

Generic Contrast Agents

Our portfolio is growing to serve you better. Now you have a *choice*.



FRESENIUS
KABI

[VIEW CATALOG](#)

AJNR

Annotated bibliography.

N Altman, J A Brunberg, A D Elster, A E George, D B Hackney,
R B Lufkin, J S Ross, J D Swartz, J L Weissman and S M Wolpert

AJNR Am J Neuroradiol 1994, 15 (2) 398-400

<http://www.ajnr.org/content/15/2/398.citation>

This information is current as
of May 20, 2025.

Annotated Bibliography

Nolan Altman, James A. Brunberg, Allen D. Elster, Ajax E. George, David B. Hackney, Robert B. Lufkin, Jeffrey S. Ross, Joel D. Swartz, Jane L. Weissman, and Samuel M. Wolpert

Pediatric Neuroradiology and Congenital Malformations

Krägeloh-Mann I, Grodd W, Schöning M, Marquard K, Nägele T, Ruitenbeek W. **Proton spectroscopy in five patients with Leigh's disease and mitochondrial enzyme deficiency.** *Dev Med Child Neurol* 1993;35:769-776

MR proton spectroscopic findings in five patients with Leigh disease are compared with 114 healthy children. Persistent elevation of the lactate peak may be especially useful for establishing the diagnosis when peripheral lactate levels have returned to normal. MR findings are illustrated. □JAB

Roth SC, Baudin J, McCormick DC, et al. **Relation between ultrasound appearance of the brain of very preterm infants and neurodevelopmental impairment at eight years.** *Dev Med Child Neurol* 1993;35:755-768

Development progress at 8 years of age was assessed in 206 infants who were less than 32 weeks gestation at birth and who underwent neonatal head ultrasound evaluations. Normal sonographic findings (n = 112) were associated with a 4% probability of major neurologic impairment; uncomplicated periventricular hemorrhage (n = 55) was associated with no adverse effect. Ventricular dilation and atrophy were significant independent predictors of neurologic impairment in all measured areas of achievement and cognition. □JAB

Poulton J. **Annotation: mitochondrial DNA and genetic disease.** *Dev Med Child Neurol* 1993;35:833-840

Alterations in mitochondrial DNA, including deletions, duplications, and point mutations, are explained in a clear and concise manner. Their relationship to phenotypic expression is discussed. Clinical disorders included are MERRF, MIELAS, Leber, Kerns-Sare, and chronic progressive external ophthalmoplegia. □JAB

Stroke

Aoki N. **Cerebrovascular bypass surgery for the treatment of Moyamoya disease: unsatisfactory outcome in patients presenting with intracranial hemorrhage.** *Surg Neurol* 1993;40:372-377

Seven patients with intracranial hemorrhage from rupture of Moyamoya vessels underwent bypass surgery, including unilateral and bilateral encephaloduroarteriosynangiosis, and encephalomyosynangiosis. Follow-up cerebral angiography failed to show revascularization and reduction of Moyamoya vessels in all but one of seven patients. One patient suffered rebleeding in the follow-up period. The authors suggest that bypass surgery for Moyamoya disease with hemorrhage is far less successful than that done for ischemia. □JSR

Skull and Craniovertebral Junction

Volpe NJ, Lessell S. **Remitting sixth nerve palsy in skull base tumors.** *Arch Ophthalmol* 1993;111:1393-1395

Report of seven patients with central skull base lesions and clinically apparent sixth nerve palsies. In each case the sixth nerve palsy remitted spontaneously at least once without surgical intervention, radiotherapy, or chemotherapy. The authors postulated a number of mechanisms. Three of the cases are illustrated with good-quality enhanced MR scans. □JDS

Lanzino G, Sekhar LN, Hirsch WL, Sen CN, Pomonis S, Snyderman CH. **Chondrosarcomas involving the cavernous sinus: review of surgical treatment and outcome in 31 patients.** *Surg Neurol* 1993;40:359-371

The authors present their experience with 31 patients with chordomas and chondrosarcomas involving the cavernous sinus, treated with an aggressive surgical approach. With a 24-month follow-up, 21% had recurrences among the 14 patients who had undergone incomplete removal, with no recurrences among the 17 patients with total resection. Seven figures with both MR and CT. □JSR

Ausman JI, Slavin KV. **Commentary. Skull base surgery: too much, too little?** *Surg Neurol* 1993;40:437-438

A conservative reply to the article by Lanzino et al (above) poses the question: what would they want a skull base team to do if they personally had a chondroma or chondrosarcoma of the cavernous sinus?

From Miami Children's Hospital (N.A.); University Hospital, Ann Arbor, Mich (J.A.B.); Bowman Gray School of Medicine, Winston-Salem, NC (A.D.E.); NYU Medical Center, New York (A.E.G.); Hospital of the University of Pennsylvania, Philadelphia (D.B.H.); UCLA School of Medicine, Los Angeles (R.B.L.); The Cleveland Clinic Foundation (J.S.R.); The Germantown Hospital and Medical Center, Philadelphia (J.D.S.); University of Pittsburgh School of Medicine (J.L.W.); and New England Medical Center Hospital, Boston (S.M.W.).

AJNR 15:398-400, Feb 1994 0195-6108/94/1502-0398 © American Society of Neuroradiology

Nose, Paranasal Sinuses, Face, and Oral Cavity

Schaitkin B, May M, Shapiro A, Fucci, M, Mester SJ. **Endoscopic sinus surgery: 4 year follow-up on the first 100 patients.** *Laryngoscope* 1993;103:1117-1120

Several factors may indicate whether surgery will be successful in relieving symptoms. ASA triad and reactive airway disease were associated with a lower success rate of sinus surgery. Sequelae such as adhesions or osteal stenosis usually cause recurrent symptoms within 6 months, although polyps may recur as late as 3 years after surgery. □RBL

Ohnishi T, Tachibana T, Kaneko Y, Esaki S. **High risk areas in endoscopic sinus surgery and prevention of complications.** *Laryngoscope* 1993;103:1181-1185

Three hundred eleven endoscopic ethmoid sinus surgeries were reviewed, and five high-risk areas were identified; 1) lamina papyracea, 2) roof of the ethmoid sinus near the anterior ethmoid artery, 3) lateral lamella of the cribriform plate, 4) ethmoid roof near the posterior ethmoid artery, and 5) area between the sphenoid and posterior ethmoid sinuses (which contains the optic nerve). □RBL

Neck and Nasopharynx

Moses BL, Eisele DW, Jones B. **Radiologic assessment of the early postoperative total-laryngectomy patient.** *Laryngoscope* 1993;103:1157-1160

The records of 132 patients were reviewed. In patients with no signs and symptoms suggesting an impending fistula, pharyngoesophagography was not necessary. The presence of air in the soft tissues of the neck was of no clinical significance in the setting of a recent neck dissection unless there was also extravasation of contrast. □RBL

Zohar Y, Buller N, Shvilly Y. **Recurrent laryngeal nerve paralysis during transvenous insertion of permanent endocardial pacemaker.** *Ann Otol Rhinol Laryngol* 1993;102:810-813

This communication includes two case reports and no images. There is, however, and unusually excellent anatomic discussion not only relating to the anatomy and vascular supply of the vagus nerve and its branches but also discussing supranuclear laryngeal paralysis. Includes information not easily available, which is of definite use to the neuroradiologist. □JDS

Benjamin B, Roche J. **Vocal granuloma, including sclerosis of the arytenoid cartilage: radiographic findings.** *Ann Otol Rhinol Laryngol* 1993;102:756-760

Vocal granulomas are uncommon benign lesions occurring along the posterior third of the cord in direct relationship to the arytenoid cartilage. Gastroesophageal reflux is a well-documented cause. Interestingly, CT scans consistently demonstrated sclerosis of the arytenoid cartilage on the side of the lesion (beautifully illustrated with CT). The authors believe that this is a secondary occurrence and strongly doubt that arytenoid sclerosis could be the cause of the phenomenon. □JDS

Nakayama M, Bradenburg JH, Hafez GR. **Dedifferentiated chondrosarcoma of the larynx with regional and distant metastases.** *Ann Otol Rhinol Laryngol* 1993;102:785-791

Approximately 1% of laryngeal cancers represent sarcomas with chondrosarcoma the most likely diagnosis in this context. They are generally low grade and seldom metastasize. The authors report two well-documented cases of dedifferentiation of these lesions several years after initial resection with widespread metastases. In one case, bony metastases are present; only this case is illustrated (MR/CT). □JDS

Degenerative and Metabolic Disease and Aging

Peterson E, Rosenblum MK, Powers JM, Alvord E, Walker RW, Posner JB. **Effect of brain irradiation on demyelinating lesions.** *Neurology* 1993;43:2105-2112

Demyelinating lesions may resemble brain tumors on CT or MR. The two MR scans in this paper are good examples of this radiologic dilemma. If treated empirically with radiation or chemotherapy the outcome can be poor, which suggests that radiation is especially injurious to patients with demyelinating disease. □SMW

Aylward EH, Henderer JD, McArthur JC, et al. **Reduced basal ganglia volume in HIV-1-associated dementia: results from quantitative neuroimaging.** *Neurology* 1993;43:2099-2104

Patients with human immunodeficiency virus 1-associated dementia complex develop selective basal ganglia atrophy. The sizes of the basal ganglia were obtained by defining their margins on MR scans. Unfortunately no MR images are provided to demonstrate the quantitative measurements. □SMW

Streifler JY, Gornish M, Hadar H, Gadoth H. **Brain imaging in late-onset GM₂ gangliosidosis.** *Neurology* 1993;43:2055-2058

MR scans in patients with late-onset GM₂ gangliosidosis (occurring during adolescence or adulthood) show cerebellar atrophy involving the vermis and the cerebellar hemispheres. The MR images are particularly striking. □SMW

Ophthalmologic Radiology

Stewart CR, Salmon JF, Murray ADN, Sperry C. **Cysticercosis as a cause of severe medial rectus muscle myositis.** *Am J Ophthalmol* 1993;116:510-511

Good-quality axial and coronal CT scans demonstrate a massively enlarged right medial rectus muscle with a central target lesion. Biopsy revealed cysticercosis. There is dramatic reduction in the size of the mass after treatment with albendazole and prednisone (posttreatment CT not included).□JDS

Bahn RS, Heufelder AE. **Review article: pathogenesis of Graves' ophthalmopathy.** *N Engl J Med* 1993;329:1468-1475.

Graves ophthalmopathy is caused by accumulation of glycosaminoglycans in all the tissues of the orbit. This cannot be prevented, only treated, and treatment is fairly drastic (steroids, radiation therapy, or surgical decompression of the orbit). Clear discussions of proposed pathogenesis, clinical findings, and available therapy are accompanied by two CT scans of the orbits (one abnormal, one normal for comparison), photomicrographs, and an illustrated flow chart. The article is detailed (radiologists can skip over the parts about T-cell epitopes and 72-kilodalton heat-shock protein) but quite fascinating.□JLW

Brain Tumors and Cysts

Authors. **Weekly clinicopathological exercises ("CPC").** *N Engl J Med* 1993;329:1335.

A neurologist presents the differential diagnosis of a ring-enhancing parietooccipital brain mass in a 71-year-old woman. Two CT scans illustrate the case. Neuroradiologists know this list well, but the neurologist includes interesting clinical facts (the typical clinical triad of brain abscess is headache, neurologic dysfunction, and fever) in this good, thorough review. The final diagnosis was . . . well, you read it! (Hint: an echocardiogram was subsequently performed.)□JLW

Temporal Bone

Magliulo G, Ronzoni R, Critofari P. **Unilateral acoustic neuroma associated with a tenth cranial nerve schwannoma.** *Ann Otol Rhinol Laryngol* 1993;102:818-819

Excellent quality MR images demonstrate an enhancing intracanalicular lesion and an additional clearly identifiable enhancing lesion interposed between the pontomedullary junction and the jugular foramen. At surgery schwannomas were removed at both locations. There was no history or physical findings to suggest neurofibromatosis type 1 or 2.□JDS

Nadol JB. **Hearing loss (review article).** *N Engl J Med* 1993;329:1092-1101

This excellent review presents the anatomy of the auditory system, many of the causes of hearing loss, and treatment. The author discusses genetic syndromes, effusion and infection, ototoxic drugs, trauma, immune-mediated hearing loss, tumors, circulatory abnormalities (hemorrhage, embolus, and hypercoagulable states), and more. There are diagrams, audiograms, a useful chart, and histopathology, but no CT or MR.□JLW

Inflammatory Disease

Luft BJ, Jafner R, Korzun AH, et al. **Toxoplasmic encephalitis in patients with the acquired immunodeficiency syndrome.** *N Engl J Med* 1993;329:995-1000

This odd study addresses two issues, neither of which is revealed in the (unrevealing) title. The authors treated 49 patients who had acquired immunodeficiency syndrome and toxoplasmic encephalitis with a new regimen of oral pyrimethamine and clindamycin, and found this effective. The authors also devised a "quantifiable" 26-item clinical neurologic assessment that allowed them to determine response to therapy. The authors recommend close monitoring for patients with a solitary lesion on MR, because lymphoma is more likely than toxoplasmic encephalitis. (No radiographs.)□JLW