources/cerebral-palsy-in-under-25s-assessment-and-management-1837570402501
151. Mallack EJ, Turk BR, Yan H, et al. MRI surveillance of boys with X-linked adrenoleukodystrophy identified by newborn screening: Meta-analysis and consensus guidelines. *J Inherit Metab Dis*. May 2021;44(3):728-739. doi:10.1002/jimd.12356
152. Whitson WJ, Lane JR, Bauer DF, Durham SR. A prospective natural history study of nonoperatively managed Chiari I malformation: does follow-up MRI surveillance alter surgical decision making? *J Neurosurg Pediatr*. Aug 2015;16(2):159-66. doi:10.3171/2014.12.Peds14301
153. Damasceno BP. Neuroimaging in normal pressure hydrocephalus. *Dement Neuropsychol*. Oct-Dec 2015;9(4):350-355. doi:10.1590/1980-57642015dn94000350
154. Kamenova M, Rychen J, Guzman R, Mariani L, Soleman J. Yield of early postoperative computed tomography after frontal ventriculoperitoneal shunt placement. *PLoS One*. 2018;13(6):e0198752. doi:10.1371/journal.pone.0198752
155. Pople IK. Hydrocephalus and shunts: what the neurologist should know. *J Neurol Neurosurg Psychiatry*. Sep 2002;73 Suppl 1(Suppl 1):i17-22. doi:10.1136/jnnp.73.suppl_1.i17

156. Reddy GK, Bollam P, Caldito G. Long-term outcomes of ventriculoperitoneal shunt surgery in patients with hydrocephalus. *World Neurosurg*. Feb 2014;81(2):404-10. doi:10.1016/j.wneu.2013.01.096
157. Wetzel JS, Heaner DP, Gabel BC, Tubbs RS, Chern JJ. Clinical evaluation and surveillance imaging of children with myelomeningocele and shunted hydrocephalus: a follow-up study. *J Neurosurg Pediatr*. Oct 19 2018;23(2):153-158. doi:10.3171/2018.7.Peds1826
158. Severson M, Strecker-McGraw MK. Cerebrospinal Fluid Leak. StatPearls Publishing. Updated August 8, 2022. Accessed January 23, 2023. https://www.ncbi.nlm.nih.gov/books/NBK538157/
159. Mantur M, Łukaszewicz-Zając M, Mroczko B, et al. Cerebrospinal fluid leakage--reliable diagnostic methods. *Clin Chim Acta*. May 12 2011;412(11-12):837-40. doi:10.1016/j.cca.2011.02.017
160. Selcuk H, Albayram S, Ozer H, et al. Intrathecal gadolinium-enhanced MR cisternography in the evaluation of CSF leakage. *AJNR Am J Neuroradiol*. Jan 2010;31(1):71-5. doi:10.3174/ajnr.A1788
161. Gordon N. Spontaneous intracranial hypotension. *Dev Med Child Neurol*. Dec 2009;51(12):932-5. doi:10.1111/j.1469-8749.2009.03514.x
162. Deline C, Schievink WI, National Organization for Rare Disorders. Spontaneous Intracranial Hypotension. National Organization for Rare Disorders (NORD). Updated September 1, 2020. Accessed January 23, 2023. https://rarediseases.org/rare-diseases/spontaneous-intracranial-hypotension/
163. Bradley WG, Jr. Magnetic Resonance Imaging of Normal Pressure Hydrocephalus. *Semin Ultrasound CT MR*. Apr 2016;37(2):120-8. doi:10.1053/j.sult.2016.01.005
164. Mohammad SA, Osman NM, Ahmed KA. The value of CSF flow studies in the management of CSF disorders in children: a pictorial review. *Insights Imaging*. Jan 28 2019;10(1):3. doi:10.1186/s13244-019-0686-x
165. National Organization for Rare Disorders. Chiari Malformations. National Organization for Rare Disorders (NORD). Updated 2014. Accessed January 23, 2023. https://rarediseases.org/rare-diseases/chiari-malformations/
166. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke*. Nov 2009;40(11):3504-10. doi:10.1161/strokeaha.109.551234
167. Welgampola MS, Young AS, Pogson JM, Bradshaw AP, Halmagyi GM. Dizziness demystified. *Pract Neurol*. Dec 2019;19(6):492-501. doi:10.1136/practneurol-2019-002199
168. Yamada S, Yasui K, Kawakami Y, Hasegawa Y, Katsuno M. DEFENSIVE Stroke Scale: Novel Diagnostic Tool for Predicting Posterior Circulation Infarction in the Emergency Department. *J Stroke Cerebrovasc Dis*. Jun 2019;28(6):1561-1570. doi:10.1016/j.jstrokecerebrovasdis.2019.03.005
169. Felix O, Amaddeo A, Olmo Arroyo J, et al. Central sleep apnea in children: experience at a single center. *Sleep Med*. Sep 2016;25:24-28. doi:10.1016/j.sleep.2016.07.016
170. Malhotra A, Owens RL. What is central sleep apnea? *Respir Care*. Sep 2010;55(9):1168-78.
171. Al-Nsoor NM, Mhearat AS. Brain computed tomography in patients with syncope. *Neurosciences (Riyadh)*. Apr 2010;15(2):105-9.
172. Strickberger SA, Benson DW, Biaggioni I, et al. AHA/ACCF Scientific Statement on the evaluation of syncope: from the American Heart Association Councils on Clinical Cardiology, Cardiovascular Nursing, Cardiovascular Disease in the Young, and Stroke, and the Quality of Care and Outcomes Research Interdisciplinary Working Group; and the American College of Cardiology Foundation: in

collaboration with the Heart Rhythm Society: endorsed by the American Autonomic Society. *Circulation*. Jan 17 2006;113(2):316-27. doi:10.1161/circulationaha.105.170274
173. Venkatesan T, Levinthal DJ, Tarbell SE, et al. Guidelines on management of cyclic vomiting syndrome in adults by the American Neurogastroenterology and Motility Society and the Cyclic Vomiting Syndrome Association. *Neurogastroenterol Motil*. Jun 2019;31 Suppl 2(Suppl 2):e13604. doi:10.1111/nmo.13604
174. Li BUK. Managing cyclic vomiting syndrome in children: beyond the guidelines. *Eur J Pediatr*. Oct 2018;177(10):1435-1442. doi:10.1007/s00431-018-3218-7
175. Angus-Leppan H, Saatci D, Sutcliffe A, Guiloff RJ. Abdominal migraine. *Bmj*. Feb 19 2018;360:k179. doi:10.1136/bmj.k179
176. American College of Radiology. ACR Appropriateness Criteria® Soft-Tissue Masses. American College of Radiology. Updated 2022. Accessed January 23, 2023. https://acsearch.acr.org/docs/69434/Narrative/
177. Kim HS, An JK, Woo JJ, Yoon RG. Superficially Palpable Masses of the Scalp and Face: A Pictorial Essay. *Journal of the Korean Society of Radiology*. 2019;80(2):283-293.
178. Zhang J, Li Y, Zhao Y, Qiao J. CT and MRI of superficial solid tumors. *Quant Imaging Med Surg*. Mar 2018;8(2):232-251. doi:10.21037/qims.2018.03.03
179. American College of Radiology. ACR Appropriateness Criteria® Acute Mental Status Change, Delirium, and New Onset Psychosis American College of Radiology. Updated 2018. Accessed January 23, 2023. https://acsearch.acr.org/docs/3102409/Narrative/
180. Ali AS, Syed NP, Murthy GS, et al. Magnetic resonance imaging (MRI) evaluation of developmental delay in pediatric patients. *J Clin Diagn Res*. Jan 2015;9(1):Tc21-4. doi:10.7860/jcdr/2015/11921.5478
181. Momen AA, Jelodar G, Dehdashti H. Brain magnetic resonance imaging findings in developmentally delayed children. *Int J Pediatr*. 2011;2011:386984. doi:10.1155/2011/386984
182. Tieder JS, Bonkowsky JL, Etzel RA, et al. Brief Resolved Unexplained Events (Formerly Apparent Life-Threatening Events) and Evaluation of Lower-Risk Infants: Executive Summary. *Pediatrics*. May 2016;137(5)doi:10.1542/peds.2016-0591
183. Gerull S, Medinger M, Heim D, Passweg J, Stern M. Evaluation of the Pretransplantation Workup before Allogeneic Transplantation. *Biology of Blood and Marrow Transplantation*. 2014/11/01/ 2014;20(11):1852-1856. doi:https://doi.org/10.1016/j.bbmt.2014.06.029
184. Kaste SC, Kaufman RA, Sunkara A, et al. Routine pre- and post-hematopoietic stem cell transplant computed tomography of the abdomen for detecting invasive fungal infection has limited value. *Biol Blood Marrow Transplant*. Jun 2015;21(6):1132-5. doi:10.1016/j.bbmt.2015.02.023
185. Joshi VM, Navlekar SK, Kishore GR, Reddy KJ, Kumar EC. CT and MR imaging of the inner ear and brain in children with congenital sensorineural hearing loss. *Radiographics*. May-Jun 2012;32(3):683-98. doi:10.1148/rg.323115073
186. Dewan K, Wippold FJ, 2nd, Lieu JE. Enlarged vestibular aqueduct in pediatric sensorineural hearing loss. *Otolaryngol Head Neck Surg*. Apr 2009;140(4):552-8. doi:10.1016/j.otohns.2008.12.035
187. Ralli M, Rolesi R, Anzivino R, Turchetta R, Fetoni AR. Acquired sensorineural hearing loss in children: current research and therapeutic perspectives. *Acta Otorhinolaryngol Ital*. Dec 2017;37(6):500-508. Sordità infantile acquisita: stato dell'arte della ricerca e prospettive terapeutiche. doi:10.14639/0392-100x-1574

188. Hiremath SB, Gautam AA, Sasindran V, Therakathu J, Benjamin G. Cerebrospinal fluid rhinorrhea and otorrhea: A multimodality imaging approach. *Diagn Interv Imaging*. Jan 2019;100(1):3-15. doi:10.1016/j.diii.2018.05.003
189. Patel KM, Almutairi A, Mafee MF. Acute otomastoiditis and its complications: Role of imaging. *Operative Techniques in Otolaryngology-Head and Neck Surgery*. 2014/03/01 2014;25(1):21-28. doi:https://doi.org/10.1016/j.otot.2013.11.004
190. Platzek I, Kitzler HH, Gudziol V, Laniado M, Hahn G. Magnetic resonance imaging in acute mastoiditis. *Acta Radiol Short Rep*. Feb 2014;3(2):2047981614523415. doi:10.1177/2047981614523415
191. Radiology ACo. ACR Appropriateness Criteria® ACR-ASNR-SPR Practice Parameter for the Performance of Intracranial Magnetic Resonance Perfusion Imaging. 2022. Updated 2022. Accessed April 26, 2023. https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Perfusion.pdf
192. Suh CH, Kim HS, Jung SC, Choi CG, Kim SJ. Perfusion MRI as a diagnostic biomarker for differentiating glioma from brain metastasis: a systematic review and meta-analysis. *Eur Radiol*. Sep 2018;28(9):3819-3831. doi:10.1007/s00330-018-5335-0
193. Arevalo-Perez J, Peck KK, Young RJ, Holodny AI, Karimi S, Lyo JK. Dynamic Contrast-Enhanced Perfusion MRI and Diffusion-Weighted Imaging in Grading of Gliomas. *J Neuroimaging*. Sep-Oct 2015;25(5):792-8. doi:10.1111/jon.12239
194. Metaweh NAK, Azab AO, El Basmy AAH, Mashhour KN, El Mahdy WM. Contrast-Enhanced Perfusion MR Imaging to Differentiate Between Recurrent/Residual Brain Neoplasms and Radiation Necrosis. *Asian Pac J Cancer Prev*. Apr 25 2018;19(4):941-948. doi:10.22034/apjcp.2018.19.4.941
195. Wang L, Wei L, Wang J, et al. Evaluation of perfusion MRI value for tumor progression assessment after glioma radiotherapy: A systematic review and meta-analysis. *Medicine (Baltimore)*. Dec 24 2020;99(52):e23766. doi:10.1097/md.0000000000023766
196. Lawson GR. Controversy: Sedation of children for magnetic resonance imaging. *Arch Dis Child*. Feb 2000;82(2):150-3. doi:10.1136/adc.82.2.150
197. Whitehead MT, Cardenas AM, Corey AS, et al. ACR Appropriateness Criteria® Headache. *J Am Coll Radiol*. Nov 2019;16(11s):S364-s377. doi:10.1016/j.jacr.2019.05.030
198. Yeh YC, Fuh JL, Chen SP, Wang SJ. Clinical features, imaging findings and outcomes of headache associated with sexual activity. *Cephalalgia*. Nov 2010;30(11):1329-35. doi:10.1177/0333102410364675
199. Yuan MK, Lai PH, Chen JY, et al. Detection of subarachnoid hemorrhage at acute and subacute/chronic stages: comparison of four magnetic resonance imaging pulse sequences and computed tomography. *J Chin Med Assoc*. Mar 2005;68(3):131-7. doi:10.1016/s1726-4901(09)70234-5
200. Chen CY, Fuh JL. Evaluating thunderclap headache. *Curr Opin Neurol*. Jun 1 2021;34(3):356-362. doi:10.1097/wco.0000000000000917
201. Pegge SAH, Steens SCA, Kunst HPM, Meijer FJA. Pulsatile Tinnitus: Differential Diagnosis and Radiological Work-Up. *Curr Radiol Rep*. 2017;5(1):5. doi:10.1007/s40134-017-0199-7
202. Yew KS. Diagnostic approach to patients with tinnitus. *Am Fam Physician*. Jan 15 2014;89(2):106-13.

203. Wingerchuk DM, Banwell B, Bennett JL, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurology*. Jul 14 2015;85(2):177-89. doi:10.1212/wnl.0000000000001729
204. Kaunzner UW, Gauthier SA. MRI in the assessment and monitoring of multiple sclerosis: an update on best practice. *Ther Adv Neurol Disord*. Jun 2017;10(6):247-261. doi:10.1177/1756285617708911
205. Radic JAE, Cochrane DD. Choosing Wisely Canada: Pediatric Neurosurgery Recommendations. *Paediatr Child Health*. Sep 2018;23(6):383-387. doi:10.1093/pch/pxy012
206. Shah LM, Salzman KL. Imaging of spinal metastatic disease. *Int J Surg Oncol*. 2011;2011:769753. doi:10.1155/2011/769753
207. Behbehani R. Clinical approach to optic neuropathies. *Clin Ophthalmol*. Sep 2007;1(3):233-46.
208. Margolin E. The swollen optic nerve: an approach to diagnosis and management. *Pract Neurol*. Aug 2019;19(4):302-309. doi:10.1136/practneurol-2018-002057
209. Kaur K, Gurnani B, Devy N. Atypical optic neuritis - a case with a new surprise every visit. *GMS Ophthalmol Cases*. 2020;10:Doc11. doi:10.3205/oc000138
210. Phuljhele S, Kedar S, Saxena R. Approach to optic neuritis: An update. *Indian J Ophthalmol*. Sep 2021;69(9):2266-2276. doi:10.4103/ijo.IJO_3415_20
211. de Graaf P, Göricke S, Rodjan F, et al. Guidelines for imaging retinoblastoma: imaging principles and MRI standardization. *Pediatr Radiol*. Jan 2012;42(1):2-14. doi:10.1007/s00247-011-2201-5
212. Razek AA, Elkhamary S. MRI of retinoblastoma. *Br J Radiol*. Sep 2011;84(1005):775-84. doi:10.1259/bjr/32022497
213. Pakalniskis MG, Berg AD, Policeni BA, et al. The Many Faces of Granulomatosis With Polyangiitis: A Review of the Head and Neck Imaging Manifestations. *AJR Am J Roentgenol*. Dec 2015;205(6):W619-29. doi:10.2214/ajr.14.13864
214. Hughes MA, Frederickson AM, Branstetter BF, Zhu X, Sekula RF, Jr. MRI of the Trigeminal Nerve in Patients With Trigeminal Neuralgia Secondary to Vascular Compression. *AJR Am J Roentgenol*. Mar 2016;206(3):595-600. doi:10.2214/ajr.14.14156
215. Jang YE, Cho EY, Choi HY, Kim SM, Park HY. Diagnostic Neuroimaging in Headache Patients: A Systematic Review and Meta-Analysis. *Psychiatry Investig*. Jun 2019;16(6):407-417. doi:10.30773/pi.2019.04.11
216. Spierings EL. Acute, subacute, and chronic headache. *Otolaryngol Clin North Am*. Dec 2003;36(6):1095-107, vi. doi:10.1016/s0030-6665(03)00128-2
217. Tyagi A. New daily persistent headache. *Ann Indian Acad Neurol*. Aug 2012;15(Suppl 1):S62-5. doi:10.4103/0972-2327.100011
218. Hadjikhani N, Vincent M. Neuroimaging clues of migraine aura. *J Headache Pain*. Apr 3 2019;20(1):32. doi:10.1186/s10194-019-0983-2
219. Chhetri SK, Gow D, Shaunak S, Varma A. Clinical assessment of the sensory ataxias; diagnostic algorithm with illustrative cases. *Pract Neurol*. Aug 2014;14(4):242-51. doi:10.1136/practneurol-2013-000764
220. Foster H, Drummond P, Jandial S, Clinch J, Wood M, Driscoll S. Evaluation of gait disorders in children. BMJ Best Practice. Updated February 23, 2021. Accessed January 23, 2023. https://bestpractice.bmj.com/topics/en-us/709

221. Standford Medicine. Gait Abnormalities. Stanford University. Accessed January 23, 2023. https://stanfordmedicine25.stanford.edu/the25/gait.html
222. Haynes KB, Wimberly RL, VanPelt JM, Jo CH, Riccio AI, Delgado MR. Toe Walking: A Neurological Perspective After Referral From Pediatric Orthopaedic Surgeons. *J Pediatr Orthop*. Mar 2018;38(3):152-156. doi:10.1097/bpo.0000000000001115
223. Marshall FJ. Approach to the elderly patient with gait disturbance. *Neurol Clin Pract*. Jun 2012;2(2):103-111. doi:10.1212/CPJ.0b013e31825a7823
224. Pirker W, Katzenschlager R. Gait disorders in adults and the elderly : A clinical guide. *Wien Klin Wochenschr*. Feb 2017;129(3-4):81-95. doi:10.1007/s00508-016-1096-4
225. Sacco RL, Kasner SE, Broderick JP, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. Jul 2013;44(7):2064-89. doi:10.1161/STR.0b013e318296aeca
226. Kernan WN, Ovbiagele B, Black HR, et al. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. Jul 2014;45(7):2160-236. doi:10.1161/str.0000000000000024
227. Easton JD, Saver JL, Albers GW, et al. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. *Stroke*. Jun 2009;40(6):2276-93. doi:10.1161/strokeaha.108.192218
228. Hong KS, Yegiaian S, Lee M, Lee J, Saver JL. Declining stroke and vascular event recurrence rates in secondary prevention trials over the past 50 years and consequences for current trial design. *Circulation*. May 17 2011;123(19):2111-9. doi:10.1161/circulationaha.109.934786
229. Wintermark M, Sanelli PC, Albers GW, et al. Imaging recommendations for acute stroke and transient ischemic attack patients: A joint statement by the American Society of Neuroradiology, the American College of Radiology, and the Society of NeuroInterventional Surgery. *AJNR Am J Neuroradiol*. Nov-Dec 2013;34(11):E117-27. doi:10.3174/ajnr.A3690
230. Robertson RL, Palasis S, Rivkin MJ, et al. ACR Appropriateness Criteria® Cerebrovascular Disease-Child. *J Am Coll Radiol*. May 2020;17(5s):S36-s54. doi:10.1016/j.jacr.2020.01.036
231. Salmela MB, Mortazavi S, Jagadeesan BD, et al. ACR Appropriateness Criteria(®) Cerebrovascular Disease. *J Am Coll Radiol*. May 2017;14(5s):S34-s61. doi:10.1016/j.jacr.2017.01.051
232. Lee M, Kim MS. Image findings in brain developmental venous anomalies. *J Cerebrovasc Endovasc Neurosurg*. Mar 2012;14(1):37-43. doi:10.7461/jcen.2012.14.1.37
233. Jensen RH, Radojicic A, Yri H. The diagnosis and management of idiopathic intracranial hypertension and the associated headache. *Ther Adv Neurol Disord*. Jul 2016;9(4):317-26. doi:10.1177/1756285616635987
234. Connors JM, Levy JH. Thromboinflammation and the hypercoagulability of COVID-19. *J Thromb Haemost*. Jul 2020;18(7):1559-1561. doi:10.1111/jth.14849

235. Tu TM, Goh C, Tan YK, et al. Cerebral Venous Thrombosis in Patients with COVID-19 Infection: a Case Series and Systematic Review. *J Stroke Cerebrovasc Dis*. Dec 2020;29(12):105379. doi:10.1016/j.jstrokecerebrovasdis.2020.105379
236. Coutinho JM. Cerebral venous thrombosis. *J Thromb Haemost*. Jun 2015;13 Suppl 1:S238-44. doi:10.1111/jth.12945
237. Ferro JM, Canhão P, Aguiar de Sousa D. Cerebral venous thrombosis. *Presse Med*. Dec 2016;45(12 Pt 2):e429-e450. doi:10.1016/j.lpm.2016.10.007
238. Atluri S, Sarathi V, Goel A, Boppana R, Shivaprasad C. Etiological Profile of Galactorrhoea. *Indian J Endocrinol Metab*. Jul-Aug 2018;22(4):489-493. doi:10.4103/ijem.IJEM_89_18
239. Huang W, Molitch ME. Evaluation and management of galactorrhea. *Am Fam Physician*. Jun 1 2012;85(11):1073-80.
240. Melmed S, Casanueva FF, Hoffman AR, et al. Diagnosis and Treatment of Hyperprolactinemia: An Endocrine Society Clinical Practice Guideline. *The Journal of Clinical Endocrinology & Metabolism*. 2011;96(2):273-288. doi:10.1210/jc.2010-1692
241. Kamihara J, Bourdeaut F, Foulkes WD, et al. Retinoblastoma and Neuroblastoma Predisposition and Surveillance. *Clin Cancer Res*. Jul 1 2017;23(13):e98-e106. doi:10.1158/1078-0432.Ccr-17-0652
242. Grasparil AD, Gottumukkala RV, Greer M-LC, Gee MS. Whole-Body MRI Surveillance of Cancer Predisposition Syndromes: Current Best Practice Guidelines for Use, Performance, and Interpretation. *American Journal of Roentgenology*. 2020/10/01 2020;215(4):1002-1011. doi:10.2214/AJR.19.22399
243. Borofsky S, Levy LM. Neurofibromatosis: types 1 and 2. *AJNR Am J Neuroradiol*. Dec 2013;34(12):2250-1. doi:10.3174/ajnr.A3534
244. Rovira À, Wattjes MP, Tintoré M, et al. Evidence-based guidelines: MAGNIMS consensus guidelines on the use of MRI in multiple sclerosis-clinical implementation in the diagnostic process. *Nat Rev Neurol*. Aug 2015;11(8):471-82. doi:10.1038/nrneurol.2015.106
245. Saguil A, Kane S, Farnell E. Multiple sclerosis: a primary care perspective. *Am Fam Physician*. Nov 1 2014;90(9):644-52.
246. Larivière D, Sacre K, Klein I, et al. Extra- and intracranial cerebral vasculitis in giant cell arteritis: an observational study. *Medicine (Baltimore)*. Dec 2014;93(28):e265. doi:10.1097/md.0000000000000265
247. Geyer M, Nilssen E. Evidence-based management of a patient with anosmia. *Clin Otolaryngol*. Oct 2008;33(5):466-9. doi:10.1111/j.1749-4486.2008.01819.x
248. Lechien JR, Chiesa-Estomba CM, De Siati DR, et al. Olfactory and gustatory dysfunctions as a clinical presentation of mild-to-moderate forms of the coronavirus disease (COVID-19): a multicenter European study. *Eur Arch Otorhinolaryngol*. Aug 2020;277(8):2251-2261. doi:10.1007/s00405-020-05965-1
249. Saniasiaya J, Islam MA, Abdullah B. Prevalence of Olfactory Dysfunction in Coronavirus Disease 2019 (COVID-19): A Meta-analysis of 27,492 Patients. *Laryngoscope*. Apr 2021;131(4):865-878. doi:10.1002/lary.29286
250. Smith R, Leonidas JC, Maytal J. The value of head ultrasound in infants with macrocephaly. *Pediatr Radiol*. Mar 1998;28(3):143-6. doi:10.1007/s002470050315
251. Pindrik J, Ye X, Ji BG, Pendleton C, Ahn ES. Anterior fontanelle closure and size in full-term children based on head computed tomography. *Clin Pediatr (Phila)*. Oct 2014;53(12):1149-57. doi:10.1177/0009922814538492

252. Cooper AS, Friedlaender E, Levy SE, et al. The Implications of Brain MRI in Autism Spectrum Disorder. *J Child Neurol*. Dec 2016;31(14):1611-1616. doi:10.1177/0883073816665548
253. Andersen BM, Miranda C, Hatzoglou V, DeAngelis LM, Miller AM. Leptomeningeal metastases in glioma: The Memorial Sloan Kettering Cancer Center experience. *Neurology*. May 21 2019;92(21):e2483-e2491. doi:10.1212/wnl.0000000000007529
254. Clarke JL, Perez HR, Jacks LM, Panageas KS, Deangelis LM. Leptomeningeal metastases in the MRI era. *Neurology*. May 4 2010;74(18):1449-54. doi:10.1212/WNL.0b013e3181dc1a69
255. Maillie L, Salgado LR, Lazarev S. A systematic review of craniospinal irradiation for leptomeningeal disease: past, present, and future. *Clin Transl Oncol*. Oct 2021;23(10):2109-2119. doi:10.1007/s12094-021-02615-8
256. Wang N, Bertalan MS, Brastianos PK. Leptomeningeal metastasis from systemic cancer: Review and update on management. *Cancer*. Jan 1 2018;124(1):21-35. doi:10.1002/cncr.30911
257. Ahmed A. MRI features of disseminated 'drop metastases'. *S Afr Med J*. Jul 2008;98(7):522-3.

ADDITIONAL RESOURCES

~~1. Abuabara A. Cerebrospinal fluid rhinorrhoea: diagnosis and management. *Med Oral Patol Oral Cir Bucal*. Sep 1 2007;12(5):E397-400.~~
~~3. American College of Radiology. Ten things physicians and patients should question: Don't do imaging for uncomplicated headache. Choosing Wisely Initiative ABIM Foundation. Updated June 29, 2017. Accessed October 24, 2022. http://www.choosingwisely.org/clinician-lists/american-college-radiology-imaging-for-uncomplicated-headache/ 5. Bley TA, Wieben O, Uhl M, Thiel J, Schmidt D, Langer M. High-resolution MRI in giant cell arteritis: imaging of the wall of the superficial temporal artery. *AJR Am J Roentgenol*. Jan 2005;184(1):283-7. doi:10.2214/ajr.184.1.01840283~~
~~6. Casanueva FF, Molitch ME, Schlechte JA, et al. Guidelines of the Pituitary Society for the diagnosis and management of prolactinomas. *Clin Endocrinol (Oxf)*. Aug 2006;65(2):265-73. doi:10.1111/j.1365-2265.2006.02562.x~~
~~7. Chase M, Joyce NR, Carney E, et al. ED patients with vertigo: can we identify clinical factors associated with acute stroke? *Am J Emerg Med*. May 2012;30(4):587-91. doi:10.1016/j.ajem.2011.02.002~~
~~8. Doty RL. Olfactory dysfunction and its measurement in the clinic. *World J Otorhinolaryngol Head Neck Surg*. Sep 2015;1(1):28-33. doi:10.1016/j.wjorl.2015.09.007~~
~~9. Freda PU, Beckers AM, Katznelson L, et al. Pituitary incidentaloma: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab*. Apr 2011;96(4):894-904. doi:10.1210/jc.2010-1048~~
~~10. Gofshteyn JS, Stephenson DJ. Diagnosis and Management of Childhood Headache. *Curr Probl Pediatr Adolesc Health Care*. Feb 2016;46(2):36-51. doi:10.1016/j.cppeds.2015.11.003~~
~~12. Dao JM, Qubty W. Headache Diagnosis in Children and Adolescents. *Curr Pain Headache Rep*. Feb 23 2018;22(3):17. doi:10.1007/s11916-018-0675-713. Kaplowitz PB. Do 6-8 year old girls with central precocious puberty need routine brain imaging? *Int J Pediatr Endocrinol*. 2016;2016:9. doi:10.1186/s13633-016-0027-5~~
1~~4~~. Radmanesh A, Raz E, Zan E, Derman A, Kaminetzky M. Brain Imaging Use and Findings in COVID-19: A Single Academic Center Experience in the Epicenter of Disease in the United States. *AJNR Am J Neuroradiol*. Jul 2020;41(7):1179-1183. doi:10.3174/ajnr.A6610

2. Naelitz B, Shah A, Nowacki AS, et al. Prolactin-to-Testosterone Ratio Predicts Pituitary Abnormalities in Mildly Hyperprolactinemic Men with Symptoms of Hypogonadism. *J Urol*. 2021;205(3):871-878. doi:10.1097/JU.0000000000001431

15. Shambhu S, Suarez L. Giant Cell Arteritis: An Atypical Presentation Diagnosed with the Use of MRI Imaging. *Case Rep Rheumatol*. 2016;2016:8239549. doi:10.1155/2016/8239549
16. Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*. Sep 26 2000;55(6):754-62. doi:10.1212/wnl.55.6.754
17. Smith R, Leonidas JC, Maytal J. The value of head ultrasound in infants with macrocephaly. *Pediatr Radiol*. Mar 1998;28(3):143-6. doi:10.1007/s002470050315
18. Wallace AN, McConathy J, Menias CO, Bhalla S, Wippold FJ, 2nd. Imaging evaluation of CSF shunts. *AJR Am J Roentgenol*. Jan 2014;202(1):38-53. doi:10.2214/ajr.12.10270
1. Abuabara A. Cerebrospinal fluid rhinorrhoea: diagnosis and management. *Med Oral Patol Oral Cir Bucal*. Sep 1 2007;12(5):E397-400.
2. Aduhelm™ [prescribing information]. Biogen. Updated July 2021. Accessed November 3, 2021. https://www.biogencdn.com/us/aduhelm-pi.pdf
3. American College of Radiology. Ten things physicians and patients should question: Don't do imaging for uncomplicated headache. Choosing Wisely Initiative ABIM Foundation. Updated June 29, 2017. Accessed November 3, 2021. http://www.choosingwisely.org/clinician-lists/american-college-radiology-imaging-for-uncomplicated-headache/
4. Beck RW, Cleary PA, Anderson MM, Jr., et al. A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. The Optic Neuritis Study Group. *N Engl J Med*. Feb 27 1992;326(9):581-8. doi:10.1056/nejm199202273260901
5. Bhasin S, Brito JP, Cunningham GR, et al. Testosterone Therapy in Men With Hypogonadism: An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. May 1 2018;103(5):1715-1744. doi:10.1210/jc.2018-00229
6. Bley TA, Wieben O, Uhl M, Thiel J, Schmidt D, Langer M. High-resolution MRI in giant cell arteritis: imaging of the wall of the superficial temporal artery. *AJR Am J Roentgenol*. Jan 2005;184(1):283-7. doi:10.2214/ajr.184.1.01840283
7. Casanueva FF, Molitch ME, Schlechte JA, et al. Guidelines of the Pituitary Society for the diagnosis and management of prolactinomas. *Clin Endocrinol (Oxf)*. Aug 2006;65(2):265-73. doi:10.1111/j.1365-2265.2006.02562.x
8. Chase M, Joyce NR, Carney E, et al. ED patients with vertigo: can we identify clinical factors associated with acute stroke? *Am J Emerg Med*. May 2012;30(4):587-91. doi:10.1016/j.ajem.2011.02.002
9. Dao JM, Qubty W. Headache Diagnosis in Children and Adolescents. *Curr Pain Headache Rep*. Feb 23 2018;22(3):17. doi:10.1007/s11916-018-0675-7
10. Doty RL. Olfactory dysfunction and its measurement in the clinic. *World J Otorhinolaryngol Head Neck Surg*. Sep 2015;1(1):28-33. doi:10.1016/j.wjorl.2015.09.007
11. Freda PU, Beckers AM, Katznelson L, et al. Pituitary incidentaloma: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab*. Apr 2011;96(4):894-904. doi:10.1210/jc.2010-1048
12. Gofshteyn JS, Stephenson DJ. Diagnosis and Management of Childhood Headache. *Curr Probl Pediatr Adolesc Health Care*. Feb 2016;46(2):36-51. doi:10.1016/j.cppeds.2015.11.003
13. Islim AI, Mohan M, Moon RDC, et al. Incidental intracranial meningiomas: a systematic review and meta-analysis of prognostic factors and outcomes. *J Neurooncol*. Apr 2019;142(2):211-221.

doi:10.1007/s11060-019-03104-3
14. Jafrani R, Raskin JS, Kaufman A, Lam S. Intracranial arachnoid cysts: Pediatric neurosurgery update. *Surg Neurol Int*. 2019;10:15. doi:10.4103/sni.sni_320_18
15. Kaplowitz PB. Do 6-8 year old girls with central precocious puberty need routine brain imaging? *Int J Pediatr Endocrinol*. 2016;2016:9. doi:10.1186/s13633-016-0027-5
16. National Health Services. Protocol for follow-up scanning in patient with a cranial meningioma v1 - Coversheet for Cancer Alliance Expert Advisory Group Agreed Documentation. National Health Services (NHS). Updated April 20, 2018. Accessed November 2, 2021. https://www.england.nhs.uk/mids-east/wp-content/uploads/sites/7/2018/05/protocol-for-follow-up-scanning-for-patient-with-meningioma.pdf
17. Radmanesh A, Raz E, Zan E, Derman A, Kaminetzky M. Brain Imaging Use and Findings in COVID-19: A Single Academic Center Experience in the Epicenter of Disease in the United States. *AJNR Am J Neuroradiol*. Jul 2020;41(7):1179-1183. doi:10.3174/ajnr.A6610
18. Shambhu S, Suarez L. Giant Cell Arteritis: An Atypical Presentation Diagnosed with the Use of MRI Imaging. *Case Rep Rheumatol*. 2016;2016:8239549. doi:10.1155/2016/8239549
19. Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*. Sep 26 2000;55(6):754-62. doi:10.1212/wnl.55.6.754
20. Wallace AN, McConathy J, Menias CO, Bhalla S, Wippold FJ, 2nd. Imaging evaluation of CSF shunts. *AJR Am J Roentgenol*. Jan 2014;202(1):38-53. doi:10.2214/ajr.12.10270

POLICY HISTORY

Date	Summary
May 2023	Updated and reformatted references Updated background section Added: • Indeterminate imaging section • Follow up of known Rathke cleft cyst ○ If no symptoms, MRI at 1/3/5 years to stability ○ With new neurological symptoms or atypical imaging features ○ Post treatment, yearly for 5 years • General Information moved to beginning of guideline with added statement on clinical indications not addressed in this guideline • Added statement regarding further evaluation of indeterminate findings on prior imaging Clarified: • Abnormal reflexes (*pathological, asymmetric, hyperreflexia)* • New onset headache - Related to activity or event (sexual activity, *exertion, Valsalva*, position), new or progressively worsening • Post concussive syndrome if persistent or disabling symptoms and *MRI* has not been performed • *Screening* for silent cerebral infarcts in early school age children and adults with *HbSS sickle cell disease or HbSβ0 thalassemia*

	• Cushing syndrome *suspected (high ACTH (>5) with cortisol suppression on low or high dose dexamethasone suppression test)* • Elevated prolactin after evaluation for another cause - neuroendocrine signs or symptoms (i.e., headache, galactorrhea, abnormal menses, infertility, or bitemporal hemianopsia) and/or *abnormal pituitary hormones (low testosterone /estrogen/ progesterone AND low or normal LH/FSH)* • Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; AND Low free testosterone and *consideration and addressment* of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use, or comorbid illness) • Tumor surveillance *as per professional society recommendations* • Note: In the pediatric population, imaging is not indicated in simple febrile seizures *or in idiopathic focal or generalized epilepsy with typical features [BECTS, childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), and juvenile myoclonic epilepsy (JME)]* • 6-month repeat scan in patients with MRI disease activity that is not associated with *new clinical symptoms on a routine follow-up scan (i.e., Radiographically isolated syndrome)* • Indications for MR Perfusion Imaging section • Brain MRI/Brain MRA - Headache associated with exercise, *exertion, Valsalva* or sexual activity Deleted: • Pediatric seizure indications and combined with adult • Anosmia (loss of smell) or dysosmia documented by objective testing that is persistent and of unknown origin (also in combo section)
May 2022	Updated and reformatted references Updated background section Combo statements added Reorganized indications Changed visual deficits section added to background Reorganized suspected tumor section Clarified: • Acute headache, sudden onset • New onset headache related to activity or event (sexual activity, exertion, position), new or progressively worsening • Visual loss in background/removed note • Low flow vascular malformations

	• Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) for screening and/or with neurological signs or symptoms • Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; ○ *Low free testosterone* and consideration of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use or comorbid illness) • Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) as per NCCN • Tumor monitoring in neurocutaneous syndromes as per tumor type • Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) To assess treatment response and surveillance of known brain lesions • To demonstrate dissemination in time for diagnosis (every 6-12 months) • To establish a new baseline (3-6 months after switching disease modifying therapy) • PML surveillance - Every 3-4 months, if high risk of PML occurrence; Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics • Examples of mental status instruments to screen for cognitive impairment • For evaluation of new non-Parkinson neurological symptoms • Binocular diplopia with concern for intracranial pathology after comprehensive eye evaluation • Trigeminal neuralgia or *neuropathy*, notably with an atypical presentation • **MRI Brain/MRI Orbit Combo** – Optic Neuritis if atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery, or recurrence • **MRI Brain/MRI Face/Sinus/Neck Combo**- Trigeminal neuralgia or neuropathy with an atypical presentation (for evaluation of the extracranial nerve course) Added: • Abnormal reflexes to neurologic deficit sections • 1-time screening for silent cerebral infarcts in school age children and adults with sickle cell disease

	• High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200 • Midline dermoid cysts/sinuses with concern for intracranial extension • Elevated prolactin in the absence of other cause: > 100, persistently elevated or neuroendocrine signs or symptoms • Follow-up of known low grade tumor (WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma) ○ For surveillance as per NCCN ○ If symptomatic, new/changing signs or symptoms or complicating factors • 6-month repeat scan in patients with MRI disease activity that is not associated with clinical activity on a follow-up scan (MS) • Note about pediatric MS imaging – same as adults except Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management • Neurosarcoidosis ○ Initial Evaluation: ▪ Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) OR ▪ Known history of sarcoidosis with neurological signs or symptoms ○ Follow up of known neurosarcoidosis: ▪ To assess treatment response ▪ Worsening signs or symptoms • Tourette syndrome to list of movement disorders in which MRI is not indicated • Occipital Neuralgia • X-linked Adrenoleukodystrophy ○ Baseline MRI between 12 and 18 months old ○ Second MRI 1 year after baseline ○ MRI every 6 months between 3 and 12 years old ○ Annual MRI after 12 years old • Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner. • Pulsatile tinnitus to combo section (MRI Brain with IAC/MRA Head/MRA Neck) • **General Combo statement**

	Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging. • **Combo Brain MRI/MRA**: ○ Neurological signs or symptoms in sickle cell patients ○ High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200 • **Brain MRI with IAC/ Brain MRA/Neck MRA (any combination)** ○ Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology ○ Note: MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA. • **MRI Brain/MRI Face/Sinus/Neck Combo-** ○ Bell's Palsy/hemifacial spams for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset • **MRI Brain/Spine Combo section** ○ Drop metastasis from brain or spine ○ Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course, and where in the CNS the likely localization(s) is/are), and other available information, including prior imaging Changed: • Thunderclap headache with continued concern for underlying vascular abnormality after initial negative brain imaging > 6 hours after onset (as well as in combo Brain MRI/MRA) Deleted: • Precocious puberty: and evidence of an accelerated bone age on x-y • Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years • Follow-up of known meningioma section/background

Reviewed / Approved by NIA Clinical Guideline Committee

Disclaimer: *National Imaging Associates, Inc. (NIA) authorization policies do not constitute medical advice and are not intended to govern or otherwise influence the practice of medicine. These policies are not meant to supplant your normal procedures, evaluation, diagnosis, treatment and/or care plans for your patients. Your professional judgement must be exercised and followed in all respects with regard to the treatment and care of your patients. These policies apply to all Evolent Health LLC subsidiaries including, but not limited to, National Imaging Associates ("NIA"). The policies constitute only the reimbursement and coverage guidelines of NIA. Coverage for services varies for individual members in accordance with the terms and conditions of applicable Certificates of Coverage, Summary Plan Descriptions, or contracts with governing regulatory agencies. NIA reserves the right to review and update the guidelines at its sole discretion. Notice of such changes, if necessary, shall be provided in accordance with the terms and conditions of provider agreements and any applicable laws or regulations.*

~~**GENERAL INFORMATION**~~

~~It is an expectation that all patients receive care/services from a licensed clinician. All appropriate supporting documentation, including recent pertinent office visit notes, laboratory data, and results of any special testing must be provided. If applicable: All prior relevant imaging results and the reason that alternative imaging cannot be performed must be included in the documentation submitted.~~